SAUNDERS
ELSEVIER

First published 2007

ISBN 978 0 7020 2828 1

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

Library of Congress Cataloging in Publication Data
A catalog record for this book is available from the Library of Congress

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The Publisher

Printed in China

Dedication

This book is dedicated to our families.

Acknowledgements

We would like to thank the many people who have helped us during the preparation of this book. Our families and friends have provided us with the constant support required to facilitate our writing. Although many of the photographs in the book were taken by the authors a number of people have kindly allowed us to use their photographs. Individuals who have helped us with photographs include: Roger Blowey, the late Peter Bridge, David Chennells, Tom Clark, Ian Dennis, Stan Done, John Mackinnon, Karin Mueller, the late Alex Noble, the late Richard Potter, Geoff Pritchard, David Strachan, Jill Thomson and Jake Waddilove. We would also like to thank Merial for the use of images from their publication Postweaning Multisystemic Wasting Syndrome: A New Emerging Disease of Swine (16th IPVS Congress, Melbourne, 2000). Despite strenuous efforts we have been unable to ascertain the origin of some of the photographs in our collection. We apologise very sincerely for any omissions from this list of acknowledgements and would be grateful if anyone whose name has been accidentally omitted would kindly let us know.

A number of colleagues and groups have allowed us to quote from their work and to use their tables. Peter Brown and Ann Davis kindly allowed us to reproduce their paper 'A review of the economic effects of disease on pig performance'. We are also pleased to acknowledge the help of the following: John Carr, Tom Alexander and the late Mike Muirhead, the British Pig Executive (BPEX), Pig CHAMP, the Department of Environment, Food and Rural Affairs (Defra), the Department of Primary Industries (Victoria, Australia), the National Office of Animal Health (NOAH), The Pig Veterinary Society (PVS), the University of Minnesota, the Veterinary Laboratories Agency (VLA), the Western College of Veterinary Medicine, Saskatoon. Andrew Gresham and Mark White have also given us helpful advice.

We would also like to thank Joyce Rodenhuis, Rita Demetriou-Swanwick and Glenys Norquay of Elsevier Ltd for their help guidance and encouragement at all stages of the production of this book.

Preface

Unprecedented changes have taken place in the pig industry during the past forty years. These changes have been accompanied by the arrival of a number of new diseases, some caused by previously unrecognised agents and others predisposed by changes in husbandry. The veterinary profession has been heavily involved in investigating and recognising these new conditions and attempting to remedy the problems caused. The profession has and must continue to work closely with the industry to ensure that the welfare and productivity of pigs is at the highest levels. It is not only important to investigate and deal with disease problems. The profession must take a lead in promoting herd health, welfare and biosecurity to provide an increasingly demanding consumer with the highest quality pig meat. Outdoor breeding and organic pig production have presented further challenges. The popularity of pet or fancy pigs has brought about a revival in the management and care of the individual pig. It is hoped that this book will provide the veterinary student, veterinary surgeon and others interested in pigs with a guide to the problems facing the industry.

In the 1960s a large herd of pigs might have consisted of 100 sows and such a herd might have been in the care of two stockpersons. Many people kept a small number of pigs in their gardens or allotments. Pig keeping was a profitable enterprise and veterinary surgeons were frequently called to treat individual sick pigs. The profit margin on each animal allowed an individual pig to be revisited and treated until it was restored to normal health. Classical swine fever was endemic and anthrax was encountered from time to time.

Piglets were weaned at 6–8 weeks of age. Most male pigs were castrated and the spaying of females had recently ceased. Crystal violet vaccine, which gave some protection against swine fever, and an effective swine erysipelas vaccine were used routinely in many herds. Enzootic pneumonia, then erroneously termed 'virus pneumonia', was widespread, causing reduced food conversion and additional days were needed to achieve pork or bacon weight on many farms. There was no vaccine against enzootic pneumonia and control was attempted by improving ventilation and including initially chlortetracycline and later tylosin in the diet. Neonatal and post weaning enteritis were quite frequently seen, with *E. coli* being identified (often by the then free Veterinary Investigation Service) as the cause in most cases. A new disease, transmissible gastroenteritis (TGE), affecting pigs of ages appeared which resulted in the death of most animals that were affected below the age of 14 days. TGE was the first of a number of new and serious diseases that affected pigs causing severe losses.

A reduction in the profitability of individual pigs resulted in larger numbers being kept on individual farms. Sow stalls and cubicles were introduced which allowed fewer stockpersons to be employed. Intensive production methods resulted in a higher incidence of stress and disease. Batch farrowing was practised and piglets were mixed and transferred to flat deck weaner pools. The paucity of stockpersons often resulted in pigs not receiving veterinary attention until serious losses of life and production had occurred. Farmers were tempted to try 'do-it-yourself' methods to reduce their veterinary costs. In many cases incorrect diagnoses were made or courses of treatment were either inappropriate or were not completed. The incidence of diseases such as sarcoptic mange increased and the spread of this disease was exacerbated by intensive husbandry. In the struggle to improve profitability, hybrid pigs were produced which combined the best features of the traditional breeds. Litter size and growth rates were the production parameters that were increased. Efforts were made to control disease in elite herds by better biosecurity. In many cases, despite better production parameters, natural resistance to disease did not improve.

Perhaps almost inevitably other important pig diseases appeared. One of the first 'new diseases' was swine dysentery which was seen after the withdrawal of tylosin as a growth promoter. It is possible that the widespread use of tylosin masked the presence of swine dysentery. Porcine parvovirus was recognised as an important cause of abortion in pigs. Porcine reproductive and respiratory disease (PRRS) was identified and spread quickly in many countries. The viral cause proved difficult to identify and much damage was done before an effective vaccine could be produced. Outbreaks of the notifiable diseases classical swine fever and foot and mouth disease at the start of the 21st century caused additional problems to the pig industry.

Two new diseases have been recognised in the past 10 years – porcine dermatitis and nephropathy syndrome (PDNS) and porcine multisystemic wasting syndrome (PMWS) – and caused serious losses in the industry. Both diseases have been shown to be associated with porcine circovirus-2, but other causal factors have yet to

be identified. PMWS in particular has caused heavy loss of life and devastating production losses. The disease has been shown to respond poorly to symptomatic treatment. The worst effects of the disease can however be minimised by improving husbandry and by reducing stress and increasing comfort. The importance of more traditional care and management has been clearly demonstrated.

The major challenges facing the pig industry have fortunately been accompanied by major developments in pig science. New diagnostic techniques have facilitated diagnosis. New vaccines and new therapeutic regimes have added to our ability to prevent and treat disease.

This book has been written to provide as much information about pig health and disease as possible in a reader friendly style. All sections of the book are illustrated where appropriate, mostly with coloured photographs. The first chapter deals with the investigation of clinical problems on pig farms. A good clinical examination is an essential part of any investigation of disease. Although the pig is not the easiest animal to examine, with care and skill a full and detailed examination can be performed. The adage 'mistakes are made by not looking rather than not knowing' is as important in the pig as in other species. The importance of herd health along with care of the individual has already been stressed. The second chapter of the book deals with herd health including herd health plans and consumer assurance schemes.

Chapters 4–11 deal with diseases of the various body systems. In each chapter the aetiology, incidence and epidemiology of specific diseases are described. Clinical examination is described with special reference to that body system. Consideration of the treatment and control of each disease is described.

In Chapter 12, the special problems of outdoor and organic pigs are discussed. The pet pig is considered in Chapter 13 and antimicrobial therapeutics are dealt with in Chapter 14. Surgical procedures including anaesthesia and analgesia are described in Chapter 15.

Post mortem examination is an important aid to the diagnosis of pig diseases. Post mortem technique is described in detail in Chapter 16, together with techniques for sampling and euthanasia. Chapter 17 contains details of the haematology and biochemistry of the pig, and the important subject of differential diagnosis is discussed in Chapter 18.

There are currently about 5 million pigs in the UK. 1.5 million tonnes of pig meat is consumed annually in the UK with half of this amount being imported. A recent manpower study by the RCVS showed that only 0.4% of veterinary time is spent working with pigs. Much consultative work is done by specialists and pig courses in veterinary schools have been shortened. It is very important that more veterinarians become skilled in the diagnosis and treatment of pig disease. The professions must remain fully involved with the industry to ensure that pigs receive the highest standard of veterinary care. Veterinary surgeons must remain at the forefront of national and international disease control and ensuring the welfare, herd health and productivity are maintained at high levels.

It is hoped that this book will provide all the information required by those who are involved in the examination and treatment of that fascinating animal the pig.

PGGJ
PDC

Cambridge 2007

Investigation of clinical problems on pig farms

Introduction

The purpose of the clinical investigation is to identify the clinical abnormalities that are present and the risk factors that determine the occurrence of the disease in the individual or population. From this information, the most likely cause can be determined and the risk factors identified. In addition, the organs or systems involved, the location, the type of lesion present, the pathophysiological processes occurring, the severity of the disease, and the epidemiology of the outbreak may be deduced. The investigation may involve subclinical disease where a problem may have been identified from substandard performance indices such as growth rates, food conversion efficiency, and litter size. In this case, the investigation may begin with a detailed analysis of farm records to characterize the qualitative and quantitative nature of the substandard performance. The chronicity, the groups affected, and the progression of the problem may be revealed by the records.

Individual animals that are sick or not doing well should be removed from the group and put into hospital accommodation for nursing until they have recovered or a decision has been taken to euthanize the animals. Mixing groups of animals or splitting a group can result in fighting and a severe setback of up to a week until a new stable dominance hierarchy is established. Great care is therefore needed when reintroducing an animal. There is a temptation to reintroduce the recovered animals or poor doer to a younger group of piglets. The disease implications of this should be considered very carefully. Rearing recovered animals or poor doers as a separate group is ideal although may not be practical or economical. Euthanasia may be the most realistic option.

When examining individual pigs, careful consideration should be given to minimizing the restraint required while ensuring the operator's safety (Figs 1.1 and 1.2).

Equipment required

When visiting a pig unit, the clinician must make sure that the following equipment is available:

- clean protective clothing and footwear,
- approved disinfectant and foot brush,
- thermometer and stethoscope,
- sterile containers for samples,
- swabs,
- equipment for post-mortem examination,
- injectable medication for sick pigs,
- tape measure or a laser measure,
- torch, and
- water or food medication for in-contact pigs.

Components of the clinical examination

The clinical investigation and diagnosis in the pig may include some or all of the following steps:

- owner's complaint,
- history of the unit (this may include analysis of records),
- history of the patient,
- signalment of the patient,
- environment of the patient,
- observation of the patient and its group,
- restraint of affected individual animals,
- physical examination of the patient (post-mortem examination is sometimes performed at this stage),
- provisional or tentative diagnosis (treatment, control, and prevention programmes may be initiated at this stage),
- further investigations,
- definitive diagnosis,
- treatment and response to treatment,
- control and prevention programme.

History of the unit

The following details should be ascertained by interview and from the farm records.

- Number and category of pigs on the unit.
- Production details: breeding, finishing unit?
- Indoor or outdoor?
- Nucleus, multiplier, or commercial?
- Disease-free status (the term *disease-free* may indicate freedom from a variable number of specified diseases).
- Flow of animals through the unit.
- Farm assurance scheme in operation?
- Current veterinary health plan.
- Vaccination programmes.

Fig 1.1 – Whenever possible, pigs should be examined quietly and with minimum restraint.

Fig 1.2 – A more detailed chest auscultation is possible in a quiet sow restrained by a farrowing crate.

- Ecto- and endoparasite control programmes.
- Routine medication.
- Biosecurity protocols.
- Production figures for the unit: targets and whether or not they were achieved.
- Reports from previous farm visits.
- Abattoir feedback reports.
- Previous and current diseases.
- Feeding system used.
- Nutritional history of affected group(s).
- Breeding policies.
- Pig flow plan for the unit (pig movement through the unit).
- Recent movements of pigs (movements book).

History of the sick pig(s)

The following details should be obtained from the best-informed person, which in most cases is the farm manager or the stockperson. This information is usually very important in formulating differential diagnoses.

- Farm herd health plan (vaccination, anthelmintics, management protocols).
- Previous disease problems on this farm or in these pigs.
- Number of pigs affected (morbidity) and number of deaths (mortality).
- Groups affected.
- Age of animal affected.
- Signs of ill health observed by the owner or stockperson.
- Duration of problem.
- Progression of disease.
- Details of other sick pigs on the unit.
- Origin of affected pigs (homebred or purchased?). If purchased:
  - date of purchase, and
  - health status of unit of origin.
- Treatment already given by stockperson.
  - Response to such treatment.
- Recent changes in diet.
- Recent changes of staff.
- Recent interruption of water and power supplies.
- Availability of carcasses for post-mortem examination.

Signalment of the pig(s)

The age, sex, and breed of the pigs may influence the likelihood of a diagnosis. Swine erysipelas is rarely seen in pigs under the age of 16 weeks. Swine dysentery is mostly seen in pigs aged 8–16 weeks. Enteric colibacillosis is seen mostly in piglets aged less than 3 weeks; it may occur in weaners but is rarely seen in adults.

The pigs' environment

The availability of food, water, and protection against extremes of weather is essential for good health to be maintained. These facilities should be readily accessible to the number of pigs being accommodated. The environment should also be secure and have handling facilities. Poor ventilation affects the incidence of respiratory disease. A high incidence of lameness may be associated with poorly maintained flooring. Faeces scattered all over the floor may indicate diarrhoea or overcrowding. The following parameters should be assessed when looking at the environment.

The indoor environment:

- ambient temperature,
- light,
- ventilation,
- stocking density,
- flooring,
- bedding,
- cleanliness,
- state of repair of fitments,

- faeces, and
- abnormal discharges (including blood and vomitus).

The outdoor environment (Fig. 1.3):
- freedom from hazards,
- availability of wallows,
- shelter from extremes of weather, and
- accessibility of other livestock.

Observation of the pig(s)

General observations

Growing pigs spend a lot of their time sleeping and huddled together. If disturbed, the pigs wake rapidly, often make a barking noise, and then run to the far corner of their pen. Fear is then replaced by interest, and the pigs approach the clinician cautiously and may start to nibble boots or overalls (Fig. 1.4). They are naturally inquisitive animals.

Sick pigs may be too ill to take part in normal activities and may be knocked over by their pen mates. Their ears droop or are held flat against the head. The curl in the tail may be lost, and they tend to 'bury themselves' in the straw. They may have a dull and depressed demeanour.

Fig 1.3 – Sows in an outdoor unit.

Fig 1.4 – Pigs cautiously approaching the clinician.

Dirty pigs may indicate that the animals are too hot and have been wallowing or lying in the dunging area, are overstocked, are in an environment that is not cleaned out regularly enough with replacement of fresh straw, or have an enteric diarrhoeic problem. Queuing at nipple drinkers indicates a problem with the provision of water. Lying patterns may indicate if the pigs are too hot (spread out) or too cold (huddled). Observation of the patient both at rest and in locomotion is usually extremely rewarding. Healthy pigs are bright, alert, and reactive. Any pig lying away from the main group or behaving abnormally may be ill. Care should be taken not to overlook individual sick pigs that are often lying down in the straw bedding out of sight in creep boxes or hutches. This often means entering the pen and inspecting the covered areas. Once in the pen, it is then possible to 'stir up the group' so that signs of disease such as lameness, coughing, and dyspnoea may become more pronounced.

Uneven condition scores in groups of piglets, weaners, and finishing pigs may indicate the presence of disease or insufficient creep or trough space. Inappropriate or rapid changes in the condition scores of the sows should be investigated. Sows in the dry sow yard should be monitored for presence of the thin sow syndrome and pressure sores from the farrowing crates. Individual sick pigs may be in poor condition and have an empty, gaunt appearance. Such pigs may be too ill to take part in normal activities and may be knocked over by the movements of other pigs. Damage caused by vices such as tail biting, flank sucking, or ear biting may be seen.

Abnormalities of body shape

Abdominal distension is relatively uncommon in pigs. Causes include peritonitis, ascites, and rectal stricture. Gaunt pigs with hollow sides are often a sign of intestinal disease.

Deviation of the snout is seen in some cases of atrophic rhinitis. *Localized swellings in muscles* may be associated with the porcine stress syndrome, injury, or vitamin E or selenium deficiency. Runt pigs are easily identified; they have a large head relative to the body and are small for their age. They are hairy and thin.

Abnormal swellings on the body surface

Scrotal and umbilical hernias are quite frequently seen in pigs. *Subcutaneous abscesses and haematomata* are frequently seen and are often caused by fighting or other superficial injuries. In boars, a swelling in the prepucial region may be caused by *distension of the prepucial diverticulum.*

Abnormalities of the skin

Pruritus is seen in a number of skin diseases. The most common cause is sarcoptic mange, which can affect animals of all ages. *Skin discoloration* can be readily observed in unpigmented breeds. Obvious signs of *sunburn* may be visible in pale-skinned pigs, especially on outdoor units. Extensive superficial scratches and bite wounds may be visible when fighting has occurred following mixing of groups.

Diarrhoea

Normal pig faeces are quite firm and well formed, and are passed in a dunging area in one corner of the pen. Pigs with diarrhoea may show faecal staining on the perineal area and often defecate in all parts of the pen.

Neurological signs

These are associated with a number of conditions in pigs. One of the most common is *head tilt*. This is mostly caused by middle ear or vestibular disease. Physical tilting of the head is sometimes seen in pigs with a large, heavy aural haematoma. *Fitting* is seen in pigs with meningitis, which can be caused by a number of serotypes of *Streptococcus suis* and also occurs in some cases of *Haemophilus parasuis* infection (Glasser's disease). Fitting may also be seen in some cases of classical swine fever and in water deprivation (salt poisoning). It can also be a feature of exotic diseases such as Teschen's disease and African swine fever. *Ataxia* is seen in animals that have suffered a spinal injury or compression of the spine by infection or a pathological fracture. *Incoordination of the forelimbs* is a feature of bowel oedema.

Udder conformation

Changes in udder conformation, which are best appreciated with the animal in the standing position, include enlargement of single glands as a result of acute or chronic mastitis and generalized enlargement in mastitis–metritis–agalactia. Inactive glands are smaller.

Respiratory signs

Abnormal breathing is readily observed, particularly following exercise. Coughing is a feature of many forms of pneumonia. Prolonged and persistent coughing may be heard in pigs suffering from enzootic pneumonia. Severely dyspnoeic pigs may breathe through their open mouths. Nasal breathing is difficult in cases of rhinitis, and a snuffling sound accompanies air movement.

Lameness

Lameness in pigs is common. Observation may indicate the prevalence, the limbs involved, the severity, and the possible cause. Pigs suffering from a fracture are often non–weight bearing on the affected limb. This is also seen in animals with painful digital abscesses. Swelling of the joints is associated with acute or chronic arthritis. Disuse of the affected limb may result in muscle atrophy and reduced joint movement. Ruptured muscles, for example hind limb adductors, result in swelling and an inability to stand.

Restraint

Whenever possible, pigs should be examined quietly and with minimum restraint. The greater the restraint, the more upset the pig becomes and the more difficult and less effective the clinical examination is. Making contact vocally and by palpation has a soothing effect. Pigs also enjoy being scratched along their backs, sides, and behind their ears, and will often stand still during the process.

The examination of one pig in a pen surrounded by many others can be difficult. Fellow pigs may attack a restrained animal, and equipment may be broken or damaged by biting. Individual animals can be removed from their pen. Separation must be minimal to avoid a returning pig being attacked and injured by its fellows. Sows and boars can be enticed into a feeder for examination using food.

Weaners and finishers can be penned in a corner behind a board. They can be effectively restrained while their temperature is taken by holding their tail and looping it around the fingers. Individual animals can be restrained in a weighing machine or turned into dorsal recumbency and placed in a cradle.

Restraint of animals in an outdoor environment can be very difficult unless a secure accessible pen is available. If such animals are seriously unwell, requiring detailed examination and longer-term treatment, they should be brought into an indoor environment.

Restraint for examination may involve the following.

- Snares. A metal or cord snare secured around the upper jaw and snout of pigs can be an effective method of restraint for techniques such as bleeding (Fig. 1.5). In weaners, a length of bale string can be used in a similar fashion.
- Pig board: used to direct the pig rather than restrain it (Fig. 1.6).
- Tail holding. Individual pigs from weaners to adults can be restrained briefly for taking temperature, looking at feet, and auscultation of the chest (Fig. 1.7).
- Small piglets can be picked up by a hind leg and held for examination (Fig. 1.8).
- Feeding stalls or weighing crates can be useful to restrain single or groups of pigs (Fig. 1.9).
- Food on the floor to distract the pig (Fig. 1.10).

Fig 1.5 – Snares are very effective in restraining pigs for clinical examination.

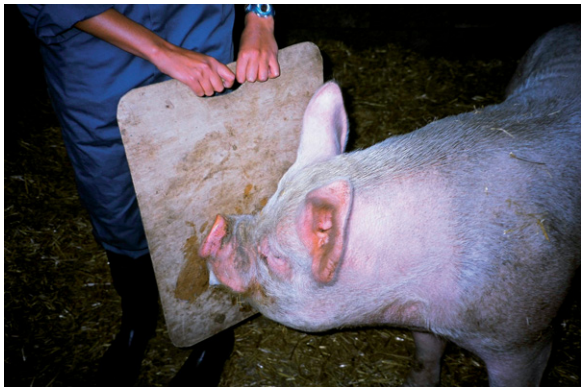

Fig 1.6 – The short nose of a normal Middle White gilt. Note the presence of a pig board to provide protection for the clinician.

Fig 1.7 – Restraint of a weaner using the tail to enable the temperature to be taken.

Fig 1.8 – Small pigs can be lifted from the floor and held in the arms.

Fig 1.9 – Feeding stalls can be used for restraint of adult pigs.

Fig 1.10 – Placing food on the floor will often distract the pig to facilitate examination.

Chemical sedation may involve the following.

- Azaperone (Stresnil, Janssen Animal Health) is the only sedative licensed for use in pigs in the UK. It is given by intramuscular injection at a dose of 1–2 mg/kg.
- General anaesthesia or heavy sedation. No drugs are currently licensed for use in pigs in the UK. An effective but expensive combination of drugs is as follows.
  - Detomidine 50–100 µg/kg given by intramuscular injection.
  - Butorphanol 0.2 mg/kg given by intramuscular injection at the same time as detomidine.
  - Ketamine 3–5 mg/kg given by intramuscular injection 5–10 min later.
  - The effects of the above drug combination can be rapidly reversed by the administration of 50 mg/kg of atipamazole by intramuscular injection.

Physical examination

Physical examination may be performed because of an individual problem or a herd problem, or as part of a routine herd health consultation. It may be part of a biosecurity protocol to examine all incoming animals while still in quarantine. The animals to be examined may be kept intensively indoors or extensively outdoors and may have organic status. These animals may be boars, dry sows, sows and piglets, weaners, growers, and finishers.

When clinicians are called on to examine individual pigs, the implications for the rest of the herd of any diseases found should be carefully considered. The level of certainty required regarding the diagnosis may be of particular importance with regard to the cost of targeted control and prevention strategies.

When entering or leaving a pig farm, the clinician must make every effort to avoid bringing infection on to a pig unit or transferring infection to another unit by following effective decontamination protocols.

The natural tendency of the pig to try to escape from any restraint or confinement limits the way in which it can be restrained, handled, and examined. Its tendency to squeal when restrained makes some aspects of the examination, such as auscultation of the chest, difficult but seldom impossible. Sows with litters and breeding boars are potentially aggressive and dangerous. Their pens should not be entered in the absence of a stockperson.

Attempting to examine one or two sick pigs in a group of 20 others can be difficult. Individual pigs can be taken out of the pen briefly for examination, but they must be reintroduced carefully, as the returning pigs may be set on, attacked, or even killed by their pen mates. The difficulties of examining pigs can be largely overcome by using a quiet and gentle approach. Patience, quietness, and a knowledge of pig behaviour are very important. In general, the greater the restraint of the pig, the less effective the clinical examination. For some procedures, such as blood sampling and x-ray examination, restraint and possibly sedation are essential.

Methods of clinical examination

There are several different approaches to the clinical examination. The *complete clinical examination* consists of checking for the presence or absence of all the clinical abnormalities and predisposing disease risk factors. From this information, a ranked list of differential diagnoses is deduced. This is a failsafe method and ensures that no abnormality or risk factor is missed.

The *problem-oriented method* (hypothetico-deductive method) combines clinical examination and differential diagnosis. The sequence of the clinical investigation is dictated by the differential diagnoses generated from the previous findings. This results in a limited but very focused examination. The success of the method relies heavily on the knowledge of the clinician and usually assumes that a single condition is responsible for the abnormalities.

Many clinicians begin their examination by performing a *general examination* that includes a broad search for abnormalities. The *system* or *region* involved is identified and is then examined in greater detail using either a complete or a problem-oriented examination.

Normal physiological values are given in Tables 1.1 and 1.2.

Physical examination of adult and growing pigs

The scope, sequence, and detail of the physical examination will vary according to the class of animal, the

Table 1.1 Normal physiological values of adult pigs

Variable	Range	Average
Temperature (°C)	38.0–39.0	38.5
Resting pulse rate (beats/min)	60–90	75
Resting respiration rate (breaths/min)	10–20	15
Oestrus cycle (days)	18–24	21
Gestation (days)	110–116	114

Table 1.2 Normal physiological values of piglets

Variable	Range	Average
Temperature (°C)	39.0–40.0	39.5
Pulse (beats/min)	100–120	110
Respiration (breaths/min)	24–36	30

temperament of the animal, and the restraint facilities available.

Temperature, pulse, respiratory rate, mucous membranes, and carcass lymph nodes

Temperature

The pig's normal rectal temperature is 39°C. A high temperature is seen in many infectious diseases such as swine erysipelas, when body temperature may be in the range 40.5–41°C. In cases of heatstroke, to which pigs are very susceptible, body temperature may rise to 43°C, which if prolonged is incompatible with life. A low temperature is not a good sign and is seen in terminally ill pigs and those with problems such as renal or hepatic failure. A low temperature is often seen in cases of the mastitis–metritis–agalactia syndrome. In diarrhoeic animals, the placement of a thermometer may induce an episode of diarrhoea.

Respiratory rate

Respiration should be observed, and the rate taken before the pig is disturbed by observation of thoracic movement. Respiratory rate is normally 15–20 breaths/min. Rapid, laboured breathing interrupted by coughing may be present in cases of enzootic pneumonia. Open-mouthed breathing is an unusual but ominous sign and is occasionally seen in cases of severe pneumonia and in the porcine stress syndrome. Exaggerated chest wall movements occur in cases of respiratory distress.

Pulse

A pulse of 60–90 beats/min is normal in adult pigs but increases rapidly if the pig is stressed. Finding a peripheral pulse can be difficult. The femoral artery can be used in young pigs and the ventral coccygeal artery in adult pigs, although rarely done. The heart rate is normally measured by cardiac auscultation. Brief periods of auscultation only may be possible. A persistently rapid pulse may be found in cases of endocarditis in which poor pulse recovery from modest exercise is also seen.

Mucous membranes

These are readily observed in the conjunctiva and the vulval mucosa. Tickling the pig behind the eye often causes slight relaxation and allows the clinician to briefly evert the lower lid to view the conjunctiva. The *capillary refill time* (normally <2s) can be measured at the vulva. The capillary refill time is the time it takes for the colour of the mucous membrane to return following blanching by applying digital pressure to the surface. Extended refill times indicate poor peripheral perfusion. Colour changes may indicate toxaemia, cyanosis, jaundice, and anaemia (Fig. 1.11). Petechial haemorrhages may be present in septicaemia or bleeding disorders.

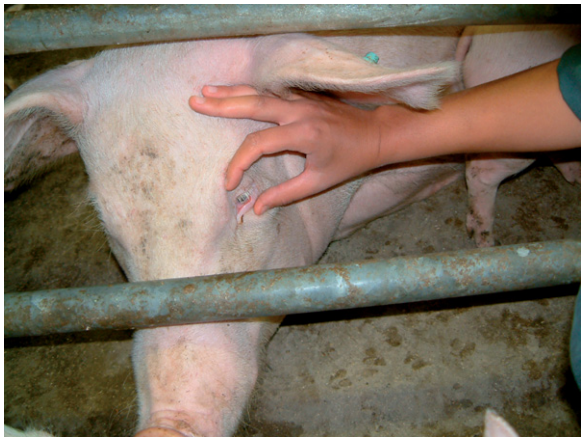

Fig 1.11 – The colour of the conjunctival mucosa can be readily seen in quiet sows.

The carcass lymph nodes

These are not palpable unless enlarged. Enlargement of a few or all the carcass lymph nodes is seen in cases of multicentric lymphosarcoma, which can affect pigs from a few days old to maturity. Marked enlargement of one submandibular lymph node with swelling of the adjacent tissues and in association with severe systemic signs is found in some cases of anthrax. Enlarged inguinal lymph nodes may be palpable or visible in cases of postweaning multisystemic wasting syndrome.

The skin

The skin should be examined visually and then manually. The palm of the hand should always be passed over the withers and shoulders of pigs to check for the presence of the raised diamond-shaped lesions that are seen in cases of subacute erysipelas. Such lesions are often palpable before being visible, especially in black pigs. Chronic skin lesions will be present if ear or flank sucking has occurred. Tail biting will be self evident.

Skin temperature

Skin temperature closely reflects body temperature. The skin often feels unrepentantly warm in pyrexic animals.

Skin colour

Skin pallor is a feature of iron deficiency anaemia, in which affected pigs may also show pica – voraciously devouring specks of earth on the clinician's boots. *Yellow discoloration* indicates jaundice, which is seen in some cases of postweaning multisystemic wasting syndrome, in hepatic cirrhosis, and in the relatively uncommon con-dition of *Leptospira icterohaemorrhagiae* infection. Large areas of the back may be *red and burned* in cases of sunburn. *Areas of purplish red discoloration* are seen in cases of septicaemia and toxaemia. Such areas may be

generalized or more localized, affecting the ears, jowl, belly, axilla, and caudal aspects of the thighs. Localized lesions are sometimes seen in cases of *Salmonella* infection and toxaemia. Skin ecchymoses are seen in some cases of classical swine fever and porcine dermatitis and nephropathy syndrome.

Skin texture

Decreased turgor occurs in dehydration, and this can be detected by pinching the skin of the ventral abdomen. Prolonged tenting of the skin results.

Ectoparasites

Sarcoptes scabiei infestation is very common in pigs, causing pruritus, self-inflicted skin damage, and thickening of the skin, especially on the head (including the ears), dorsum of the back, perineum, and limbs. The external auditory canal is often filled with dark wax in which mites may just be visible. The large dorsoventrally flattened louse *Haematopinus suis* is readily seen on the dorsal surface of the body.

Lesions of skin disease

The raised red, diamond-shaped lesions of *swine erysipelas* have been mentioned above. In peracute erysipelas, the pig may die before skin lesions become apparent. *Ringworm* caused chiefly by *Trichophyton mentagrophytes* is characterized by light-brown, slightly raised lesions seen chiefly on the back and flanks. *Pityriasis rosea*, which is characterized by raised centrifugal irregular lesions, may appear suddenly on the ventral surface of the body in growing pigs.

Skin injuries

Skin injuries are mostly the result of fighting after mixing. They vary from superficial scratches to deep extensive lesions caused by the tusks. Subcutaneous haemorrhage leads to the formation of *haematomata*. *Abscess formation* frequently follows injury. Haematomata are usually fluctuant but cool to the touch; abscesses are warm. Differential diagnosis can be confirmed using ultrasonographic scan or by aspirating the contents.

The head and neck

The clinician should examine and perform appropriate tests to establish that normal hearing and sight are present.

The eyes

Foreign bodies in the eyes and ears of pigs are extremely uncommon. *Nystagmus* is often visible in cases of meningitis. Severe corneal damage may occur in pigs that are fitting. *Exophthalmus* may be seen in cases of mediastinal lymphosarcoma, and eyelid oedema is a feature of bowel oedema. *Ocular discharge* is seen in some respiratory diseases and in classical swine fever. Conjunctivitis is relatively common in suckling piglets.

The ears

The ears are vulnerable to injury by fighting and loss of tags. Sarcoptic mange may affect the pinna and the external auditory canal. Head shaking caused by mange infestation may lead to the development of haematomata in one or both ears.

The mouth

The mouth is rarely affected by disease but can be briefly observed by using a smooth stick to encourage the opening of the mouth. Heavy sedation or general anaesthesia is required for a more detailed examination.

The snout

Close examination may confirm the presence of rhinitis, and deviation of the snout is seen in some cases of atrophic rhinitis.

The neck

Nodular swellings just caudal to the ear are usually the result of fibrosis caused by earlier injections. The jugular vein cannot be visualized or palpated.

The chest and abdomen

The heart

Detailed auscultation of the heart is difficult unless the pig is severely ill, very quiet, or asleep (Fig. 1.12). Auscultation is non-productive when the animal is eating, because of the sound of chewing and grunting. This should be borne in mind when deciding on a method of restraint. Gross abnormalities such as loud murmurs may be audible, and an assessment of the heart rate can be

Fig 1.12 – Brief auscultation of the heart of a weaner pig is possible in some cases. Forcible restraint of the pig makes auscultation impossible.

made. The stethoscope is pushed forwards under the triceps muscle to the fifth rib, and both sides of the chest should be auscultated. First and second heart sounds can be identified. An apex beat may be palpable in normal piglets and in animals with cardiac enlargement. A systolic murmur may be detected over the tricuspid valve in cases of endocarditis. Fluid sounds may be audible in early cases of pericarditis, but later on the heart sounds may become muffled. Fluid sounds are occasionally audible in mulberry heart disease.

The lungs

Detailed auscultation is difficult. Pigs suffering from respiratory disease are often distressed, and any attempt to restrain them can make their distress worse. The lung field extends from the shoulder caudally to the 13th rib in the dorsal chest and as far back as the seventh rib ventrally. Wheezing (narrowed airways), bubbling sounds (intermittent unblocking of bronchioles), and high-pitched squeaking sound (pleuritis) may be audible in *enzootic pneumonia* and other causes of pneumonia, when there is usually pathology in the ventral lung field. In *pleuropneumonia*, abnormal lung sounds may be audible over the dorsal lung field. Percussion of the chest is unrewarding.

The abdomen

The *abdomen* should have a full but not distended or bloated appearance. Deep palpation of the abdomen is resented, but fetal presence and movement are palpable in recumbent sows in late pregnancy. Severe pain may be elicited when the sublumbar area is palpated in the presence of pyelonephritis. In quiet animals, auscultation reveals borborygmi.

Abdominal distension can be the result of *rectal stricture*. Affected animals pass intermittent flatus and scant watery black faeces. The presence of a stricture can be confirmed by digital rectal examination. *Ascites* is uncommon and associated with hepatic cirrhosis. In both of the above conditions, affected animals are often in very poor bodily condition. Percussion and auscultation may indicate a gas-filled viscus with acute obstruction. Percussion and palpation for a fluid thrill is difficult to assess. A less common cause of abdominal distension is *peritonitis*. Scrotal and umbilical hernias are common in pigs. Ultrasonography facilitates further evaluation of abdominal contents.

The perineum

The colour of faecal staining on the perineum may be of diagnostic value. Dark tar-like faeces containing blood are seen in cases of swine dysentery. Dysentery is also seen in some cases of salmonellosis and porcine intestinal adenomatosis. It is rarely seen in enteric colibacillosis. *Rectal and vaginal prolapses* are not uncommon in pigs.

The urinary system

Pyelonephritis is seen chiefly in sows and gilts within a week of service. The passage of dark-coloured urine containing blood, pus, and cellular debris is often the first sign of the disease. Initially, the animal is pyrexic and may walk with an arched back. Toxaemia and uraemia develop rapidly. If urine is not passed naturally on standing, a sample can be readily taken via a catheter.

Lameness: the limbs and feet

Lameness is a common problem in pigs. Observation may indicate which limb(s) is affected and the severity of the lameness. Further clinical examination requires restraint, palpation, and manipulation of the affected limb (Fig. 1.13). Comparison with the normal limb may help identify the abnormality. Foot and upper limb lesions each account for 50% of clinical lameness.

Weaner pigs and young finishers can be carefully lifted by an assistant while an affected limb is examined in detail. In quiet sows and boars the foot may be lifted briefly, but this often causes rapid kicking movements in the pig, making examination difficult. Heat, swelling, and pain may be detected. Massage of the ventral body surface encourages the animal to remain in recumbency, enabling the foot and lower limb to be examined in detail before the pig stands (Fig. 1.14). The cause of lameness can often be determined before the pig stands up and the degree of lameness is observed. The sole should be checked for white line disease, and the coronary band for swelling and discharging sinuses.

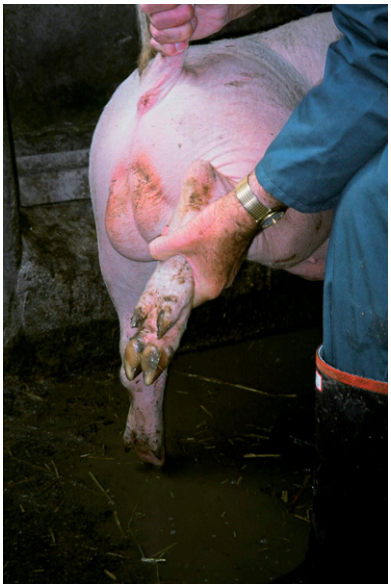

Fig 1.13 – Manual inspection of the foot in an adult pig on a snare. Lifting the feet (especially the forefeet) is resented in most pigs.

Fig 1.14 – If the lame sow is lying quietly, her feet can be examined with ease while she is recumbent. The severity of lameness is determined at a later stage when she is encouraged to stand.

Foot lameness

Overgrowth and cracking of the hooves causes lameness, especially in breeding animals. Solar necrosis and white line disease can also cause lameness, while infection of the third phalanx ('bumble foot') may cause the leg to be non-weight bearing. Vesicles and subsequent ulcers on the coronary band may be seen in foot and mouth disease and in swine vesicular disease. Similar lesions may be caused by contact with irritant solutions. Biotin deficiency may cause multiple hoof cracks.

Joint disease

Numerous organisms cause septic arthritis in various age groups of pigs. These include *Mycoplasma hyosynoviae*, *M. hyorhinis*, *Streptococcus suis*, *Haemophilus parasuis*, and *Arcanobacter pyogenes*. Most infections are characterized by pyrexia, distension of the joint capsule, and lameness in one or several limbs. In neglected cases the joint infection can spread to the bones, causing osteomyelitis, ankylosis of the joints, and severe muscle wasting. Osteochondrosis may be present in rapidly growing animals.

Fractures

The affected limb is usually non-weight bearing. Crepitus may be detected when the limb is gently manipulated. Concurrent auscultation with a stethoscope increases the acuity. Fracture may be predisposed by osteomalacia and osteodystrophy. Detailed diagnosis is aided by radiography of the anaesthetized pig if economically justified. Slippage and displacement of the femoral head is seen in young boars. The affected limb is just weight bearing, but the animal is severely lame. The condition is part of the leg weakness syndrome, where osteochondrosis is the main pathological lesion.

Muscle damage

This is usually the result of an accident, for example when gilts are mounted by a heavy boar. Rupture of the adductor muscles may result in inability to stand. Affected muscles may be swollen and painful to the touch. Ultrasonography confirms the extent of muscle damage.

The external genitalia, mammary gland, and rectal examination of the sow

The *vulva* is frequently damaged by *biting injuries* including lacerations inflicted by other sows, especially after weaning or in late pregnancy when the vulval lips can become swollen. A *vulval haematoma* involving one or both lips of the vulva may develop as a result of crushing pressure sustained during farrowing or by the sow rubbing her hindquarters against her farrowing crate.

A *vaginal discharge* may be seen during normal pregnancy in some sows. It may also indicate recent abortion. A pale yellowish white discharge is seen in some cases of the mastitis–metritis–agalactia syndrome. A foul-smelling, watery vaginal discharge with placental remnants after farrowing may indicate that one or more fetuses have been retained. Affected animals may be toxaemic and very ill. A vulval discharge is normal in sows up to 5 days after farrowing and consists of mucus with white flocculent and sometimes odourless bloody material.

A uterine prolapse involving one or both uterine horns may occur during or most commonly after farrowing. Affected sows may be found dead as a result of internal haemorrhage.

A mild postural partial vaginal prolapse is sometimes seen in recumbent pregnant sows.

The udder

The normal mammary gland and teats should be evenly soft and free from injury (Fig. 1.15). The udder becomes firm and turgid as farrowing approaches. In the lactating sow, a bead of milk can usually be expressed from each teat. Larger amounts can be expressed only when the sow is either farrowing or letting down her milk in the presence of attentive piglets. Cranial teats may not be patent due to teat necrosis. Inverted teats or blind teats may be detected. Abrasions and lacerations are common because of trauma from the floor or the teeth of the piglets.

Acute mastitis usually affects only a small number of mammary glands and mostly occurs within a few days of farrowing. The sow is initially pyrexic, but the temperature falls as she rapidly becomes very ill. The affected parts of her udder are extremely hard, and the skin becomes discoloured through septicaemia and toxaemia. If a milk sample can be obtained, it may be abnormal. Mastitis–metritis–agalactia is usually seen within 48 h of farrowing. Body temperature is subnormal. The whole udder becomes mildly inflamed and indurate. *Chronic*

Fig 1.15 – The entire udder should be palpated during the clinical examination.

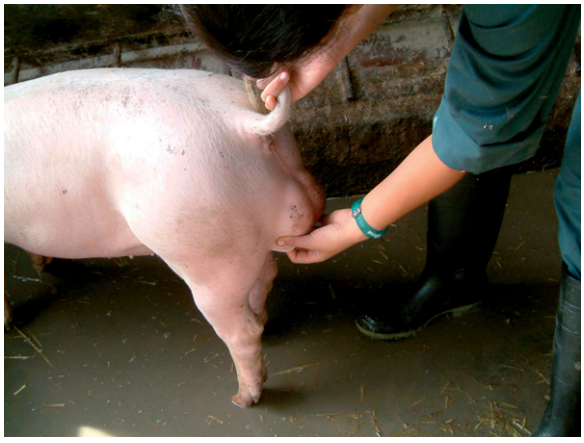

Fig 1.16 – Testicular palpation in a young boar.

mastitis is usually seen in dry sows after weaning. One or more mammary glands become very hard as thick-walled abscesses are formed, some with discharging tracts.

Rectal examination in the sow

Rectal examination in the sow can be carried out with a well-lubricated, gloved hand. The dry faeces of the sow and her narrow pelvis make the procedure less informative than in the larger farm species. The caudal genital tract, the bladder, the blood vessels within the pelvis, and sometimes the ovaries can be palpated. It may be possible to palpate the caudal pole of a grossly enlarged left kidney.

External genitalia in the boar

The testes should be of equal size and on palpation have the consistency of a ripe tomato (Fig. 1.16). Softening and reduction in size may be seen in cases of testicular degeneration. The skin covering the scrotum is often damaged by fighting injuries. Vasectomy wounds, if present, will be found in the inguinal region. The penis can be palpated within the prepuce, and the spiral anterior pole is found 10 cm from the anterior end of the prepuce. The sigmoid flexure of the penis in boars is anterior to the scrotum and palpable in thin boars subcutaneously between the hind legs. The presence of the large fluctuant prepucial diverticulum in boars has been mentioned above. Squeezing this swelling will mostly cause foul-smelling fluid to exude from the prepuce (Fig. 1.17). The diverticulum can easily be mistaken for an abscess or hernia, with dangerous consequences. The clinician is advised to wear gloves when handling this area. Bleeding from the prepuce may occur at or after service, and the erect penis should be examined at mating for evidence of wounds or other damage. Cases of suspected infertility are investigated as in the bull.

Fig 1.17 – Palpation of prepucial diverticulum.

The sow and her litter

When examining a sow with a litter of piglets in a farrowing crate, it is prudent to remove the piglets out of harm's way by barricading them in the creep area. The sow and her litter should always be considered as a single unit. Ill health in a sow rapidly leads to reduced milk production, with piglet starvation and hypoglycaemia. Continuous vocalization by the litter is an indication that the sow is unwell with a poor milk supply. Milk consumption is reduced in sick piglets and may predispose to maternal mammary disease.

Sow and piglet behaviour

Sows with litters may be aggressive and, unless in a farrowing crate, should be approached with great care. Access to the sow and her litter can be difficult on outdoor units. Sows in such units are visible at feeding time, and

their litters can be inspected at this time. Sows that farrow indoors are usually fed twice daily and should consume each meal within 10–15 min.

A quiet sow usually remains in lateral recumbency when approached. Massage of the udder and a quiet voice encourage her to remain in lateral recumbency, greatly facilitating examination. If disturbed, she usually rolls into sternal recumbency and gets to her feet. If nervous or aggressive, she may bark and snap. A very ill sow may require assistance by lifting her tail when rising and may utter a thin reedy squeal.

Healthy piglets spend much of their time in the warmth of their creep area between feeds. When hungry, they approach the sow, nuzzle her udder, and squeak impatiently to encourage her to roll on her side and feed her litter. Sows usually feed their litters at intervals of between 1 and 2 h throughout the day and night. Once stimulated (and sometimes spontaneously), the sow calls her litter to feed by making a low-pitched intermittent grunting sound. Piglets asleep in the creep usually respond to this and should be watched as they leave the creep. Some individual normal piglets may be deeply asleep and fail to hear and respond to their mother's call. Signs of piglet lameness may be observed as they come to feed. Scouring and signs of rhinitis may be seen at this stage.

In response to their mother's call, the piglets vocalize more loudly and seek their own teat. The larger, stronger piglets are usually found on the pectoral teats. In response to the piglets' voices and their nuzzling, the sow lets down her milk. She emits a rapid, intermittent, rather contented grunting sound, 'talking to her pigs'. As soon as milk let-down commences, the piglets cease to vocalize, hang back on their teat, and drink voraciously. Milk let-down lasts for only 60–90 s. After feeding, piglets often nuzzle the sow's udder, sleeping near the sow or returning to their creep.

Unhealthy piglets may fail to reach their mother and find a teat. They rapidly become thin and may attempt to drink water or urine on the floor. Initially, they seem hyperactive and excessively vocal. Piglets are very prone to both hypothermia and hypoglycaemia, and without food and warmth they deteriorate rapidly. Fitting and coma may follow. If the piglets are scouring, pale yellow, green, or grey diarrhoeic faeces may be seen in the pen.

Position of the sow for physical examination

The sow may be in one of four positions: lateral recumbency, sternal recumbency, 'dog sitting' position, and standing. A resting quiet sow usually remains in lateral recumbency when approached. Massage of the udder and a quiet voice encourage her to remain in lateral recumbency; this greatly facilitates examination. If disturbed, sows usually roll into sternal recumbency and get to their feet. A sow in sternal recumbency or the dog sitting position can be assisted to stand by lifting with the tail. If nervous or aggressive, she may bark and snap after being forced to change position. The lateral and standing positions facilitate the examination. The standing position gives the best access for the examination. The examination is as described under *Physical examination of adult and growing pigs*.

Physical examination of piglets

Respiratory movements can be counted and assessed before handling.

Small piglets can be readily picked up for examination (Figs 1.18 and 1.19). When lifted by a hind limb, the piglet will often remain silent. Febrile piglets feel very hot when handled. Body temperature is taken per rectum in the normal way. Pulse is best recorded by cardiac auscultation and is in the region of 150 beat/min.

The skin

The skin is thin and readily damaged (and exposed to the risk of infection) by other piglets and contact with unsuit-

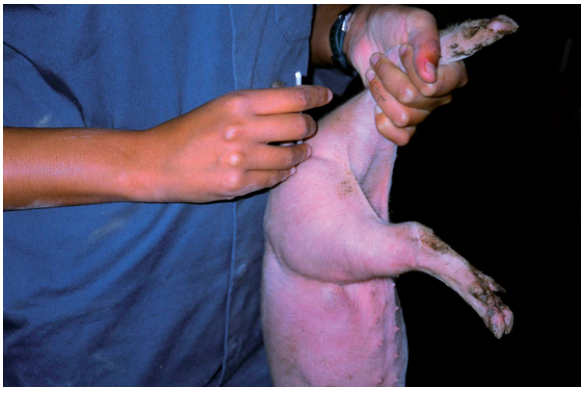

Fig 1.18 – Piglets can be lifted by one or both hind legs. They can be held like this without distress for short periods and seldom vocalize.

Fig 1.19 – Vocalization usually occurs if piglets are lifted with a hand round their thorax. Closing the piglet's mouth reduces the volume of sound.

able floor surfaces and walls. Teat and tail necrosis may result. Extensive lacerations and large haematomata can result from accidental damage by the sow. Dermatitis covering most of the body is caused by *Staphylococcus hyicus* in greasy pig disease. With dehydration, there is prolonged skin tenting when the loose skin behind the ear or on the ventral abdomen is pinched.

The head and neck

Bite wounds on the snout can result from damage by the sharp incisors of other piglets. The mouth should be checked to see if these teeth are present or whether there is infection in the gums following tooth clipping or necrotic lesions on the tongue.

The chest

Auscultation of the chest is difficult, especially if the piglet is vocalizing. In some cases of pericarditis, muffling of the heart and splashing sounds are audible. A stronger than normal apex beat may be palpable in cases of cardiac enlargement such as in iron deficiency anaemia. In cases of rhinitis, the loud lung sounds may be audible but may be referred sounds from the nose.

The abdomen

Distension of the abdomen is mostly caused by an imperforate anus. This is seen mostly in male piglets. In some females, a rectovaginal fistula is present. Inguinal and scrotal hernias may be present but are more obvious later in life.

Nipples

Damage and loss of nipples indicate poor floor covering.

The perineum

Diarrhoea in piglets is very common. Faecal colour may suggest which infection is present: *Escherichia coli* and *rotavirus*, yellow; *Clostridium perfringens*, dark and blood-stained; *transmissible gastroenteritis*, green and watery; and *iron deficiency anaemia*, pale and pasty.

Lameness: the limbs and feet

Lameness in piglets is very common and may result from crushing injuries or from damage caused by unsuitable flooring (Fig. 1.20). Superficial necrosis is common on the skin of the dorsal carpi and the caudal surfaces of the hocks, particularly on abrasive concrete floors. Joint ill – septic arthritis – may involve one or several joints and can quickly result in severe lameness, preventing affected animals reaching their sow to drink. If untreated, chronic damage to the joints occurs rapidly, with atrophy of disuse in associated muscle groups. Cracks and necrotic solar lesions may be caused by contact with metal mesh flooring and may affect the whole litter.

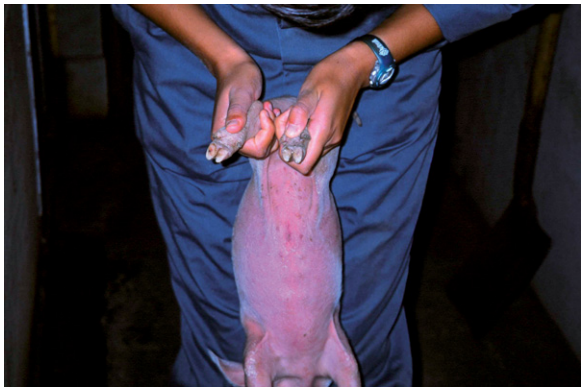

Fig 1.20 – Examination of a piglet for solar ulceration or bruising.

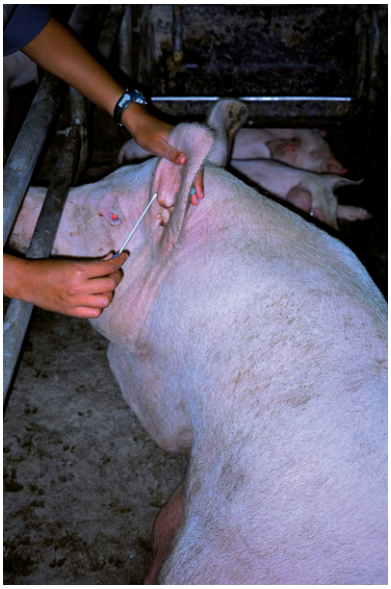

Fig 1.21 – Swabbing of the ears is well tolerated to investigate cases of suspected sarcoptic mange.

Tail necrosis

Loss of the tips of the tails and tail skin necrosis may be seen when adjacent walls are too close to the farrowing crate, causing frictional damage during suckling.

Further investigations

The decision to do further investigations should be influenced by the additional cost and how useful the results will be. Keeping the owner informed about the future cost benefits is a wise precaution.

Post-mortem examination can be of great diagnostic value in outbreaks of porcine disease. It may provide immediate confirmation of a tentative diagnosis and enables further samples to be taken for laboratory tests. Skin scrapings and samples of blood, faeces, urine, vaginal swabs, and earwax can be collected in the pig (Fig. 1.21).

Fig 1.22 – Collection of blood from a piglet. The piglet is restrained in dorsal recumbency. The Vacutainer needle is inserted into the anterior vena cava between the point of the shoulder and the manubrium of the sternum.

Often, a sow will urinate after she has stood up, enabling a urine sample to be collected. Ultrasonography and radiography can be useful in the pig but must be used with care on the farm. Feedback from slaughterhouse material can provide quantitative and qualitative disease information.

Blood sampling in pigs

The anterior vena cava, jugular vein, and ear vein are usually used when blood sampling. The milk vein, cephalic vein, and tail vein can also be used.

The anterior vena cava is used for pigs weighing 20 kg or less (Fig. 1.22). The animal is restrained in dorsal recumbency by holding the front and back legs in extension. A V-shaped trough is very useful for this purpose. The Vacutainer needle is inserted into the anterior vena cava between the point of the shoulder and the manubrium of the sternum. The needle is directed towards the heart, and the tube engaged to collect the sample.

The jugular vein can be used in larger pigs of 20 kg and above. The jugular vein is not visible in the pig. Large pigs are restrained using a snare around the upper canine teeth (Fig. 1.23). The point of insertion of the hypodermic needle is midway between the shoulder and the manubrium. The needle is directed upwards.

The marginal ear veins are usually identified in all ages of pigs but can more readily be used in larger pigs due to their larger size (Fig. 1.24). Good restraint is required, and using local anaesthetic gel before inserting the needle into the skin makes the technique easier. The vein can be raised by placing the hand across the base of the ear or by encircling the base of the ear with an elastic band. The walls of the veins are quite thin, and the needle sometimes slips out of the vein, with haematoma formation. Withdrawal of blood must be done very slowly to avoid the vein collapsing. Only small volumes are usually obtained. This site is useful for intravenous injections.

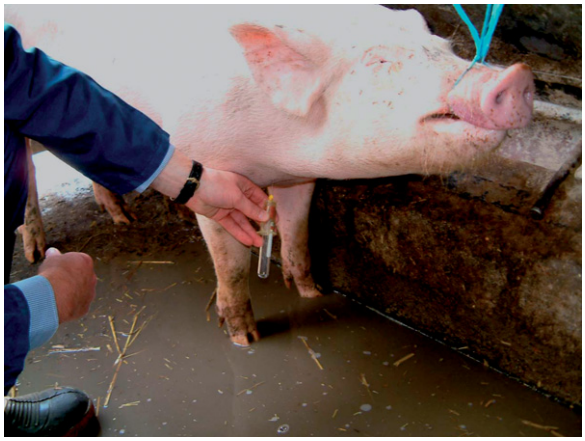

Fig 1.23 – Collection of blood from an adult pig, using a snare. The point of insertion of the hypodermic needle is midway between the shoulder and the manubrium.

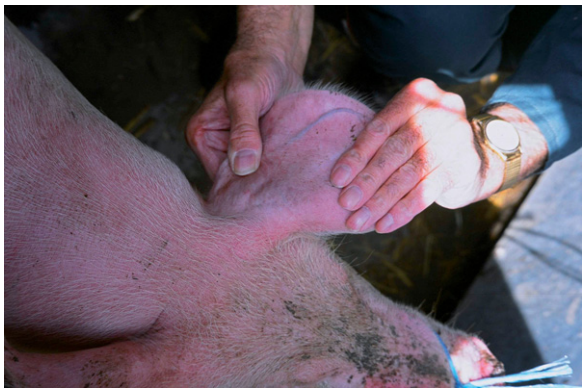

Fig 1.24 – The ear vein can be used for intravenous injections. It is less useful for blood sampling, because it readily collapses when negative pressure is applied.

The milk vein is visible lateral to the teats and travels subcutaneously on the lateral part of the abdominal musculature in growing pigs. Restraint is required, and local anaesthetic gel can be helpful to minimize movement. In sows and gilts, the vein can be identified and punctured between the mammary glands. Collection is sometimes possible without restraint in the standing or lying position.

The tail vein can usually be used in adult animals. The medial caudal vein lies in a groove under the tail, next to the artery. The operator raises the tail with one hand and punctures the vein with the other. Vacuum tubes and 20-gauge needles are used. The puncture site is at the first freely movable tail joint. This is around the fifth tail vertebra. In adult pigs, the needle should be inserted at an angle of 45° to the skin. In smaller pigs, it is recommended to hold the tail nearly horizontally and to stick the needle in nearly parallel to the skin.

Treatment, prevention, and control programmes

Once a diagnosis is made and the risk factors identified, appropriate treatment, control, and prevention programmes can be devised. These may include in-feed or water medication, management changes, modifications to buildings, and vaccination. For example, if a unit has an outbreak of pneumonia caused by *M. hyopneumoniae* and *Pasteurella multocida*, the following action plan may be suggested.

- Pigs: ensure the correct stocking densities, do not mix different ages or groups, keep less than 200 pigs in any one air space, and source pigs from one place.
- Buildings: optimize or improve the ventilation, and minimize dust and ammonia levels.
- Therapeutics: administer in-feed medication to control the pneumonia for a limited period until a vaccination programme is complete.
- Monitoring: lung scoring at slaughter.

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2

Population medicine

Introduction

Disease control and production systems

The greatest impact of the veterinarian on a pig unit in optimizing production and welfare is devising and implementing control and prevention programmes for important problems at a herd or group level. A basic understanding of the structure of pig production and how this relates to disease control is therefore necessary. There are essentially three stages in production.

- Stage 1: breeding, gestation, farrowing, and suckling.
- Stage 2: rearing of young weaned pigs in nurseries up to 25–35 kg.
- Stage 3: Growing and finishing to slaughter weight.

There is a range of production systems, which vary in the degree of physical separation of the three stages and the measures taken to reduce the transfer of disease between the different groups, ages, and classes of pigs. One-site production systems (farrowing to finishing) have all three production stages occurring at one site. In traditional two-site production systems, stage 3 occurs on a different site to stages 1 and 2. Some two-site production systems combine stages 2 and 3. In three-site production systems, all three stages occur at a different site.

Isowean systems (or segregated rearing) use the principle that piglets remain relatively free from common major pathogens endemic in a herd until after weaning. In this system, following weaning they are raised in isolated cohorts away from other pigs and other age groups on a separate site. They are likely to remain relatively free from common major pathogens.

In medicated systems using antimicrobials, sows are medicated prior to farrowing and during lactation. Piglets are treated with medication (antibiotics) until weaning. *Haemophilus parasuis*, *Pasteurella multocida*, *Bordetella bronchiseptica*, *Actinobacillus pleuropneumoniae*, and *Mycoplasma hyopneumoniae* have been minimized in this way provided that postweaning exposure is eliminated by rearing in isolation.

The all in, all out by site building or by room is an important protocol in disease control. The site, building, or room is populated on a single day and is depopulated in a day. This is cleaned, disinfected, dried, and left empty for up to 1 week before repopulating. Each batch of pigs remains as a single batch from weaning to market. The pigs are never mixed with older infected pigs.

Hysterectomy-derived piglets that are subsequently reared in isolation from the sow to maintain their specific pathogen-free status can be used to produce a minimal disease herd (high-status herd) that can be categorized as free from specific pathogens (specific pathogen-free). This system prevents the pigs becoming infected from the sow.

Disease control in breeding organizations is given very high priority, and the structure is often described as a pyramid structure with the nucleus (high health status, high genetic merit) herds at the top, followed by the multiplier herds and then the commercial herds. Movement is very strictly controlled within the pyramidal structure, with the flow of pigs (contact) down the pyramid, never up it, in order to protect the relative health status of the herds.

Outdoor units are usually limited to stage 1: breeding, gestation, farrowing, and suckling. Weaned pigs are usually reared intensively.

A review of the economic effects of disease on pig performance

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Summary

Pig performance from weaning to slaughter did not improve throughout the 1990s. Primarily, this was due to the introduction of porcine reproductive and respiratory syndrome (PRRS) virus and swine influenza virus into the UK pig populations. Before the industry could recover, postweaning multisystemic wasting syndrome (PMWS) and epidemic porcine dermatitis and nephropathy syndrome (PDNS) arrived in 1999 and spread throughout the country, causing performance to deteriorate further. The effects of the disease on daily live weight gain (DLWG), food conversion efficiency, mortality, and drug usage are

discussed and where possible quantified. Possible remedial measures are identified.

Introduction

Health and nutrition are the two major factors governing physical and economic performance of pigs from weaning to slaughter. With good nutrition and in the absence of major disease, good DLWG is achieved with improved feed conversion efficiency (FCR).

Prior to 1990, establishing and maintaining a high health status herd was relatively straightforward, and maintenance of this high health status was achieved with good biosecurity. At that time, specific diseases were controlled by use of antibiotics and vaccines where appropriate and where available. The introduction of PRRS virus into the UK in 1991 (Paton et al. 1991, White 1991) presented the British pig industry with a new problem. Antibiotics were of little use, and no vaccine was available. New management techniques were adopted to control this serious viral disease. All in, all out strategies coupled with three-site production and early weaning, with or without medication, became commonplace and afforded reasonable control of the disease. The health status of the UK pig population stabilized and was not seriously disturbed again until 1999, when PMWS was introduced into England (Potter 2000). This disease syndrome has had a prolonged and devastating impact on both the physical and the economic performance, from which the industry has not yet recovered. The outbreaks of classical swine fever in East Anglia in 2000 and of foot and mouth disease in 2001 have added fuel to the fire and left the future viability of the British pig industry balancing on a knife edge.

Physiological effects of disease on performance

The presence of disease is associated with either weight loss or lowered rate of weight gain, depending on severity plus reduced FCR. Energy and protein required to mount an immune response are directed away from muscle growth (Spurlock 1997, Dial 2002). The challenge to the immune system will induce a number of effects.

- Immunological challenge will cause a decrease in insulin-like growth factor (Hevener 1999). This factor is closely associated with animal growth rates and ultimate body size.
- Cytokines produced as part of the immune response suppress secretion of growth-promoting hormones (Klasing 1998).
- Decreased feed intake or anorexia due to pathogenic infection and immune system activation.
- The metabolic response to infection causes nutrients to be directed away from tissue growth in support of immune function.
- Concurrent subclinical infections will create cumulative effects, which may significantly worsen performance.

These physiological pathways are summarized in Figure 2.1.

Whatever the disease, in whatever body system, the physiological effects are the same, varying only in severity. Prior to 1999, the major disease syndromes adversely affecting pig performance from weaning to finish were respiratory disease (notably PRRS and enzootic pneumonia) and enteric disease, with a few miscellaneous conditions contributing on occasion. In the second half of 1999, PMWS and associated epidemic PDNS were diagnosed for the first time in the UK (Potter 2000) and have blighted pig performance ever since. Each of these syndromes will be considered and the individual and combined effects on performance assessed. Disease in a finishing herd is expressed by reductions in DLWG, decreased FCR, raised mortality, increased drug cost, and uneven growth (see below).

Expression of disease:

- increased DLWG,
- increased FCR,
- increased mortality,
- increased drug cost, and
- increase in uneven pigs.

Respiratory disease

There are three compartments to the respiratory tract, namely upper, middle, and lower. The upper respiratory tract comprises the nostrils, nasal cavity, sinuses, and nasopharynx, and disease of these areas is associated with sneezing. The middle compartment includes the larynx, trachea, and main bronchi. Disease affecting these areas is associated with coughing. The lower respiratory compartment is the bronchiolar and alveolar region, and disease of this area is associated with dyspnoea (Done et al. 1994). The major respiratory pathogens commonly affecting growing pigs in the UK are identified in Table 2.1.

Prior to 1990, clinical respiratory disease invariably was associated with a single pathogen, which could be

Table 2.1 Respiratory tract pathogens of the pig

Type	Pathogen
Bacteria	*Mycoplasma hyopneumoniae*
	Actinobacillus pleuropneumoniae
	Haemophilus parasuis
	Pasteurella multocida
	Streptococcus suis
	Mannheimia haemolytica
	Bordetella bronchiseptica
Viruses	Porcine reproductive and respiratory syndrome virus
	Swine influenza
	Porcine circovirus 2
Helminths	Ascarid larval migration

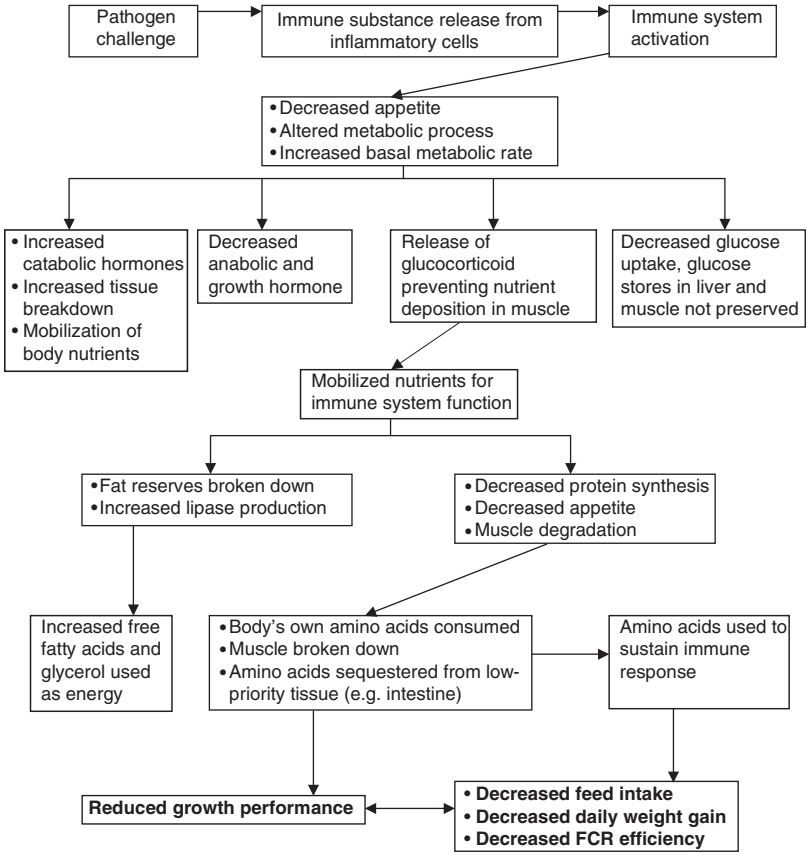

Fig 2.1 – Summary of physiological pathways. FCR, food conversion rate.

identified fairly readily and treated. If the primary agent was viral or a mycoplasma, secondary bacterial infection occurred commonly but could usually be controlled by use of an antibiotic. In the early 1990s, PRRS descended on the pig world and revolutionized the disease situation in the UK pig population, precipitating a new respiratory syndrome, which became known as the porcine respiratory disease complex (PRDC). PRRS so changed the health environment that it was not long before swine influenza in all its subtypes entered the arena and added to the severity and complexity of porcine respiratory disease. The clinical signs and therefore economic impact of PRDC depends on a number of factors, including nutrition, management, immune status of the population, strains of primary virus(es), and complexity of secondary bacterial invaders. Morbidity is always high, and mortality may be high also.

The economic impact of respiratory disease on any individual unit will depend not only on the clinical severity of the disease but also on current cost of production figures. These vary widely from farm to farm. Feed cost per kg live weight gain can vary from 24 to 40 p per pig. Mild respiratory disease will result in decreased feed intake, decreased DLWG, deteriorating FCR, and the necessity to sell pigs at a lower finished weight. A hidden bonus of mild respiratory disease is a reduction in back fat (P2) of the finished carcass. This observation was confirmed when efficient enzootic pneumonia vaccines became available. Good control of enzootic pneumonia by judicious use of vaccine increased P2 measurement by 1–2 mm per pig initially until diets were reformulated.

For every 10% of lung tissue affected by pneumonia, DLWG is reduced by 22–37 g/day. Studies in Germany reveal a reduction in DLWG of 34–50 g/day in pigs with lung lesions compared with pigs with healthy lungs (Klawitter et al. 1998). In Denmark, a combination of enzootic pneumonia and *Actinobacillus pleuropneumoniae* resulted in a growth reduction of 30.3–58.8 g/day (Baekbo et al. 2002).

It has been demonstrated that clinical PRRS in growing and finishing pigs can reduce DLWG by up to 492 g/day in the initial insult and to a lesser degree in the following days (Greiner et al. 2000). Despite this being an experimental situation, the devastating impact of this infection on feed intake and DLWG is echoed in clinical situations seen on farms following PRRS infection.

When PRDC becomes severe, mortality rate and cull rate rise significantly and DLWG can be reduced by up to 50%, at least doubling daily financial losses. During the 1990s, the industry and the veterinary profession began to get to grips with this new disease scenario. New management strategies such as medicated early weaning, segregated early weaning, and all in, all out – coupled with the new efficient enzootic pneumonia vaccines – brought some semblance of control to PRDC. However, in 1999 PMWS arrived in the UK. Although it moved around the country fairly slowly, over the next 2 years the health of the UK pig population deteriorated once more, with disastrous economic consequences.

PMWS–PDNS

Porcine dermatitis and nephropathy syndrome was recorded in the UK by Smith et al. (1993) and White and Higgins (1993). Between 1993 and 1998, there have averaged nine recorded outbreaks of this condition in the UK (Gresham et al. 2002), with low morbidity and mortality. The disease was christened sporadic PDNS.

In 1999, PMWS was diagnosed for the first time in England in association with acute PDNS (Potter 2000). PMWS is believed to be associated with porcine circovirus type 2 (Ellis et al. 1998), but the relationship between PMWS and PDNS is still unclear. Many veterinarians working in the field believe that the causal organism for PMWS is still to be identified.

Postweaning multisystemic wasting syndrome and associated epidemic PDNS are now widespread throughout the UK, and over the past 5 years this disease syndrome has devastated the British pig industry, with high mortality, often up to 25%.

Economic losses have frequently exceeded £10 per pig (Done 2002). In severe outbreaks, incurred costs have added 15 p/kg to the cost of production of a finished pig.

The mode(s) of transmission of the causal agent has not been identified, and the disease does not appear to stimulate herd immunity. Control measures revolve around strict attention to hygiene, good nutrition, stable social groups, and all in, all out housing policies. Shortage of labour on many farms has made efficient implementation of these policies difficult, and this disease syndrome is one of the major reasons why the British pig industry is currently selling 17.4 finished pigs/sow per year (Thames Valley Cambac, personal communication, 2004).

Enteric disease

Prior to PMWS, approximately 70% of disease costs could be related to alimentary and respiratory disease (Done 1999). Disease of the alimentary tract can be broadly divided into small intestinal diseases (enteritis) and large bowel disease (colitis).

Small intestinal disease most commonly occurs within 10 days of weaning and is usually associated with presence of toxigenic *Escherichia coli*, sometimes complicated by *Salmonella* species infection. The clinical expression of disease and severity of disease depend not only on infection but also on housing, nutrition, and hygiene. Up to 50% of all weaned pigs may be affected, and mortality levels may reach 10%. Even when diarrhoea does not persist for very long, there can be a significant reduction in DLWG and some animals may be permanently stunted due to chronic intestinal scarring and villous atrophy. Optimum feed intakes after weaning maximize DLWG and FCR. Severe postweaning diarrhoea therefore can increase cost of production by between 1 and 2 p/kg.

Since the development of PMWS, enteritis has become more common in older pigs, frequently associated with *Salmonella* infection. Diarrhoea is a common clinical feature of PMWS, but the cost implications have already been discussed. Inflammation of the large bowel is known as colitis, while inflammation of the terminal part of the small intestine is known as ileitis. Proliferative enteropathy is a specific disease of the ileum, frequently spreading into the colon and caused by an intracellular organism known as *Lawsonia intracellularis*. An investigation into field cases of porcine colitis on 85 pig units in the UK (Thomson et al. 1998) identified *Brachyspira pilosicoli*, *Yersinia pseudotuberculosis*, *Lawsonia intracellularis*, *Salmonella typhimurium*, and *Brachyspira hyodysenteriae* as the major pathogens. There was a combination of pure and mixed infections. Despite successful treatment of batches of pigs, colitis persisted as a chronic problem on many units, with diarrhoea and body weight loss the main clinical features. Ileitis and colitis tend to develop in pigs ranging between 20 and 40 kg, with an estimated prevalence of 5 and 15%, respectively. Mortality rate varies widely, from 1 to 25% (Taylor 1995). In recent times, there has also been an indication that several enteric conditions may be similarly implicated in a clinical situation, the enteric equivalent of PRDC. This has increased cost in terms of both the diagnostic testing and the treatment required.

Ileitis cost can vary from £0.92 per pig to £13.87 per pig depending on severity (Lawrence 1999). Colitis associated with *Brachyspira* species infection is estimated to reduce FCR by between 0.05 and 0.2, increasing time to finish by up to 3 weeks. Again, the presence of PMWS has had an adverse impact on the prevalence of colitis, possibly aggravated by the compulsory ban on some antibiotic growth promoters in July 1999. However, the total ban on antibiotic growth promoters that came into force in January of 2006 has had less of an impact than initially feared, possibly due to the degree of preparation, initiated by both the pharmaceutical and the feed companies. Therefore, it is likely that the economic impact of ileitis and colitis has increased since 1999, but there is no

published work to confirm this other than the estimations of the cost of PMWS highlighted previously.

Miscellaneous conditions

Sarcoptic mange

The most significant ectoparasite of the growing and finishing pig in the UK is *Sarcoptes scabiei* var. *suis* (White 1994). The disease has a serious economic effect on growing pigs as well as welfare implications. The disease increased in incidence during the 1990s with the advent of chronic respiratory disease associated with immunosuppressant viral infection such as PRRS and swine influenza. A survey of slaughter pigs in the UK in 1990 identified a 70% incidence (McMullin et al. 1992). Cargill and Dobson (1979) reported experimental disease as producing a reduction in DLWG and FCR of between 9.2 and 12.5%, while Gaafer et al. (1986) reported that successful control of the disease produced a 5.5-kg improvement in slaughter weight and a 0.1 improvement in FCR. The authors' personal experience confirms the economic importance of this disease.

Intestinal parasites

Ascaris suum, the pig ascarid, currently constitutes the major economically significant nematode in the UK (White 1994). As indicated elsewhere in this review, *Ascaris suum* must be considered in the differential diagnosis of respiratory disease but also causes high levels of liver condemnations at the abattoir and depressed DLWG and FCR in heavily infested herds. Both growth rate and feed efficiency may be depressed by up to 10%.

In view of the highly resistant nature of the ascarid egg and the move to more extensive housing systems with solid floors, infection with this parasite will remain highly significant for the foreseeable future.

Meningitis

While clinical meningitis can be caused by several bacteria and viruses, the commonest cause of meningitis in growing pigs in the UK is *Streptococcus suis* type 2 (Done et al. 1998). There are 35 known serotypes of *Streptococcus suis*, and many can cause outbreaks of septicaemia or meningitis in growing pigs (Sandford and Higgins 1992, Higgins et al. 1995). Infections can occur at any age, but most cases are in piglets between 3 and 12 weeks, although pigs of finishing weights may be affected.

Clinical incidence of the disease varies between 1 and 10%, with up to 3% mortality. Segregated early weaning and medicated early weaning technologies do not eliminate *Streptococcus suis* infection in weaned pigs, because pigs are infected during birth or the first few hours of life (Robertson and Blackmore 1989) and because prophylactic use of antibiotic does not eliminate the carrier state (Amass et al. 1996). Consequently, *Streptococcus suis* is responsible for a significant portion of the treatment costs associated with raising high health status pigs.

Arthritis

Arthritis is common in the growing pig. The most common infections of joints are erysipelas, *M. hyosynoviae*, and *Streptococcus suis*. Osteochondrosis is the most common non-infectious cause of arthritis. Clinical incidence ranges from 1 to 5% normally, but in outbreaks of erysipelas or *Streptococcus suis* incidence may rise to 10%. In such circumstances, arthritis becomes an economically significant disease resulting in an increased culling rate, increased treatment costs, and increased condemnation rate at the abattoir.

Discussion

As with all other types of farming, pigs are farmed for profit, hopefully. Profit is derived from the margin between the price received for the finished carcass and the costs incurred to produce that carcass. The major cost is feed, and up to 75% of total feed purchased is used from weaning to slaughter. Presence of disease adversely affects feed intakes and efficient utilization of feed. The physiological pathways responsible for this are highlighted in Figure 2.1. The pathogen load a unit carries is initially related to the health status of the purchased breeding stock, and subsequently is affected by the proximity of other pig farms, unit biosecurity, stockmanship, housing, hygiene, and the production system on the farm.

Even before PMWS–PDNS, respiratory and enteric disease could reduce unit performance by up to 50% of the genetic potential of the animal (Kingston 1999). During the period 1990–1998, there was no improvement in feeding herd performance in terms of DLWG and FCR, and over the same period mortality rose (Anonymous). Since that time, PMWS–PDNS has appeared and physical performance has deteriorated still further.

The emergence of PRRS and swine influenza viruses in the 1990s made the UK pig population more susceptible to the other infectious diseases identified in this chapter. The subsequent emergence of PMWS and epidemic PDNS in 1999 has made the situation much worse, and the UK pig industry has not yet recovered. In addition, enteric disease incidence was aggravated by the ban on certain growth promoters in July 1999, although a total ban on these products in 2006 had less impact. During the 1980s and 1990s, use of growth promoters gave an increased income of £2.50 per pig (Lawrence 1998). The compulsory reduction in copper inclusions in pig feeds in 2004 has resulted in increased incidence of loose faeces, and the authors have recorded an average reduction in DLWG of 34 g/day, with a maximum recorded deterioration of 70 g/day. The economic impact of disease on any individual unit will depend not only on the severity of the disease but also on current cost of production figures. A 0.1 deteriora-

tion in FCR costs between 75 and 90 p (BOCM Pauls, personal communication, 2004), while a reduction of DLWG of 20 g/day costs between £1.00 and £1.50 per finished pig.

To combat the costs of general background pathogen challenge and PMWS, many farmers have adopted all in, all out stocking policies coupled with strict hygiene and disinfection procedures. All have experienced some benefit, but on those farms where the individual buildings were close together improvement has been modest, barely compensating for increased costs incurred. In the authors' experience, the most cost-effective management change has been a partial depopulation of the unit with thorough cleansing and disinfection, and weaning off site for a period of 4–6 weeks with a breeding herd medication programme during that time specifically designed to eliminate pathogens endemic to the unit. Such programmes have reduced time to slaughter by 2 weeks and given a cost of production benefit of £7.50–£10.00 per pig. Two years after completion of the programme, many producers still have empty finishing pens due to improved physical performance.

These programmes not only demonstrate the benefits of health – they highlight, very clearly, the true cost of disease.

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Biosecurity

Biosecurity can be defined as the outcome of all activities undertaken by an entity to preclude the introduction of disease agents into an area that one is trying to protect.

When a farm or site is affected by disease, the impact can be devastating on the health of the pigs and the economics of production. A good biosecurity programme helps to reduce the risk of pathogens being introduced or transferred from the farm. Some of the recommendations will not be applicable to all farms. Risks need to be identified and protocols devised to minimize these risks. Hazard analysis of critical control points is a useful framework for identifying risk and actions that will reduce the risk. Biosecurity protocols may have to change as the risk of new and emerging diseases entering the UK increases or becomes recognized. The National Pig Association and the British Pig Executive (BPEX) are in the process of developing a self-assessment biosecurity form for pig keepers. A guide to biosecurity is available on the US National Pork Board web site.

There are considerable benefits to the producer, public health, and also the veterinary surgeon of improving biosecurity protocols. These include:

- improved animal welfare;
- improved production, productivity, and profit;
- increased value of the herd;
- decreased use of medicines and antibiotic resistance;

- increased potential for export;
- refining of the role of the veterinarian from 'firefighter' to 'health manager'; and
- enhanced herd health programmes.

There are seven basic principles for the prevention and control of infectious diseases.

1. Ideally, maintain a closed herd with no introduction of animals and no contact with outside animals.
2. If new introductions are unavoidable, isolate, test, and treat incoming animals for targeted important diseases.
3. Control visitors and reduce the risk of transmission of pathogens on motor vehicles.
4. Control and prevent direct and indirect contact with wildlife and other domestic species.
5. Ensure that feed and water are free of contamination.
6. Define and monitor the herd's health status.
7. Establish an agreed disease control programme including biosecurity protocols.

Biosecurity protocols should be evidence-based. The tests and examinations that are required should be selected on the basis of evidence that the disease under consideration is of economic significance to the farmer. A risk assessment of the disease should be made. Will the effects of the disease establishing itself within the herd be so great that the costs of testing, treatments, and vaccination are worthwhile? The test implemented must be chosen and the results interpreted with an understanding of the limitations of the test (sensitivity, specificity, and disease prevalence).

When introducing new animals to the herd and considering target disease, attempts should be made to establish the following:

- the disease status of the resident herd,
- the disease status of the farm of origin,
- the clinical signs of the disease,
- the epidemiology and transmission of disease,
- the diagnostic tests available and their parameters (sensitivity and specificity),
- the vaccines available and their efficacy,
- prophylactic treatments and their efficacy, and
- the economic importance and the potential risk (prevalence) of the disease.

Replacement and new stock (gilts, boars, semen, and embryos)

Incoming infected stock is the greatest risk to herd health from external pathogens through direct contact. They may be incubating or carrying disease pathogens. Isolation and quarantine enables observation, testing, treatment, and vaccination of newly acquired animals to reduce this risk.

Ideally, the isolation facility would be at least 3 km away from other pigs, although this is practically impossible in most cases. A distance of 300 m is a suggested minimum. The isolation unit should be totally enclosed to minimize the risk of aerosol and wildlife spread, with an all in, all out pig flow with cleaning, disinfection, and a break time of 7 days between batches. The duration of quarantine suggested ranges from a 30-day minimum to an ideal in excess of 60 days (enzootic pneumonia). The risk of pathogen spread by the workers responsible for the replacements in isolation should be minimized. This can be achieved by using workers who have no other contact with pigs. If this is practically impossible, then strategically organizing the working day to minimize the potential transfer of pathogens from the replacements to the resident herd is usually feasible. The risk can further be reduced by the use of showering, isolation unit-dedicated clothing, and disinfection procedures. The design of the building should ensure that it can be easily cleaned and disinfected with appropriate effluent and drainage systems.

In order to perform a disease risk assessment of incoming animals, the recipient herd veterinarian should communicate with the source herd veterinarian regarding the health status of the herd and the evidence supporting it. This is sometimes called replacement profiling. Information requests should include records of clinical disease, performance records, serological monitoring results, necropsy findings, and abattoir monitoring results from the British Pig Health Scheme (BPEX), and the *Salmonella* (zoonosis action plan, ZAP) enzyme-linked immunosorbent assay (ELISA) testing of meat juices. Disease status of the recipient herd should also be established so that quarantine protocols are appropriate.

The animals in quarantine should be tested, treated, and vaccinated for targeted diseases, for example ear swabs and skin scrapes for sarcoptic mange, faecal samples for *Salmonella* bacteriological screening, and serology for PRRS virus. Daily detailed observations should be made to identify clinical signs of disease. All sick animals in the isolation facility should be thoroughly investigated, including post-mortem examinations if required. Blood testing and vaccination programmes should be coordinated to avoid confounding results. Statistical approaches can be used to identify a reliable number of animals to test given a nominated prevalence and the sensitivity and specificity of the test for a targeted disease. Alternatively, all animals can be tested. All results should be obtained and interpreted before the replacements are introduced into the recipient herd.

Many pathogens, including parvovirus, PRRS virus, *Brucella*, Aujeszky's disease, and classical swine fever, have been isolated from the semen of infected boars. Semen should ideally be purchased from accredited

Fig 2.2 – Farm site location: isolation will improve biosecurity. (Courtesy of D. Chennells.)

Fig 2.3 – Muck spreading may increase the risk of infection to other livestock and outdoor pigs. (From Pritchard et al. (2005), with permission.)

Table 2.2 Distance of possible aerial spread of some common swine pathogens

Disease agent	Possible distance spread
Actinobacillus pleuropneumoniae, *Pasteurella multocida*, *Haemophilus parasuis*, *Mycoplasma hyosynoviae*, *Streptococcus suis*, porcine reproductive and respiratory syndrome virus, postweaning multisystemic wasting syndrome	Short distances: up to 1 km
M. hyosynoviae, swine influenza virus	Intermediate: 1–10 km
Parvovirus, Aujeszky's disease, foot and mouth disease	Long distance: >10 km

(Courtesy of Dr J. Carr, Iowa State University, Ames.)

Fig 2.4 – Perimeter fences and gates should be clearly signed. (From Pritchard et al. (2005), with permission.)

disease-free sources. Embryos have also been associated with porcine PRRS virus and classical swine fever.

Herd location and perimeter security

In order to minimize the risk of spread between farms, a distance of 3 km between holdings is suggested (Fig. 2.2). This is the usual recommendation for high-status herds, although 500 m has been suggested as an absolute minimum for commercial herds if there is no alternative. Similarly, it is suggested that the farm should be sited at least 500 m from a public road. The level of risks will be affected by the direction of prevailing winds and the type, density, biosecurity, and drainage of units in the vicinity. Slurry spraying may increase aerosol or airborne spread (Fig. 2.3). Aerosol transmission of *M. hyopneumoniae*, Aujeszky's disease, and foot and mouth disease is 3 km or more. Table 2.2 indicates the potential danger of aerial spread of selected pathogens, and Table 2.3 summarizes the possible transfer routes of the major pathogens of the pig. There should be deterrents to avoid unwanted access: the presence of a boundary fence, a single entrance, warning notices, and locked gates. Cars should be parked outside in demarcated areas; entrance gates and doors should be locked and a poster with protocols for visitors clearly visible. Feed delivery entrances and loading bays should be sited on the perimeter fence, which should otherwise be secure (Fig. 2.4). Loading areas outside should be outside the fence.

Wildlife

Wildlife (birds, rodents, feral cats, and wild boars) may act as vectors of pig diseases (Figs 2.5–2.8). There should be an ongoing programme of pest control and secure storage of food. Contact with wildlife should be minimized by the use of proofed housing. Outdoor pig units should be aware of the increased risk. Rodents can carry pathogens responsible for atrophic rhinitis, *Escherichia*

Table 2.3 Possible transfer routes of the major pathogens of the pig

Pathogen	Other pigs	Pork products[a]	Knacker[b]	Transportation systems	Locality of neighbouring pig units	Presence of a major road	Purchased second-hand equipment	Clothing from another unit	Birds, rodents, cats, dogs, flies	Feed and water	Bedding and straw[c]	Staff owning their own pigs	Staff visiting pig markets, shows, and slaughterhouses	Veterinarians and other advisers	Visitors[d]	New utensils
Actinobaculum suis																
Actinobacillus suis																
Actinobacillus pleuropneumoniae																
African swine fever																
Arcanobacter pyogenes																
Ascaris suum																
Aujeszky's disease, pseudorabies																
Bordetella bronchiseptica																
Borrelia spiralis																
Brachyspira hyodysenteriae																
Brachyspira pilosicoli																
Brucella suis																
Classical swine fever																
Circovirus 1 and 2																
Clostridium difficile																
Clostridium perfringens																
Congenital tremor virus?																
Cytomegalovirus																
Escherichia coli cystitis																
Escherichia coli diarrhoea																
Escherichia coli bowel oedema F18 Ste2x																
Enterovirus																
Epidemic diarrhoea virus																
Eperythrozoon suis																

Table 2.3 Possible transfer routes of the major pathogens of the pig—cont'd

Pathogen	Other pigs	Pork products[a]	Knacker[b]	Transportation systems	Locality of neighbouring pig units	Presence of a major road	Purchased second-hand equipment	Clothing from another unit	Birds, rodents, cats, dogs, flies	Feed and water	Bedding and straw[c]	Staff owning their own pigs	Staff visiting pig markets, shows, and slaughterhouses	Veterinarians and other advisers	Visitors[d]	New utensils
Erysipelothrix rhusiopathiae							■	■				■	■	■		
Foot and mouth virus and other vesicular viruses		■					■	■				■	■	■		
Haemophilus parasuis					■											
Haematopinus suis	■		■	■			■	■				■	■	■		
Hyostrongylus rubidus			■	■			■	■			■	■	■	■		
Isospora suis	■		■	■			■	■				■	■	■		
Lawsonia intracellularis			■	■			■	■				■	■	■		
Leptospirosis	■				■	■			■	■	■				■	
Metastrongylus apri			■	■			■	■			■	■	■	■		
Mycoplasma hyopneumoniae																
Mycoplasma hyosynoviae																
Oesophagostomum dentatum			■	■			■	■			■	■	■	■		
Parvovirus	■		■	■	■	■	■	■								
Pasteurella multocida (toxigenic)											■					
Pasteurellosis	■		■	■	■	■	■	■	■	■	■	■	■	■	■	
Postweaning multisystemic wasting syndrome																
Porcine reproductive and respiratory syndrome virus									■	■	■					
Ringworm	■		■	■	■	■	■	■	■	■	■	■	■	■	■	
Rotavirus	■		■	■	■	■	■	■	■	■	■	■	■	■	■	
Salmonellosis	■	■	■	■	■	■	■	■	■	■	■	■	■	■	■	
Sarcoptes scabiei	■		■	■	■		■	■				■	■	■		
Spirochaetal colitis			■	■	■				■							
Staphylococcus hyicus	■		■	■	■	■	■	■	■	■	■	■	■	■	■	

(continued)

Table 2.3 Possible transfer routes of the major pathogens of the pig—cont'd

Pathogen	Other pigs	Pork products[a]	Knacker[b]	Transportation systems	Locality of neighbouring pig units	Presence of a major road	Purchased second-hand equipment	Clothing from another unit	Birds, rodents, cats, dogs, flies	Feed and water	Bedding and straw[c]	Staff owning their own pigs	Staff visiting pig markets, shows, and slaughterhouses	Veterinarians and other advisers	Visitors[d]	New utensils
Stephanurus dentatum																
Streptococcus abscess																
Streptococcus arthritis																
Streptococcus suis joint ill																
Streptococcus suis meningitis																
Strongyloides ransomi																
Swine influenza virus																
Swine pox virus																
Transmissible gastroenteritis																
Toxoplasma gondii																
Trichinella spiralis																
Trichuris suis																

Light shading: possible pathogen transfer routes.
Dark shading: usually faecal contamination.
[a]For example, ham, salami, sausage, and pizza.
[b]Placement of dead pig disposal area.
[c]Note source of manure for straw.
[d]Note electricity and gas service people.

Fig 2.5 – Outdoor pigs with direct and indirect contact with birds. (From Pritchard et al. (2005), with permission.)

Fig 2.6 – Seagulls in close proximity to outdoor pigs. (From Pritchard et al. (2005), with permission.)

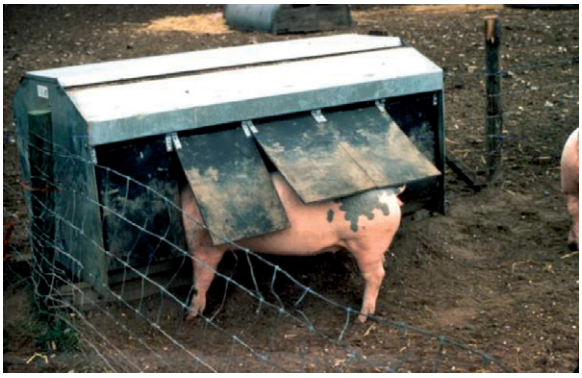

Fig 2.7 – Flaps on feeding troughs can reduce the contamination of the feed. (From Pritchard et al. (2005), with permission.)

Fig 2.8 – Wild boars may escape or may be maliciously released and become feral. (From Pritchard et al. (2005), with permission.)

coli scours, leptospirosis, rotavirus diarrhoea, *Lawsonia intracellularis*, salmonellosis, and swine dysentery. Dogs can spread swine dysentery. Wild animals can carry leptospirosis and Aujeszky's disease. Feral cats are potential source of toxoplasmosis and salmonellosis for pigs. Birds (starlings and gulls) may also transmit *Bordetella*, salmonellosis, avian tuberculosis, classical swine fever, PRRS, influenza, and transmissible gastroenteritis. Flies may act as mechanical vectors of transmissible gastroenteritis, *Streptococcus suis*, and PRRS virus. Feral wild boar are carriers of classical swine fever in some European countries. Hedgehogs, foxes, and rats can carry some *Leptospira* serovars (e.g. *Leptospira bratislava*) that can infect in-contact pigs.

Feed, bedding, and water

Salmonella can be introduced with contaminated feed. External feed supplies should be sourced from mills operating in accordance with relevant Department for Environment, Food and Rural Affairs (Defra) and agricultural codes of practice that ensure adequate biosecurity

Fig 2.9 – Loading areas should be sited at the perimeter fence. (From Pritchard et al. (2005), with permission.)

and quality control. This includes using ingredients obtained from sources with a satisfactory bacteriological record. Storage containers on farms should ensure that contamination by wildlife is avoided. Ideally, mains water should be used or periodic bacteriological testing of borehole water. It had been suggested that disinfectant tablets at appropriate dose rates could be used in feeder tanks to increase the quality of the drinking water. Straw and shavings should be free from contamination, sourced with care, and stored correctly to prevent contamination.

Transportation

Farm transport should be washed, disinfected, and allowed to dry after every load and should use a perimeter fence loading–unloading bay (Fig. 2.9). The same applies when using contract hauliers. Dirty trucks and trailers should be rejected.

Vehicles can potentially transmit pathogens when manure adheres to the tyres or the vehicle frame. There is evidence that *Actinobacillus pleuropneumoniae*, transmissible gastroenteritis, and *Streptococcus suis* can be spread in this way. Classical swine fever and foot and mouth disease have been spread from farm to farm by contaminated vehicles. Vehicles from other farms, markets, and slaughterhouses present an increased risk of introducing infection. Cleaning and disinfecting should take place off site on a hard standing. Wheel arches and the undersides of vehicles need particular attention (Fig. 2.10). Feed lorries should make deliveries outside the perimeter fence to reduce the risk. Protocols are needed for disposing of packaging such as cardboard and for office deliveries. Livestock loading and unloading should take place at a purpose-built loading bay with good washing, disinfecting, and drainage facilities far removed

Fig 2.10 – External cleaning and disinfection are important to reduce contamination by faecal material. (From Pritchard et al. (2005), with permission.)

Fig 2.11 – Internal cleaning and disinfecting will reduce the risk of cross-infection. (Courtesy of D. Chennells.)

from other pigs and close to the perimeter fence (Fig. 2.11).

Employees, visitors, tools, and equipment

Ideally, employees and visitors will not have any other contact with pigs. If there is contact, there should be a protocol regarding a time gap before returning to work. A pig-free days policy for visitors (veterinarians) usually specifies 48–72 h for nucleus herds and high health status herds. Showering in, change of clothes, and masks may be requested in addition to the prescribed down time. With commercial herds, 12 h (overnight) may be a reasonable time gap provided that there is a change of clothes, hand washing (including fingernails), hair washing, showering, and nose blowing. Decontamination of vehicles used to visit different farms is essential. Farm-dedicated clothing and boots should be provided along with appropriate changing rooms. Visits should be prioritized so that visits to high health status herds precede those to herds further down the company pyramid.

All visitors' vehicles should be parked in a well-marked dedicated parking area outside the entrance. Visitors ideally should shower in and out, wear clean clothing provided by the farm, and have a break time between visits to pig farms. A visitors' log should be kept that includes all relevant details including the date of the last contact with pigs. Hats, gloves, and masks may have an increased awareness value in addition to reducing the risk of introducing infection. Basic biosecurity for visitors (drivers) and employees should include protocols to ensure clean boots, clothing, and equipment.

No pig products should be allowed on to the farm; all other food for human consumption should be consumed in a designated area and waste food correctly disposed of.

Great care is needed if second-hand equipment is purchased from other pig farms. If tools used on the farm are to be used off site, then they should be cleaned and disinfected before leaving and on return.

Fig 2.12 – Carcasses should be disposed of appropriately. (From Pritchard et al. (2005), with permission.)

Carcass disposal

Carcass disposal is an important consideration (Fig. 2.12). The Animal By-Products Regulations (2003) prohibit on-farm burial. The National Fallen Stock Scheme with a chargeable farm collection service started operating at the end of 2004. A separate isolated precollection carcass storage facility on a concrete area that can easily be cleaned and disinfected should be located at the periphery of the farm to minimize the risk posed by collection vehicles. An on-farm low-capacity incineration unit

requiring approval by the State Veterinary Service is an alternative.

Dedicated clothing for handling carcasses and decontamination protocols are sensible precautions.

Cleaning, disinfecting, and deliveries

The longevity of infectivity can be quite long, for example porcine parvovirus and porcine circovirus type 2 are very resistant and survive for several months under common UK conditions, and *Brachyspira hyodysenteriae* may survive up to 40 days in moist faeces. PRRS virus has been isolated from cleaned and disinfected trailers rested overnight in cold conditions, and has been isolated in the environment for up to several weeks following depopulation.

The cleaning and disinfecting procedure is important in reducing the risk of transferring pathogens from external sources, between internal locations, or between batches of pigs using the same housing. Pathogens may be present on contaminated vehicles, clothing, boots, or pens. The efficacy of the disinfectant is dependent on its activity, dilution, and the presence of organic material. The removal of visible organic material is an advisable prerequisite. With boots, this entails using brushes, detergents, and water before using the disinfectant (Fig. 2.13). In the case of vehicles, this may include jet spraying the underside of vehicles, wheels, and wheel arches (care is needed not to create contaminated aerosols) before disinfecting. With pens, this usually entails jet washing with detergent and water of the floors, walls, ceilings, and equipment before the application of the disinfectant. Pens should be allowed to dry before the next batch of pigs is admitted. Ideally, the pen should be left empty for 3–7 days, with exposure to sunlight if possible.

Fig 2.13 – Foot baths with brushes and appropriate disinfectant will reduce the risk of introducing infection. (From Pritchard et al. (2005), with permission.)

The entrance to the farm should be a hard concrete surface that should be routinely cleaned and disinfected.

The supplies and products should be delivered to the reception, with the driver and vehicle remaining outside the entrance.

Intraherd security

All the principles discussed above apply equally to intrafarm biosecurity, although the risk assessment will be different. External security of building (vermin-proof); all in, all out policies; cleaning and disinfecting between houses; and treatment prior to moving or mixing may be important in controlling the spread of diseases between different ages, groups, and houses where pigs are kept.

The physical biosecurity measures of a biosecurity checklist are as follow (Pritchard et al. (2005), with permission.)

- Isolated location with a single approach road and single entrance (avoid public rights of way).
- Farm office or reception area off site, with clear instructions for visitors and with a means of communication (bells or phones).
- 'Keep out' signs and 'Don't feed the pigs' signs (outdoor units).
- Strategically placed, well-maintained effective disinfectant foot baths and cleaning equipment.
- Off-site parking area with easily cleaned and disinfected hard standing.
- Well-maintained wildlife-proof perimeter fence.
- Changing, showering, and washing facilities and separation of clean and dirty areas.
- Dedicated on-site and off-site equipment, vehicles, clothing, and boots.
- Loading–unloading bay for live pigs away from the main herd.
- Completely separate bay for carcass removal or on-farm incineration facilities.
- Appropriate isolation facilities.
- Feed bins sited on the farm perimeter with farm-dedicated blower pipes.
- Bird- and vermin-proof buildings and food stores.
- Appropriate facilities and protocols for slurry or manure collection and disposal.
- Off-site lorry or trailer washing and disinfecting facilities.

Assurance schemes

Farm assurance governs the production methods to produce safe and wholesome foods that comply with all statutory and voluntary codes of good agricultural practice. The objective of an assurance scheme is to assure consumers that they are buying safe food that has been

produced in a welfare- and environmentally friendly system.

Farm assurance is likely to have a positive impact on disease control and prevention, animal welfare, and the education and awareness of employees. Costs to the farmer include the time devoted to record keeping and the relevant inspection and membership fees.

Potential benefits to farmers include the following.

- A source of added value by commanding a price premium (if the consumer makes a preferential choice).
- Security of market access or barrier to import penetration: requires supermarkets to implement farm-assured purchasing strategies with rigour and consistency.
- Source of improved management: audits may reveal areas that can be improved.

The following are potential costs associated with farm assurance.

- Membership and inspection costs.
- Compliance costs: inversely related to the competence and attention to detail of the producer.

Assured British Pigs

Assured British Pigs (ABP) is the assurance body responsible for setting the standards for the pig meat supply chain. Standards are set on all stages of the supply chain, including animal feed, farm production, pig transport, and processing. There are currently 2600 members, accounting for 90% of all pig production in the UK. Membership enables the producer to sell under the umbrella of both the little red tractor mark and the Meat and Livestock Commission's British meat quality standard mark. A cornerstone of the ABP is the veterinary health plan (VHP), which is drawn up by a veterinary surgeon and reviewed annually. The standards within the scheme either meet or exceed the UK minimum.

As in areas such as its 'prohibition of castration and the sale of weaned pigs and stores through auction markets and the discouragement of tail docking and teeth clipping without a justifiable cause', the health plan also includes details and protocols regarding medication and records.

Auditing of the scheme at farm level involves an annual independent inspection supplemented by a quarterly report by a veterinary surgeon. The requirement for quarterly ABP inspections by a veterinarian is a bone of contention with some producers, but there is likely to be an added benefit. The scheme also undertakes traceability checks at the abattoir, which include antimicrobial residue levels in carcasses and salmonella testing.

In the case of ABP inspections, the most frequent non-compliance categories were sharp edges on structures and fittings, use of non-assured haulage, failure to review cleansing policy with regards to the salmonella code, home mixes, sourcing food ingredients from non-assured sources, lack of environmental enrichment, and standard manuals not present, signed, or read.

The certification standards include the following.

General requirements

Producers are expected to adhere to all relevant current legislation and codes of practice in addition to the standards of the scheme. Key booklets must be kept and signed to indicate that they have been read by the staff. General standards for the unit are defined and graded. There must be a biosecurity plan for visitors.

The state of repair, safety, suitability, and protocols for cleaning and cleanliness of the buildings are checked. The lighting requirements and the need for socialization are included. The need for hospital pens and the subsequent treatment of sick animals and the methods of handling stock are defined. Ventilation and feeding system must be working properly, with appropriate alarm systems in the event of malfunction.

Protocols for emergencies, effective pest control, effective annual worming of farm dogs and recording the action, and appropriate waste disposal procedures including carcass disposal are expected. Availability of nesting material, appropriate feeding regimens for sows, and environmental enrichment materials are checked.

Pig space requirements, ventilation, and temperature

Maximum stocking levels for grouped animals and space requirements for individual animals, such as sows in farrowing crates and boars in pens, are defined. Appropriate type of flooring is expected.

The thermal comfort of the pigs will be evaluated, and stockpersons are expected to be able to recognize signs of heat and cold stress.

Water and feed

The water requirements for different feeding systems, ages, and stocking densities with regard to trough space and access to nipple drinkers are defined.

The diet is expected to be qualitatively and quantitatively suitable for each class of pig, with adequate access and avoidance space. The feeding of waste food must conform to the current legislation, and other external feed ingredients must be from certified sources. Records must be kept. Home mixing must comply with a code of practice.

Protocols for the labelling and storage of medicated feed are required. The producer is defined as either using or not using permitted growth promoters.

Breeding stock

Appropriate body scores at service and farrowing are expected. Sows should not be placed in the farrowing crate more than 7 days prior to their farrowing date, and they should be removed once the piglets are weaned. Piglets should be placed in stable groups in growing accommodation once they are weaned at a minimum age of 28 days. Preweaning piglets should be thermally comfortable, with dry lying areas.

Health and veterinary practices

Only competent operators are permitted to perform tooth clipping, tail docking, boar tusk trimming, nose ringing, and ear tagging within the current legislative framework, and records must be kept. Treatment and medication must be recorded appropriately in a medicines record book and withdrawal periods observed. Usage level and concentration levels of all growth promoters must be recorded. Medicines need to be stored and used properly, and needles disposed of appropriately. A protocol for dealing with broken needles lodged within the animal must be followed and records kept. Mortality level must be recorded and reviewed. An action plan is required if levels are unacceptable or increase.

Veterinary health plan

There must be a VHP prepared and regularly updated by the attending veterinary surgeon. This may include protocols for routine procedures, vaccination programmes, deworming protocols, ectoparasite control programmes, and medicines recording and usage. There must be a biosecurity programme for replacement animals, and records must be kept of the herd performance including data on disease. These should be reviewed in relation to appropriate target figures. Action plans are required if vices are present. Abattoir ZAP (salmonella) results are required and action plans implemented depending on the results. A veterinary health planner has been produced by the Pig Veterinary Society that outlines the areas that must be covered to satisfy the scheme.

Movements, loading, and transport

The standards required include loading facilities, types of transport, driver experience, stocking densities, travelling times, documentation, and methods of handling.

Outdoor production requirements

Requirements are appropriate site selection and accommodation, stocking densities, breed type, incoming animal quarantine periods, vermin control, and appropriate provision of fallows and shaded areas. A training area for electric fence familiarization should be present.

Residue monitoring and zoonosis action plan (salmonella)

Routine residue checks are made at the slaughterhouse on carcasses. If the maximum residue levels are exceeded, the veterinary surgeon must investigate the incident and submit a report to the producer and the assurance scheme.

The ZAP (salmonella) scheme of BPEX forms part of the assurance scheme, and standards and actions are defined dependent on the results, with the aim of reducing the incidence of salmonella in the herd.

Veterinary health plans

The aims of the veterinary health plan

Veterinary health plans are designed to reduce the prevalence of current disease, prevent the outbreak of further disease, and improve and maintain pig welfare. They should provide a management tool that is integral to the functioning of the unit. The benefit to the farmer is usually an increase in the profitability of the enterprise. The need for a VHP is also an important component of the standards required by assurance schemes. Agreement between the veterinarian and the owner, manager, or stock keeper is crucial in promoting adoption and compliance.

The VHP should encourage best evidence-based health and welfare practices and should be tailored to meet the specific needs of individual farms. This should include reduction of the use of medication in favour of alternative methods of prevention and control. The plan should encompass and reflect the current legislation and the Code of Recommendations for the Welfare of Livestock (Defra).

Formulating the veterinary health plan

A detailed assessment of the farm is required when formulating the VHP. This should include a farm visit. The initial visit should establish the current performance of the unit or set in place recording systems to do so, the health status of the herd with regard to targeted and important diseases, performance targets for the future, and a monitoring and surveillance programme. Regular routine farm visits at least every 3 months are recommended to appraise and review the health, welfare, and productivity parameters of the unit. The implementation of the plan and the suitability of the VHP should be reviewed in relation to the findings following each visit. Appropriate amendments should be made to optimize the plan in response to the outcome of the visits.

The plan should be written down, comprehensible, precise, and readily available for consultation on the farm. It should indicate the protocols and procedures to be followed and to which class or group of animal it refers. The timing of the prescribed procedures should also be stipulated.

Recording, monitoring, and reviewing

Recording and monitoring of essential parameters are vital to understanding the production and disease status of the herd. The important and common conditions identified on the farm should be recorded in order to determine their incidence. These conditions should include diarrhoea, pneumonia, lameness, mastitis, vices such as tail biting and flank sucking, fighting wounds, shoulder sores, and inappropriate condition scores (CSs). Deaths should be recorded in the different groups of animals on the farm so that mortality rates are generated. Production and fertility parameters should be measured to identify subclinical conditions or suboptimal management.

Targeted surveillance programmes may include routine environmental swabbing for salmonella, routine serological testing for enzootic pneumonia, feedback from slaughter surveillance through the British Pig Health Scheme for a range of important conditions, routine post-mortems of selected clinical cases, and a review of drug usage.

Collating and reviewing the collected data with the production of a written report are important. Action plans may be a specific outcome of the review or follow further investigations of the problem(s). The health plan should not be static but should be reviewed prospectively in response to predicted changes or reactively in response to improvement, decline, or changed circumstances of the herd. If possible, the same veterinarian should be responsible for a given herd to ensure commitment, continuity, communication, and consistency.

Medicines

The VHP should include standard protocols for the use of medicines on the farm. These should include dose rates, duration of use, indications of use, withdrawal times, and how the response can be assessed and the action to be taken if no improvement occurs within a stated time frame. The VHP should provide clear instructions of the legal requirements to record the appropriate information in a suitable medicines recording book. If in-feed medication is being supplied using a medicated feedstuff prescription, it must be included in the VHP and recorded similarly. There should be a quarterly review of the drug usage and the instructions dependent on the farm assessment. The legal requirements for the storage of medicines, the need for appropriate training in the administration of medicines, and the safe disposal of clinical waste should be explicit. It would also be advisable that it is a requirement for the staff to have read the Responsible Use of Antibiotics in Agriculture Alliance guidelines, Responsible Use of Antimicrobials in Pig Production, and the Veterinary Medicines Directorate's Code of Practice on the Responsible Use of Animal Medicines on the Farm.

Disease control programmes

Vaccines

The online Responsible Use of Medicines in Agriculture Alliance (RUMA) Guidelines: Responsible Use of Vaccines and Vaccination in Pig Production are a useful resource (see Appendix 4). The recommendations for individual vaccines vary, and manufacturers' details should be consulted regarding vaccine programmes. Before implementing a vaccine programme, the problem should be identified accurately and the risk should be assessed. Consider if vaccination is a cost-effective option and whether this strategy meets the long-term aims and goals of the unit. Consider whether you wish to provide active immunity in the adult or growing pig, or provide passive immunity to piglets. The following vaccines are currently available in the UK:

- *Erysipelothrix rhusiopathiae*.
- *Escherichia coli* (various strains, e.g. K88, K98, and K99).
- Clostridial vaccines, usually multivalent vaccines: *Clostridium perfringens* type B, *C. perfringens* type C, *C. perfringens* type D, *C. septicum*, *C. tetani*, *C. novyi* type B, and *C. chauvoei*.
- *Lawsonia intracellularis* (proliferative enteritis).
- *M. hyopneumoniae* (enzootic pneumonia).
- *Haemophilus parasuis* (Glasser's disease).
- Aujeszky's disease (porcine herpes virus-1): not present in Britain but available for strategic use in Northern Ireland.
- Atrophic rhinitis (*P. multocida* and *Bordetella bronchiseptica*).
- Porcine parvovirus.
- PRRS.
- Antisera: *C. perfringens* type B and *C. perfringens* type C.

Protocols for the vaccines currently in use on the farm should be included in the VHP, indicating the vaccine to be used, the class of animal to vaccinate, the timing of the vaccine, the precautions to be observed while using the vaccine, the dose to be used, and the route of administration. The VHP should also indicate where and what records need to be kept.

Ectoparasites and endoparasites

Mange (*Sarcoptes scabiei*) and lice (*Haematopinus suis*) are commonly found on pig units. Where present, the VHP should contain an effective control programme. Treatment of the sow before farrowing is important in reducing infestation of the piglets.

Endoparasites including *Ascaris suum*, metastrongyles (lungworm), and *Oesophagostomum* may have a significant economic impact on infected farms. The VHP should

contain a routine worming programme or a periodic monitoring programme involving collecting and analysing samples of dung.

Production and target figures

The parameters below should be recorded in order to gain an accurate measure of the productivity of the herd.

- Sow and piglet. Sow identity, service data, and boar used; failure to farrow and abortion data; number born alive, number born dead, number mummified; total litter weight at birth; piglet individual identification; piglet death, including date and reason; date of weaning and age at weaning; number weaned; and litter weight.
- Weaners. Date of entering unit; weaner identification; death, including date and reason; weight on entry (group); date of exit from unit; weight on exit (group); food consumed; and cost of food.
- Growers and finishers. Date of entering unit; pig identification; death, including date and reason; weight on entry (group); date of exit from unit; weight on exit (group); food consumed; and cost of food.

From these primary data, secondary indices can be computed. Important indices are shown in the table of targets. The figures can be used to compare the current performance of the unit to the previous performance, the national average, the relative performance of the top 10% of herds, and the relative performance compared with the bottom 10% of national herds. The indices may identify the impact and character of subclinical disease, clinical disease, and incorrect management. Tables 2.4–2.8 give UK and US figures for selected monitored herds.

Table 2.4 Annual results for all breeding stock up to September 2004

	Average	Top third[a]	Top 10%[a]
Herd structure			
Average no. of sows and gilts	474	563	556
Average no. of unserved gilts	40	47	38
Sow replacements (%)	45.4	45.2	47.6
Sow sales and deaths (%)	42.3	42.4	47.2
Sow mortality (%)	4.7	4.7	6.6
Sow performance			
Successful services (%)	82.7	83.1	79.2
Litters/sow per year[b]	2.21	2.29	2.33
Non-productive days[c]	40	28	25
Pigs born/litter			
Alive	10.74	11.17	11.48
Dead	0.80	0.81	0.82
Mummified	0.09	0.10	0.14
Total	11.63	12.08	12.44
Mortality of pigs born alive (%)	10.4	9.5	10.0
Pigs reared/litter	9.63	10.11	10.33
Pigs reared/sow per year[b]	21.3	23.1	24.1
Weight of pigs produced (kg)[c]	7.3	7.1	6.8
Average weaning age (days)	26	26	25
Feed usage[d]			
Sow feed/sow per year (tonnes)	1.334	1.301	1.228
Feed/pig reared (kg)	69	63	55
Feed costs[d]			
Sow feed cost/tonne (£)	110.36	110.07	106.93
Sow feed cost/sow per year (£)	147.27	143.17	131.34
Feed cost/pig reared (£)	7.65	6.91	5.96

[a]Selected on the basis of pigs reared/sow per year.
[b]Per sow data exclude unserved gilts.
[c]Excludes gestation, lactation, and a 6-day weaning to service interval.
[d]Pigs sow data include unserved gilts.
(From Meat and Livestock Commission 2005 Pigs: annual year book 2005. Online. Available: http://www.mlc.org.uk.) (Courtesy of BPEX.)

Table 2.5 Annual comparison of results for outdoor and indoor breeding herds up to September 2004

	Outdoor herds	Indoor herds
Herd structure		
Average no. of sows and gilts	676	362
Average no. of unserved gilts	60	30
Sow replacements (%)	23.1	47.2
Sow sales and deaths (%)	31.2	44.7
Sow mortality (%)	1.6	5.9
Sow performance		
Successful services (%)	n/a	78.8
Non-productive days[a]	48	35
Litters/sow per year[b]	2.10	2.23
Pigs born/litter		
Alive	10.57	10.87
Dead	0.66	0.92
Mummified	0.02	0.14
Total	11.26	11.92
Mortality of pigs born alive (%)	9.10	11.80
Pigs reared/litter	9.61	9.58
Pigs reared/sow per year[b]	20.10	21.40
Weight of pigs produced (kg)[c]	7.80	7.40
Average weaning age (days)	30	27
Feed usage[d]		
Sow feed/sow per year (tonnes)	1.446	1.240
Feed/pig reared (kg)	72	63
Feed costs[d]		
Sow feed cost/tonne (£)	107.22	112.81
Sow feed cost/sow per year (£)	155.04	139.90
Feed cost/pig reared (£)	7.68	7.23

[a]Excludes gestation, lactation, and a 6-day weaning to service interval.
[b]Per sow data exclude unserved gilts.
[c]Pigs sold or transferred out at weaning.
[d]Per sow data include unserved gilts.
n/a, not recorded by all herds.
(From Meat and Livestock Commission 2005 Pigs: annual year book 2005. Online. Available: http://www.mlc.org.uk.) (Courtesy of BPEX.)

Table 2.6 Rearing results for all herds up to September 2004

	Average	Top third[a]	Top 10%
Herd structure			
Average no. of pigs	1449	1712	613
Pig performance			
Weight of pigs at start (kg)	7.4	7.1	7.6
Weight of pigs produced (kg)	36.5	34.7	34.4
Mortality (%)	5.0	3.1	3.5
Feed conversion ratio	1.84	1.65	1.59
Daily gain (g)	449	461	418
Feed usage and costs[b]			
Feed cost/tonne (£)	197.35	194.47	175.56
Feed cost/kg gain (p)	36.35	32.04	27.91
Feed cost/pig reared (£)	10.58	8.84	7.48
Sales			
Average sale value (£)	33.09	37.78	38.06
Sale weight (kg)	36.1	35.7	34.3
Liveweight price (p/kg)	91.58	105.82	111.08

[a]Selected on feed cost/kg liveweight gain.
[b]Home-mixed feed prices do not include milling and mixing charges.
(From Meat and Livestock Commission 2005 Pigs: annual year book 2005. Online. Available: http://www.mlc.org.uk.) (Courtesy of BPEX.)

Table 2.7 Comparison of outdoor and indoor rearing herds up to September 2004

	Outdoor rearing herds[a] average	Indoor rearing herds[b] average
Herd structure		
Average no. of pigs	2285	1216
Pig performance		
Weight of pigs at start (kg)	7.2	7.3
Weight of pigs produced (kg)	36.8	34.4
Mortality (%)	3.7	4.0
Feed conversion ratio	1.72	1.88
Daily gain (g)	486	453
Feed usage and costs		
Feed cost/tonne (£)	171.01	200.13
Feed cost/kg gain (p)	29.34	37.54
Feed cost/pig reared (£)	8.68	10.17

[a]Pigs born and reared outdoors.
[b]Includes pigs born outdoors or indoors and reared indoors.
(From Meat and Livestock Commission 2005 Pigs: annual year book 2005. Online. Available: http://www.mlc.org.uk.) (Courtesy of BPEX.)

Table 2.8 Parameters recorded by PigChamp for 225 US herds (year ending 2004)

Measurement	Mean	Upper 10th percentile	Lower 10th percentile
No. of repeat services	472.42	103.00	984.00
Per cent repeat services	13.17	7.10	19.90
Total no. of services	3768.60	7573.00	896.00
No. of sows farrowed	3042.52	6388.00	721.00
Farrowing rate	77.72	85.40	67.80
Total pigs born	35 031.81	72 304.00	8220.00
Average total pigs/litter	11.51	12.40	10.70
Total pigs born alive	31 399.66	65 186.00	7147.00
Average pigs born alive/litter	10.34	11.20	9.40
Average pigs born alive/sow per year	23.26	26.18	19.99
Total stillborn pigs	2756.66	578.00	5953.00
Average stillborn pigs	0.92	0.60	1.30
Total mummified pigs born	866.49	82.00	1904.00
Average mummies/litter	0.25	0.10	0.40
Sows farrowed and weaned	2937.29	6075.00	690.00
Total pigs weaned	27 323.94	56 896.00	6239.00
Pigs weaned/litter weaned	9.10	9.80	8.30
Preweaning mortality	12.47	8.40	17.70
Average piglet weaning weight	12.20	14.00	11.00
Average age at weaning	18.23	20.30	16.10
Pigs weaned/mated female/year	21.25	23.70	18.20
Pig weaned/female per year	20.27	23.10	17.30
Ending boar inventory	17.80	33.00	0.00
Females entered	740.06	1516.00	155.00
Sows and gilts culled	607.01	126.00	1244.00
Culling rate	43.82	29.10	58.90
Sow and gilt deaths	118.83	18.00	289.00
Death rate	7.87	3.70	12.00
Average female inventory – average gilt pool inventory	1296.28	2642.20	323.90

(From PigChamp, with permission. Online. Available: http://www.pigchampinc.com.)

Veterinary health plan (Pig Veterinary Society, UK, with permission)

1. OBJECTIVES

1.1 The purpose of the VHP is to encourage a proactive approach to the maintenance of the health and welfare of pigs on the farm.
1.2 The VHP must be agreed between the attending veterinary surgeon and the client, together with those caring for the pigs. It will summarize the measures agreed. It is a confidential document that will remain on the farm.
1.3 The VHP should encourage 'best practice' husbandry and preventive medicine and should be designed to meet the specific needs of the individual farm.
1.4 The VHP must have a framework that can be audited.
1.5 The VHP must avoid duplication and refer to existing farm policy statements where relevant. Many of the requirements relating to health and welfare of pigs are addressed within the Farm-assured British Pigs, Scottish Pig Industry Initiative, and other assurance schemes' annual and quarterly veterinary reports.
1.6 The VHP must be produced annually when necessary, and amended quarterly as part of the veterinary visit report.

2. HERD HEALTH SECURITY

2.1 There must be ongoing monitoring and approval of replacement stock and semen, which will lead to matching and improving farm health status. Quarantine and acclimatization procedures, including vaccination policies, must be assessed and agreed.
2.2 As visitors are a potential source of infection, a written visitors policy must be agreed with the veterinary surgeon, and all persons entering the unit must be recorded in a visitors book.
2.3 Risk associated with farm machinery, such as tractors and those of contractors who have to enter the pig unit working area, must be assessed and suitable precautions devised.
2.4 A policy regarding all goods entering the farm, such as feed, bedding, and materials, must be established so that the risk of infections is minimized.
2.5 The management of pig loading facilities at the unit perimeter, including hygiene precautions, should be assessed and agreed.
2.6 The danger of infection from neighbouring pig units and pig lorries on nearby roads must be assessed and a means devised to reduce the risks of the disease entering the farm.

3. STOCKMANSHIP

The VHP will recognize the importance of stockmanship by identifying key personnel; agreeing with them inspection, reporting, and recording procedures; and assessing their competence and training needs.

4. HERD PERFORMANCE

4.1 Include a regular assessment of herd performance with agreed performance targets.
4.2 When necessary, an action plan should be developed to keep performance levels at the targets agreed.
4.3 Monitor condemnations and 'dead on arrivals' and relate them to conditions on the farm. Corrective action should be taken when levels are outside acceptable targets.

5. DISEASES, DEFECTS, AND BEHAVIOURAL DISORDERS

5.1 Identify, record, and rank transmissible disease agents known to be present within the farm by assessment of their potential impact on welfare, food safety, and the cost of production. Agree, where necessary, the appropriate methods of control, always maintaining an emphasis on improved husbandry.
5.2 Identify, record, and rank diseases, defects, and behavioural disorders known to occur within the farm but not readily associated with specific transmissible agents, according to their impact on welfare and production. Agree appropriate methods of control, always including an emphasis on improved husbandry.
5.3 Establish appropriate treatment protocols, including the use of medication, husbandry routines, and environmental changes.

6. REVIEW OF MEDICINE USAGE

6.1 A quarterly review of each treatment programme involving medicines must be carried out, with the conclusions being documented.
6.2 A quarterly review of the authorization and competence of individual stockpeople to administer medicines must be carried out.
6.3 The VHP must include a list of medicines that are permitted to be used on the farm, the circumstances of their use, their dose rate, and their withdrawal period. Updated medicines lists are to be provided as required.

(continued)

Veterinary health plan (Pig Veterinary Society, UK)—cont'd

6.4 The proper storage of medicines and the safe disposal of used medicine containers must be assured.

7. HYGIENE

A written cleansing programme must be agreed with the attending veterinary surgeon. The plan will take account of the layout of the farm, the pig flow within it, and targeted transmissible disease agents.

8. ENVIRONMENT

The VHP must assess and confirm that the environment within which the pigs are housed is conducive to good health and, where necessary, identify the means of resolving any environmental problems. Particular emphasis must be given to stocking density and to the incidence of lameness and behavioural problems.

9. FOOD SAFETY

A regular assessment of the farm and the health of the animals on the farm with regard to food safety should be made. This should include consideration of any monitoring schemes in place, for example ZAP scores, laboratory reports, and abattoir reports. Any areas of concern that are found should be noted and an appropriate plan of action put in place. This should be reviewed at the next quarterly assessment at the latest, or sooner if a serious problem is identified.

10. ASSOCIATED POLICIES

Associated policies to be drawn up in conjunction with the veterinary surgeon and to be made available on the farm. These should take into account the particular needs of the farm, its health status, other livestock and activities on the farm, and its relationship to other farms either by proximity or regular contact.

- Pest control
- Visitors policy
- Disposal of fallen stock
- Disposal of sharps and pharmaceutical waste
- Cleansing programme

11. PERIODIC VETERINARY HEALTH PLAN REVIEW

The VHP should be completed on a quarterly basis to facilitate a simple auditing of the document. Where other review sheets exist, they should be appended in support of this review. All assessments, agreements, and approvals must be confirmed by written veterinary reports held together on file at the farm as a working VHP document.

Period under review: _____

Assurance scheme no.: _____

11.1 Major diseases known or thought to be present

Disease	Control methods	Changes since last report

Veterinary health plan (Pig Veterinary Society, UK)—cont'd

11.2 Feed or water medication

Pig type	F/W	Condition	Treatment	Duration	Review

11.3 Other regular medication and vaccination

Pig type	Age	Condition	Treatment	Review

11.4 Key performance factors

Month(s): _____ Year: _____		Target	Actual
Litter size	Born alive		
	Born dead		
	Mummified		
	Sow mortality (%)		
	Preweaning mortality (%)		
	Rearing herd mortality (%)		
	Finishing herd mortality (%)		
	Transport loss (%)		
Growth rate	Rearing		
	Finishing		
Abattoir	Dead on arrivals		
	Condemnations		

(continued)

Veterinary health plan (Pig Veterinary Society, UK)—cont'd

11.5 Environmental assessment

Please comment on the thermal and structural environment and its interaction with the health and welfare of the pigs, noting especially any incidence of lameness and behavioural problems.

Section of the unit	Comments

11.6 Farm policies

Please review the farm policies and note if any action is required.

	In place	Action required
Pest control		
Visitors policy		
Disposal of fallen stock		
Disposal of sharps, pharmaceutical waste, and syringes		
Cleansing programme		

11.7 Food safety

Please review the farm with regard to food safety and note the results of any monitoring schemes that are in place and any action that is required.

Monitoring results	Action required

Veterinary health plan (Pig Veterinary Society, UK)—cont'd

11.8 Staff and training

For a first report, record the relevant training that staff have undertaken in pig welfare, pig health, and safe use of medicine usage, using the key below to indicate the training provider. Please identify where there is need for training. For subsequent reports, record any staff changes and their training and requirements.

Staff name	Position	Pig welfare	Pig health	Medicines usage	Drug authorization and competence

Sow condition scoring

Sow condition scoring is an important technique for monitoring health and the suitability of the feeding programmes. Because of the demands of lactation and pregnancy, there will always be a cyclical change in the CS of the sows (Fig. 2.14). Body condition is usually lost during lactation and needs to be regained during the following pregnancy. Ideally, the sows should enter the farrowing house at CS 3.5. A CS less than 3.0 is too low, and CS greater than 4.0 is too high. At weaning, an acceptable CS would be 2.5. CSs less than this indicate a problem. During pregnancy, the sow should gain approximately 1 CS. At no stage should the sow be less than CS 2.0. The trends in sow CS on a unit can be assessed if different groups of sows are at different stages of the reproductive cycle. Boars should maintain a CS of 3.

Sows should be condition scored at weaning, at service, at mid-pregnancy, and at farrowing. At weaning, the sow is likely to be in the lowest CS following weaning because of the demands of the lactation. Poor CS results in an increase in the weaning to oestrus interval and may progress to the thin sow syndrome. Young sows in their first or second parity are particularly at risk, and delaying mating to the second heat in sows in very poor condition has been shown to increase the litter size. Although service usually occurs only a few days after weaning, some young sows lose CS dramatically in this period because of preferential muscle growth. At-risk individuals or groups should be given additional supplementation to attain target CSs for the next farrowing. The comparative CSs at mid-pregnancy relative to the weaning and service CSs give an indication of the success of remedial feeding strategies. Suboptimal CS at this stage can still be corrected before farrowing. Condition scoring at this point is a guide to the success of the chosen feeding. Although poor CSs recorded at farrowing cannot be corrected, the recognition of the problem enables inappropriate feeding regimens to be modified and changed in the future.

A rough guide to the feeding levels per day for normal average sows during gestation are up to 95 days, 2.5 kg; days 96–100, 3.0 kg; days 101–110, 3.5 kg; days 111–113, 2.5 kg; days 114–115, 1.0 kg; and day of farrowing, 0.5 kg. To avoid the excessive loss of condition, sows need to consume sufficient quantities of a high-energy, high-protein feed during lactation. Gilts may need additional feed 2 weeks before farrowing to optimize mammary development and lactation. To optimize intake, the ambient temperature around the sow should not be excessively high, and consideration may need to be given to insulating the creep area to reduce the heat output. Intake can also be increased by wetting the food. In addition, the water supply should be easily accessible and adequate. Water requirements during lactation may exceed 20 L/day. Flow rates of 1.5 L/min are required. Feeding the sow 3 times a day may increase the overall intake in low-CS sows, but mealtimes need to be separated by 5–6 h to allow stomach emptying to facilitate intake. On the day of farrowing, sows have little appetite. Feed levels should be increased gradually (0.5-kg increments) on the day after farrowing, starting at 1 kg twice daily and rising to 4.0–4.5 kg twice daily by 2 weeks after farrowing. Increases should be started in the afternoon to maximize intake.

If condition has been lost during lactation, then the best time to replace it is during the early to middle part of the following pregnancy. Thin sows can be given extra feed

Pig condition scoring diagram

Score Number	Condition	Description	Shape of Body
5	Overfat	Hips and backbone heavily covered	Bulbous
4	Fat	Hips and backbone cannot be felt	Tending to bulge
3.5	Good condition	Hips and backbone only felt with difficulty	Tube shaped
3	Normal	Hips and backbone only felt with firm pressure	Tube shaped
2.5	Somewhat thin	Hips and backbone felt without firm pressure	Tube shaped but flat (slab) sides
2	Thin	Hips and backbone noticeable and easily felt	Ribs and spine can be felt
1	Emaciated	Hips and backbone visible	Bone structure apparent (ribs and backbone)

Condition scores from left to right, 1: 2: 3: 4: 5:

Score:
1. Emaciated
2. Thin, backbone prominent
3. Ideal condition during lactation and at weaning, backbone just palpable
4. Slightly overweight, cannot find the backbone
5. Body rotund, over fat

Note: The 'condition score' and 'back fat' correlation does differ between breeds

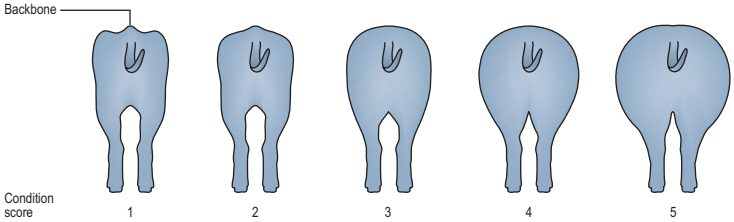

Fig 2.14 – Sow condition scoring. Note: the condition score and back fat correlation differs between different breeds. (After Muirhead and Alexander 2002.)

immediately after service. It is best to wait for at least 4 weeks after service before increasing the feed intake of gilts that require more fat cover because of the possibility of decreased embryo survival in gilts with high nutrient intakes in this period.

Environmental comfort (Code of Recommendations for the Welfare of Livestock: Pigs (Defra 2003)

A pig should be free to turn around without difficulty at all times. The animal should be able to stand up, lie down, and rest without difficulty. There should be a clean, comfortable, and adequately drained place in which it can rest, see other pigs, maintain a comfortable temperature, and have enough space to allow all the animals to lie down at the same time. For a pen holding individual pigs, no internal side of the pen should be less than 75% of the length of the pig, with the internal area not less than the square of the length of the pig. The exception to this is the sow in the farrowing crate.

The farrowing accommodation should be constructed and be sufficiently big to allow the sow to rise without difficulty. Standards from the Code of Recommendations for the Welfare of Livestock are in Tables 2.9–2.15.

Gases, dust, and bacteria

High levels of ammonia, carbon dioxide, and hydrogen sulphide gases can have adverse effects on human and animal health. Some authorities suggest that target levels should be ammonia, <10 ppm; carbon dioxide, <3000 ppm; and hydrogen sulphide, <5 ppm. Humans can detect by sense of smell ammonia at 5 ppm and hydrogen sulphide at 1 ppm. Specialized instruments are needed to measure these gases accurately. Dust levels can be measured using specialized equipment, and a target of less than 0.23 mg/m^3 has been suggested. Atmospheric bacterial load can be measured using specialized equipment and should be kept as low as possible. A rate of 100 000–120 000 colony forming units/m^3 is considered to be acceptable.

Table 2.9 Space requirements

	Minimum total floor area (m^2/pig)
Average weight (kg)	
<10.0	0.15
10.1–20.0	0.20
20.1–30.0	0.30
30.1–50.0	0.40
50.1–85.0	0.55
85.1–110.0	0.65
>110.0	1.00
Gilts	
<6 animals	1.80
7–39 animals	1.64
>40 animals	1.48
Sows	
<6 animals	2.48
7–39 animals	2.25
>40 animals	2.03
Boar	6.00
Boar pen used for service	10.00

(From Department for Environment, Food and Rural Affairs 2003 Code of Recommendations for the Welfare of Livestock: Pigs. Online. Available: http://www.defra.gov.uk.)

Table 2.10 Maximum width of openings and minimum width of slats when slatted concrete floors are used

Category of pig	Maximum width of opening (mm)	Minimum slat width (mm)
Piglets	11	50
Weaners	14	50
Rearing pigs	18	80
Gilts after service and sows	20	80

(From Department for Environment, Food and Rural Affairs 2003 Code of Recommendations for the Welfare of Livestock: Pigs. Online. Available: http://www.defra.gov.uk.)

Table 2.11 Temperature guidelines

Category of pig	Temperature (°C)
Sows	15–20
Suckling pigs in creeps	25–30
Weaned pigs (3–4 weeks)	27–32
Later weaned pigs (≥5 weeks)	22–27
Finishing pigs (porkers)	15–21
Finishing pigs (baconers)	13–18

(From Department for Environment, Food and Rural Affairs 2003 Code of Recommendations for the Welfare of Livestock: Pigs. Online. Available: http://www.defra.gov.uk.)

Table 2.12 Minimum trough space on rationed feed

Weight of pig (kg)	Trough space (cm)
5	10
10	13
15	15
35	20
60	23
90	28
120	30

(From Department for Environment, Food and Rural Affairs 2003 Code of Recommendations for the Welfare of Livestock: Pigs. Online. Available: http://www.defra.gov.uk.)

Table 2.13 Minimum daily water requirement

Weight of pig (kg)	Daily requirement (L)	Minimum flow rate through nipple drinkers (L/min)
Newly weaned	1.0–1.5	0.3
Up to 20 kg	1.5–2.0	0.5–1.0
20–40 kg	2.0–5.0	1.0–1.5
Finishing pigs up to 100 kg	5.0–6.0	1.0–1.5
Sow and gilts preservice and in pig	5.0–8.0	2.0
Sows and gilts in lactation	15–30	2.0
Boars	5.0–8.0	2.0

(From Department for Environment, Food and Rural Affairs 2003 Code of Recommendations for the Welfare of Livestock: Pigs. Online. Available: http://www.defra.gov.uk.)

Table 2.14 Guidelines for nipple drinkers

Type of feeding	Pigs per nipple drinker
Rationed	10
Unrestricted	15

(From Department for Environment, Food and Rural Affairs 2003 Code of Recommendations for the Welfare of Livestock: Pigs. Online. Available: http://www.defra.gov.uk.)

Table 2.15 Water trough space

Weight of pig (kg)	Trough space per head (cm)
Up to 15	0.8
15–35	1.0

(From Department for Environment, Food and Rural Affairs 2003 Code of Recommendations for the Welfare of Livestock: Pigs. Online. Available: http://www.defra.gov.uk.)

The routine farm (advisory) visit

Assurance schemes require regular timed visits (usually every 3 months) to participating farms, and the visit may be tailored to the requirements of the scheme. The visit may be part of a planned programme of visits to a unit independent of the requirements of the assurance scheme, or it may include the requirements of the scheme. Monthly visits are thought to be the optimum. The objectives of the visit will vary depending on whether it is an indoor nucleus, a multiplier or commercial herd, or an outdoor herd. Visits regarding specific herd disease investigations

are described in Chapter 1. Assurance scheme guidelines and the Code of Recommendations for the Welfare of Livestock: Pigs (Defra) provide a useful framework for the farm visit and are recommended even if the visit is not related to the scheme.

Preparation for the visit should include an examination of production indices, abattoir reports, and disease incidence since the last visit to identify potential problem areas. The internal and external biosecurity of the farm should be inspected. Each unit on the farm, including the farrowing house (Fig. 2.15), the weaners (Fig. 2.16), the fatteners (Fig. 2.17), the service area and dry sow yards (Fig. 2.18), the boar accommodation (Fig. 2.19), and the isolation area and hospital pen (Fig. 2.20) should be inspected. A checklist should be used ideally. The animals should be observed and inspected for signs of disease, vices, and abnormal behaviour. The accommodation should be inspected with regards to fixture (state of

Fig 2.17 – Growers. (Courtesy of D. Chennells.)

Fig 2.15 – Sow with piglets in a farrowing crate. (Courtesy of D. Chennells.)

Fig 2.18 – Sows in a straw yard. (Courtesy of D. Chennells.)

Fig 2.16 – Weaners in a flat deck pen. (Courtesy of D. Chennells.)

Fig 2.19 – Middle White boar.

Fig 2.20 – Hospital pens need to be checked. (Courtesy of D. Chennells.)

Fig 2.21 – Damaged slats are likely to cause injury. (Courtesy of D. Chennells.)

electrical wiring) and fittings (Fig. 2.21), drainage, bedding (dry and deep enough), hygiene, lighting, ventilation, air quality (Fig. 2.22), and environmental enrichment (e.g. chains, tyres, and balls).

The ventilation inlets and outlets should be checked to ensure that they are not blocked (Figs 2.23–2.25). Draughts may be obvious but must also be checked at pig level. Lying patterns may indicate a problem with draughts. Dust levels can be appreciated by visible inspection and how difficult it is to breathe. Ammonia can be detected by smell and the induction of runny eyes. Ammonia below 5 ppm has no effect, at 5–10 ppm can be detected by smell, and above 10 ppm increases eye irritation. Hydrogen sulphide from slurry smells of rotten eggs, and toxic levels can be lethal. Carbon monoxide detectors can be used to investigate suspected faulty heating systems. Smoke bombs can be used to investigate the air flow in more detail. It is suggested that this is done at the end of the visit, as smoke is often slow to clear and

Fig 2.22 – Dusty environments will predispose to respiratory disease. (Courtesy of D. Chennells.)

Fig 2.23 – Fans should be kept clean. (Courtesy of D. Chennells.)

Fig 2.24 – The condition of external vents should be checked. (Courtesy of D. Chennells.)

Fig 2.25 – Spaced boarding will improve the ventilation.

Fig 2.26 – Stocking density, lighting, and bedding quantity and quality should be assessed.

may impact on other observations that you may wish to make.

Lying patterns of animals indicate the degree of thermal comfort.

Cold pigs:

- lie on the floor with legs tucked under their bodies to reduce floor contact,
- lie huddled with other pigs,
- lie close to a wall,
- lie away from damp or cooler areas, and
- may shiver and become hairy.

Comfortable pigs:

- lie with legs spread out, some in contact with each other, and
- lie alone on the periphery of the group.

Hot pigs:

- pant >40 breaths/min,
- lie away from other pigs and do not pile up,
- lie in wet or cooler areas,
- try to wallow and are therefore dirty, or
- try to dig.

Environmental digital thermometers are useful to check appropriate temperatures at pig level. There should be sufficient bedding to allow all the pigs in the group to lie down comfortably in the bedded area. Excessive coughing should be noted; counting the number of coughs over time for a defined group of pigs will give an objective measure.

The quality and quantity of feed should be reviewed, including the nutrition plan for each class of animal on the farm. The method of feeding and the related equipment should be inspected to ensure appropriate storage to avoid wet and mouldy food and wasteful floor spillage from broken troughs, pipes, and containers. The stocking density (Fig. 2.26), the trough space, and the availability of water (Fig. 2.27) should be assessed. Drinker leakage,

Fig 2.27 – The provision of water is vital.

location, height, angle, flow, and colour of water should be inspected.

The cleansing policy, the flow of pigs, and the implementation of all in, all out approaches should be analysed.

The inspection may be followed by the clinical examination and necropsy of selected sick pigs for diagnosis and monitoring. There may be additional collection of laboratory samples if this was deemed necessary and cost-effective.

There should always be a written report with appropriate costings and cost benefits of any advice suggested. There should be a review of the VHP with regard to the implementation of the plan and the possible amendments in view of the current status and management policy of the herd. The report should be discussed in detail, ideally at a follow-up visit within 7 days. The report should try to motivate the staff by congratulating the farm on improvements and performance targets being achieved, while at the same time drawing attention to

areas that need attention to maintain good welfare and productivity. It is suggested that the areas needing attention should be prioritized and the actions required kept under seven so as not to overwhelm or discourage the stockpersons.

In addition to specific comments and recommendations, it is suggested that the report should also contain information about the medicines on the unit and their basic use, in-feed medications, in-water medications, routine injectable or oral doser medication, and vaccines used (what, to what, and when). In addition, there should be a written assessment of welfare on the unit. This may include an assessment and justification of any potentially injurious routine husbandry procedures such as tail docking, teeth clipping, castration, and nose ringing; an assessment of the basic environment for the various areas, ages, and groups of pigs; and an assessment of the space stocking densities, trough space allowance, and provision of water. There should also be comments regarding the suitability and implementation of the cleansing and disinfection programmes and waste disposal protocols. Recommendations should also be included regarding the suitability of current and future training programmes.

Abattoir monitoring

Scoring systems and tests to be used at the abattoir have been developed to provide qualitative and quantitative disease information for producers and their veterinarians. Examples include lesion scoring systems for atrophic rhinitis (turbinates), sarcoptic mange (skin lesions), and enzootic pneumonia (lung lesions). For example, with the enzootic pneumonia scoring system, a score up to 55 is awarded based on the site and extent of the lesion in the lung lobes.

The British Pig Health Scheme (BPEX)

The British Pig Health Scheme is based on the Wholesome Pigs Scotland scheme.

This scheme involves a cohort of trained veterinarians equipped with palm computers carrying out carcass scoring in all assured abattoirs by visual and sometimes manual appraisal in England and Scotland, which account for 90% of British slaughter pigs. The results are sent to the producers and their veterinarians within 48 h of slaughter.

The conditions to be monitored include enzootic pneumonia, pleurisy, pericarditis, pyaemia, pleuropneumonia-like lesions, peritonitis, milk spot liver, hepatic scarring, papular dermatitis, and tail biting.

The presence or absence of pathological lesions will be recorded, and the percentage of affected pigs calculated. Producers will be able to benchmark their comparative health status.

Zoonosis action plan (salmonella)

The BPEX ZAP (salmonella) programme uses a meat juice ELISA to monitor the pig exposure to salmonella organisms. In the 12 months to 30 June 2004, 142 776 samples were tested, with 23.2% of these tested positive. At the end of each month, holdings are allocated to one of three categories based on the antibody status of pigs they have supplied during the previous 3 months.

- Level 3: = or >75% of samples tested positive.
- Level 2: 50–74% of samples tested positive.
- Level 1: <50% of samples tested positive.
- Level 0: status not assigned – too few samples.

Holding in category levels 2 and 3 is required to develop and implement an action plan for the control of salmonella. Holdings in level 1 should strive to improve their situation. These cut-off points may be lowered in time.

Antimicrobial medication

See Chapter 14 (Antimicrobial therapeutics).

Checklists for indoor herds

Checklists for outdoor herds can be found in Chapter 12 (*Outdoor pigs and organic herds*).

Indoor farrowing area

Environment

- Temperature.
- Farrowing house.
- Air quality (dust, ammonia, and draughts).
- Fans (working or blocked).
- Lighting.
- Design of farrowing pen.
- Crate.
- Creep area.
- Fixtures and fittings.
- Provision of water quantity and quality (sows and piglets).
- Nipple drinkers (site, function, and number).
- Condition of bedding and slats.
- Condition of flooring (rough or smooth).
- Cleansing policy.
- Hygiene.
- Drainage.
- Signs of diarrhoea and other abnormal discharges.

Sow health
- CS.
- Frequency of feeding and feed intake.
- Health problems (mastitis, mastitis–metritis–agalactia, prolapse, vaginal haematomata, pressure sores, uterine discharges, or pyelonephritis).
- Litter size, parity, stillbirths, mortality, and congenital defects.
- Culling rate (reasons for culling).
- Observations.

Piglet health
- Lying patterns.
- Growth (even, uneven, or thin).
- Contentment (quiet or noisy).
- Lameness (joint ill).
- Tail or teat necrosis.
- Hernias.
- Diarrhoea.
- Abnormalities (splay leg or tremors).
- Respiratory disease (rhinitis or pneumonia).
- Ocular discharge (conjunctivitis).
- Skin (greasy pig disease).

Procedures and protocols
- Frequency of checking sow due to farrow.
- Review current treatments.
- Tail docking.
- Teeth clipping.
- Iron injections.
- Sow induction protocols.
- Routine therapeutic antibiotics.
- Age at weaning.

Records
- Date into crate.
- Date of farrowing.
- Date of weaning.
- Date out of crate.
- CS sows weaning.
- Litter size and stillbirths.
- Piglet mortality.
- Number weaned.
- Weight at birth.
- Weight at weaning.
- Feed sows.
- Feed piglets.

Biosecurity
- Footbaths.
- All in, all out policy.

Indoor dry sow yard

Environment
- Air quality (ventilation, dust, ammonia, and draughts).
- Temperature (hairy pigs?).
- Lying patterns.
- Lighting.
- Stocking density (bedded or unbedded).
- Provision of water quantity and quality.
- Nipple drinkers (site, function, and number).
- Condition of bedding: straw (usage) and slats (size).
- Condition of flooring (rough or smooth).
- Cleansing policy.
- Hygiene.
- Drainage.

Health
- CS.
- Uterine discharges.
- Fighting wounds.
- Lameness.
- Chronic mastitis.
- Vulval biting.
- Pressure sores (shoulders).
- Faecal quality.
- Culls (reasons).
- Mortality rate (causes).

Feeding
- Method and frequency of feeding.
- Consumption.
- Trough space.

Protocols and procedures
- Vaccination programmes.
- Ecto- and endoparasite control.
- Preparation for farrowing crate (washing).
- Servicing protocols (natural or artificial insemination).
- Mixing protocols.
- Pregnancy-testing protocols.
- Cleansing protocols.

Records
- Date of birth.
- Weaning date.
- Service dates (boar or semen used).
- Sows not in pig.
- Sows in pig.
- Sows repeating.
- Abortions.
- Oestrus abnormalities.

Biosecurity
- Foot baths.
- Other methods.

Boar pen

Environment
- Air quality (ventilation, dust, ammonia, and draughts).

- Temperature.
- Lighting.
- Size of pen (bedded or unbedded).
- Provision of water quantity and quality.
- Condition of bedding.
- Condition of flooring (rough or smooth).
- Cleansing policy.
- Hygiene.
- Drainage.
- Environment enrichment.

Health

- CS.
- Lameness.
- Fertility.
- Date of last physical examination.
- Claws (normal or overgrown).
- Tusks (normal, overgrown, or misshapen).
- Bright, alert, and reactive.
- Pressure sores.
- Pruritus.
- Service performance (libido).

Protocols and procedures

- Service protocol.
- Boar usage.
- Vaccinations.
- Ecto- and endoparasite control.

Feeding

- Quantity, quality, and frequency.

Records

- Date of birth.
- Date of service.
- Conception rates.

Biosecurity

- Foot baths.
- Cleansing and disinfecting.

Weaners

Environment

- Air quality (ventilation, dust, ammonia, and draughts).
- Temperature.
- Lighting.
- Stocking density (bedded or unbedded).
- Provision of water quantity and quality.
- Trough space.
- Nipple drinkers.
- Condition of bedding.
- Condition of flooring (rough or smooth).
- Cleansing policy.
- Hygiene.
- Drainage.
- Environment enrichment.
- Abnormal discharges in pen (dysentery, pus, and frank blood).
- Faecal quality.

Health

- Lying patterns.
- Assess weight for age (evenness of group).
- Condition.
- Lameness.
- Coughing.
- Sneezing.
- Pot-bellied pigs.
- Hernias.
- Pruritus (mange and lice).
- Fighting.
- Diarrhoea.
- Nervous signs.
- Rectal prolapse.
- Vices (tail biting, flank and ear sucking).
- Abscesses.

Protocols and procedures

- Vaccinations.
- Ecto- and endoparasite control.
- Antimicrobial therapies.

Feeding

- Quantity, quality, and frequency.
- Trough space.

Records

- Mortality rate.
- Diarrhoea, pneumonia, lameness, and vices.
- Date of weaning.
- Weight at weaning.
- Weight on entering grower phase.
- Number of days as weaner.
- Growth rates.

Growers and finishers

Environment

- Air quality (ventilation, dust, ammonia, and draughts).
- Temperature.
- Lighting.
- Stocking density (bedded or unbedded).
- Provision of water quantity and quality.
- Trough space.
- Nipple drinkers.
- Condition of bedding.
- Condition of flooring (rough or smooth).

- Cleansing policy.
- Hygiene.
- Drainage.
- Environment enrichment.
- Abnormal discharges in pen (dysentery, pus, and frank blood).
- Faecal quality.

Health

- Lying patterns.
- Assess weight for age (evenness of group).
- Condition.
- Lameness.
- Coughing.
- Sneezing.
- Pot-bellied pigs.
- Hernias.
- Pruritus (mange and lice).
- Fighting.
- Diarrhoea.
- Nervous signs.
- Rectal prolapse.
- Abscesses.

Protocols and procedures

- Vaccinations.
- Ecto- and endoparasite control.
- Antimicrobial therapies.

Feeding

- Quantity, quality, and frequency.
- Trough space.

Records

- Mortality rate.
- Diarrhoea, pneumonia, lameness, and vices.
- Date of dispatch to abattoir.
- Weight on entering grower phase.
- Number of days as grower.
- Feed consumed.
- Growth rates.

Treatment and hospital pens

Environment

- Well ventilated but not draughty.
- Straw bedding to assist thermoregulation.
- Well lit.
- Clean fresh water should be available at all times.
- There should be an effective cleansing and disinfection and cleansing policy.
- Isolation pens (infectious conditions).

Health

- Presumptive diagnosis.
- Treatment plans and records.
- Defined end points.
- Signs.
- Morbidity.
- Mortality.
- Post-mortem results.

Protocols and procedures

- Treatment protocols.
- Post-mortem.
- Sampling and laboratory tests.
- Isolation.
- Euthanasia protocols.
- Disposal of carcass.
- Broken needles.

Feeding

- There should be provision of easily accessible palatable food.
- Quantity should be sufficient to support compensatory growth during the convalescent period.

Records

- Group of origin.
- Individual identification for future reference.
- Clinical and treatment records.

Biosecurity

- Foot baths.
- Change of clothing.
- Washing.

Medicine

- Storage.
- Recording.
- Disposal of clinical waste and sharps.

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Diseases of the musculoskeletal system

Introduction

Musculoskeletal problems are very common in pigs of all ages and are a major cause of culling, especially in breeding stock. They can also severely compromise the welfare of affected pigs. In most cases, the presenting clinical sign is lameness, the cause of which can be determined only by a careful clinical examination.

Aetiology of musculoskeletal disease

All components of the musculoskeletal system can be involved in disease and must be considered when an attempt is made to diagnose the cause of the problem. Accurate diagnosis is paramount in determining the cause, prognosis, treatment, and prevention of musculoskeletal problems in pigs. Nearly 60% of pigs with musculoskeletal problems examined by the Veterinary Laboratories Agency (VLA) in 1996–2003 were found to be suffering from arthritis. Nearly 50% of these were suffering from non-specific arthritis for which the exact cause was not determined.

Epidemiology of diseases of the musculoskeletal system

The environment of pigs and the fact that they are often kept together in large numbers predispose them to musculoskeletal problems. Poor management, with accommodation in a bad state of repair and inadequate handling facilities, may predispose to musculoskeletal disease in indoor units. The quality of outdoor accommodation may also lead to orthopaedic problems. Paddock quality may be poor and may be made worse by the rooting activity of the pigs. Brick and concrete waste may have been overlooked and is capable of causing severe injury to pigs, especially if they are frightened. Hot air balloons are particularly frightening to outdoor pigs and may cause panic and orthopaedic injuries. Some problems have been blamed on the rapid growth of the modern pig and the demands placed on its musculoskeletal system in both indoor and outdoor accommodation. Musculoskeletal problems may affect just a single animal, but infectious conditions may affect a large number of animals.

Clinical signs of diseases of the musculoskeletal system

The most common clinical sign caused by this group of diseases is lameness. If untreated, lameness is rapidly followed by the atrophy of disuse of muscle tissue and compromise of carcass quality. Unless diagnosis and treatment are rapid, permanent damage is caused, welfare is compromised, and the pig becomes unsaleable. Clinical examination of the lame pig can be difficult, and additional evaluation may require (potentially costly) x-ray and pathological examinations.

Clinical examination of the musculoskeletal system

In most other domestic species, the first stage of an examination for lameness is to see the patient move in a controlled manner. Movement may be observed on a number of surfaces, and lameness may be especially pronounced on hard surfaces. The patient is observed when turning, walking over obstacles, and at different speeds. The affected limb(s) is identified and the clinician notes the severity of the lameness and the degree to which the animal can bear weight on the affected limb(s). The leg is then examined in detail. The examination involves comparing the limb with the opposite unaffected limb and examining each part of the limb for signs of abnormality. The whole limb is closely examined for signs of injury, heat, pain, and swelling. The movements of each joint are evaluated, and any distension of the joint capsule or reduction in movement is noted. The foot is examined with care, because, as in most of the larger species, it is frequently the seat of the cause of lameness.

The nature and behaviour of the pig make it less easy to follow this regimen of examination. It is difficult to move the pig in a controlled fashion, and few pigs will stand still while their limbs are examined in detail. Pain often causes lame pigs to be recumbent, although they will attempt to rise if disturbed. If the pig is recumbent, a quite detailed examination of the patient can often be made without disturbing it. Gentle rubbing of the ventral abdomen may encourage the pig to remain in recumbency while the limb is examined. Once the examination is complete, the pig can be encouraged to get up when movement is observed and evaluated (see Figs 1.13 and 1.14).

Diagnosis of diseases of the musculoskeletal system

The diagnosis and treatment of musculoskeletal problems in pigs require a careful, detailed, and patient clinical examination. A large number of infectious agents have been associated with arthritis in pigs. Samples of joint fluid may be required for cytology and culture to identify the causal organism in major outbreaks of this disease. Ultrasonography can be very helpful in assessing the severity of the pathology in damaged muscle. If a fracture is suspected, radiology may be helpful but is costly and may be difficult to arrange under farm conditions. Additional tests may be necessary in some cases. If a trace element deficiency or vitamin is thought to be involved, samples from the animals and their food may be needed.

Treatment of diseases of the musculoskeletal system

This will depend on the cause of the problem and whether treatment is possible or likely to be successful. Antibiotic therapy can be very successful in infectious conditions. Treatment must be commenced in good time before serious pathology is established and reduces the prognosis for recovery.

Control of diseases of the musculoskeletal system

This can be considered under two main headings. Because many diseases in this group are associated with defects in the environment, efforts should be made to ensure that the environment is as hazard-free as possible. The incidence of neonatal joint disease can be substantially reduced by ensuring good uptake of colostrum. Early diagnosis and treatment of many musculoskeletal problems are essential if the prognosis for recovery is to be good. Pigs of all ages should be observed carefully to detect any sign of lameness at an early stage. Any lame animal should be carefully examined to determine the cause of its problem and whether the problem is likely to spread to other pigs in the group.

Veterinary Investigation Diagnosis Analysis sample submissions: musculoskeletal diseases, 1996–2003

See Table 3.1.

Table 3.1 Veterinary Investigation Diagnosis Analysis sample submissions: musculoskeletal diseases, 1996–2003

Condition	Incidence (%)
Arthritis (other)	49.90
No diagnosis	12.94
Acute stress myopathy	6.68
Osteochondritis dissecans	6.05
Myopathies (others)	5.64
Mycoplasma arthritis	5.22
Streptococcus suis arthritis	4.59
Skeletal defects (others)	3.76
Musculoskeletal (others)	3.76
Erysipelas arthritis	1.04
Splay leg	0.42

Congenital and hereditary conditions of the musculoskeletal system

A large number of congenital abnormalities have been reported in pigs. Some, like crooked tail, are relatively unimportant. Others, such as arthrogryposis, are not commensurate with life. Sporadic abnormalities are usually not investigated. If a number of cases are seen, a full investigation into possible genetic or toxic causes should be performed.

Fig 3.1 – Splay leg. (Courtesy of R.W. Blowey.)

Splay leg

Splay leg (Fig. 3.1) is an important abnormality that may severely compromise the survival of neonatal piglets.

Incidence

Splay leg is quite common. The condition accounted for 2% of the Veterinary Investigation Diagnosis Analysis (VIDA) diagnoses for musculoskeletal problems in 2002. It is seen especially in Landrace, Large White, and Welsh breeds, and also in hybrids of these breeds.

Aetiology

The aetiology of splay leg is unknown but may be multifactorial. It may be caused by a polygenic hereditary abnormality but is also caused or exacerbated by a choline deficiency or fusarium toxicity.

Pathology
This is a myofibrillar hypoplasia.

Epidemiology
Splay leg affects up to 4% of UK neonatal piglets. Morbidity in a litter can be 25% and the mortality of affected piglets 50%. Death is usually caused by crushing injuries. Affected piglets cannot take evasive action or suckle. They may also develop hypoglycaemia or hypothermia. Males are affected more frequently than females. Affected animals often have a low birth weight. Splay leg is exacerbated by slippery flooring in the farrowing area.

Clinical signs
Affected piglets are recognized within a few hours of birth. They cannot stand on their hind legs, which are splayed out laterally. The forelegs may be normal or also splayed. Affected piglets can suck milk if they can get to the teats.

Treatment

Piglets can be helped by loosely tying their hind legs together with a figure of eight tape placed just above the hocks. This should be done as soon as possible after birth. This will help up to 50% of piglets walk quite normally. Help is needed for a few days only, by which time piglets are walking normally and the ties can then be removed.

Control

Control is difficult because the exact cause is unknown. Vigilance is required to recognize and treat affected piglets. If a hereditary aetiology is suspected, it may be wise to change the boar.

Crooked or kinky tail

The condition (Fig. 3.2) is said to occur in up to 2% of all piglets. It is seen chiefly in Large White and Landrace breeds. Instead of being straight or curled, the tail has a sharp angle along its length caused by an autosomal dominant gene with low penetrance. The condition is not of great significance, as the growth of affected piglets is normal.

Syndactyly

This is an uncommon condition sometimes called mule foot. Piglets are born with a single hoof instead of the normal cloven structure. Dominant genetic inheritance is suspected.

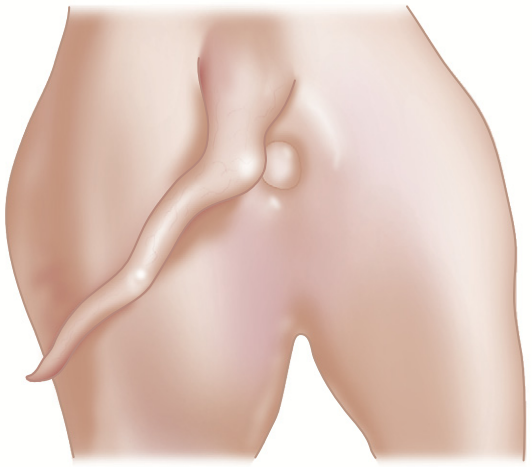

Fig 3.2 – Kinky tail.

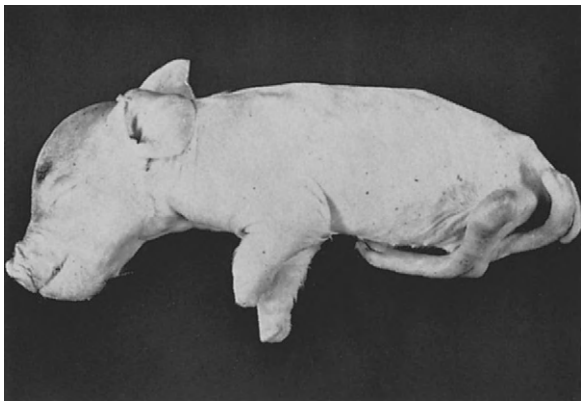

Fig 3.3 – Piglet with micromelia of forelegs and arthrogriposis of hind legs.

Polydactyly

Polydactyly is another uncommon condition, in which piglets are born with additional accessory digits. The condition is caused by an autosomal dominant gene that is lethal in its homozygous form. Affected piglets are heterozygous.

Legless piglets

This is an uncommon genetic defect caused by an autosomal recessive gene. Piglets are born with no or incomplete legs (Fig. 3.3) and must be euthanized.

Arthrogryposis

Piglets with arthrogryposis are born with fused limb joints. The condition may cause dystocia at the time of delivery. It is caused by an autosomal recessive gene. Arthrogryposis may also be caused by toxicity from pregnant sows eating hemlock, thorn apple, and tobacco plants. Affected piglets should be euthanized.

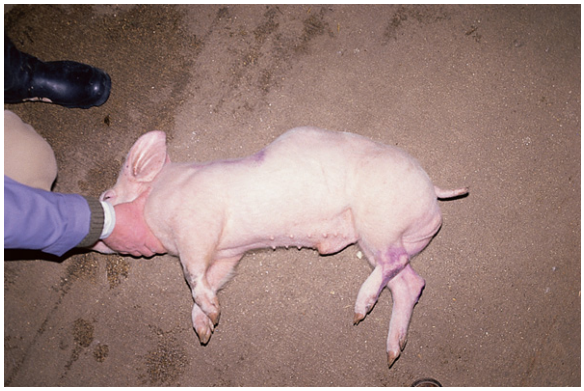

Fig 3.4 – Humpy-backed pig.

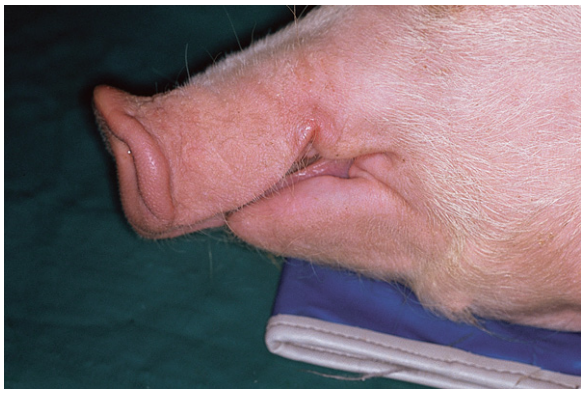

Fig 3.5 – Humpy-backed pig with small lower jaw.

Kyphosis and scoliosis

These axial skeletal defects are occasionally seen in individual piglets at birth. Their cause is thought to be genetic, but in other species viruses affecting the developing embryo are sometimes responsible. Treatment is not possible. Although some affected piglets survive and grow, they never reach a saleable state and euthanasia at birth is advised. Pigs affected by the humpy-backed pig syndrome show signs of severe kyphosis (Figs 3.4 and 3.5).

Pietrain creeper syndrome

Pietrain creeper syndrome is a primary myopathy seen in Pietrains and also Landrace pigs.

Aetiology

The condition is caused by an autosomal recessive gene. The condition has been reported in only one UK herd.

Clinical signs

Piglets appear normal at birth but show signs of muscular tremors at 2–4 weeks of age. By 12 weeks, they are completely recumbent. The carpal joints are flexed, and piglets attempt to walk on the toes of their partially flexed hind limbs. No neurological defects have been noted, and piglets may manage to eat and drink.

Differential diagnosis

The differential diagnosis is congenital tremor.

Treatment

There is no treatment. Euthanasia of affected piglets is required.

Dwarfism

Dwarfism is uncommon. Reports from Denmark have identified the cause as an autosomal recessive gene that causes both dwarfism and chondrodysplasia.

Congenital porphyria

The exact cause of the condition is unknown, but it may be hereditary. Piglets are born with a defect in porphyrin metabolism, affected animals producing excessive amounts. Porphyrins are excreted in faeces and urine but are also deposited in teeth and bones, causing a red discoloration. The affected animal may grow normally, but parts of the carcass may be unsaleable.

Inherited thick forelegs

Aetiology

The condition is a congenital hyperostosis caused by an autosomal recessive gene.

Clinical signs

Piglets are born with thickened forelegs, especially below the elbows. There is local oedema, and the bones appear thickened with an irregular, partially detached periosteum. Standing is difficult, and affected piglets are easily crushed by the sow. The skin over the affected parts of the leg is tense and discoloured.

Diagnosis

Post-mortem examination reveals that the periosteum is separated from underlying bone.

Treatment

No treatment is available. Affected piglets are euthanized.

Asymmetrical hindquarters

Incidence

This condition is uncommon but is reported in the UK and Europe.

Aetiology

The exact cause is unknown, but the condition is thought to be suboptimal growth of muscle rather than a degeneration. The cause is possibly genetic and the condition is seen in some herds, affecting families within those herds. The occasional case is iatrogenic, possibly associated with poor injection technique resulting in peripheral nerve damage.

Clinical signs

Male and female piglets are affected. They appear normal at birth, but by weaning time the asymmetry is apparent. No lameness is present in affected piglets.

Treatment

No treatment is effective. Euthanasia is used.

Control

The veterinarian should check the ancestral history of affected animals.

Porcine stress syndrome

Porcine stress syndrome (Fig. 3.6) is a very important syndrome that can severely compromise welfare and cause major economic loss.

Incidence

The condition has occurred in a number of breeds. Up to 11% of Landrace pigs may be affected. It accounted for 7.7% of the diagnoses made by the VLA for conditions affecting the musculoskeletal system of pigs. Cases have also been seen in Pietrain and Duroc pigs.

Aetiology

Porcine stress syndrome is an inherited defect caused by an autosomal recessive gene with incomplete penetrance. The gene responsible is the porcine stress syndrome gene, also known as the halothane sensitivity gene, involved in the calcium release channel of the sarcoplasmic reticulum. The physiological problem causes the muscle to be hypersensitive to various stresses. In response to stress, there is a rapid onset of anaerobic glycolysis and loss of control of skeletal muscle metabolism.

Epidemiology

Four syndromes are seen, which are described under *Clinical signs*. The genetic background to the condition means that it can be transmitted from one generation to the next, with continued serious welfare and economic consequences. Performance of affected animals may be reduced.

Fig 3.6 – Porcine stress syndrome: the skin shows alternating blanched and reddened areas. Muscle rigidity can be seen prior to death.

Clinical signs

The four main syndromes are as follow.

Porcine stress syndrome

Affected pigs show an abnormally severe response to stress, including travel, mixing, and weighing. Hot ambient temperatures exacerbate the problem. Initially, pigs may show muscle tremors, quickly followed by dyspnoea with open-mouthed breathing. Body temperature rises rapidly to very high levels, and the pig is very distressed. The skin is blotched with purple patches. The pig collapses, muscular spasm is seen, and death occurs. The victim may be attacked by its fellow pigs, which hastens its death.

Malignant hyperthermia

Malignant hyperthermia occurs in response to exposure to halothane anaesthesia and also to treatment with succinylcholine. Within a very short time, signs of abnormality are seen: muscle rigidity develops, temperature rises, and large amounts of lactic acid are produced. Heart rate increases and cardiac dysrhythmia is present. Death follows rapidly.

Pale soft exudative pork

Rigor sets in very rapidly after slaughter. Lactic acid is produced, and the pH of muscle falls. The meat loses its colour and normal consistency. The carcass later loses its

rigor, and tissue fluid drips out. Stress prior to slaughter exacerbates the effects of pale soft exudative pork. Dark, firm, dried pork is also seen and may be the result of prolonged stress.

Back muscle necrosis

Necrosis of parts of the longissimus dorsi muscle occurs. There is pain and swelling over the affected area, which gradually resolves over a period of 2 weeks. Damaged muscle never regenerates, and carcass value decreases. Some deaths occur. One or both muscles may be affected.

Diagnosis

This is based on the history and clinical signs. A number of diagnostic tests can be used, including those that may help to recognize the condition before it becomes a major clinical problem.

Halothane sensitivity test – The pig is carefully exposed to halothane and its response observed. This does not identify all affected animals.

Creatinine kinase assay – The pig is exposed to a carefully controlled exertion test and blood creatinine kinase assayed after 8 h. Creatinine kinase levels are much higher than normal in porcine stress syndrome pigs. The test is not 100% reliable.

Polymerase chain reaction test – This is used to detect the porcine stress syndrome gene in meat.

Blood typing – The absence of AO and presence of Ha blood groups – which helps identify animals in which the gene may be present – should detect affected animals and also heterozygous carriers.

Post-mortem – Post-mortem examination shows areas of pale muscle, low pH, and poor long-term setting qualities.

Treatment

Acepromazine and droperidol have been used to try to reduce the effects of halothane on susceptible pigs. In most cases, the rapidity and severity of the symptoms seen in the various clinical syndromes precludes any effective treatment, and most cases end fatally.

Control

Careful genetic selection of breeding stock is required. Cull affected animals and carriers. Reduce stress: lairage time of 1–3 h is ideal. If too short, pale soft exudative pork may occur; if too long, dark, firm, dried pork may develop.

Osteochondrosis: leg weakness in pigs

Incidence

Osteochondrosis is a very common and important condition in growing and older pigs.

The condition accounted for nearly 6% of the musculoskeletal problems in the VIDA survey of 1996–2003. The condition is also known as leg weakness, arthrosis, and epiphysiolysis. Some authorities have suggested that the condition is a dyschondroplasia, as it appears to be a problem associated with cartilage and is developmental, starting quite early in life.

Aetiology

The cause is unknown. The condition may have some genetic predisposition, but some of the evidence for this and other possible causes is conflicting. The pathological defect is believed to be a malfunction of cartilage production. Defects of conformation may develop from the disease but also contribute to it.

Epidemiology

The problem occurs in up to 80% of pigs in some surveys, with all breeds and both sexes being involved. Not all pigs with pathological lesions show signs of lameness. The condition is also associated with joint abnormalities, especially in the long bones, their growth plates, and their epiphyses, and in the vertebral joints.

A number of factors have been identified as having a possible role in the aetiology of the problem.

Rapidly growing pigs

Rapidly growing pigs have been considered to be susceptible to leg weakness, but there is *no* proof of this. Some slow-growing breeds, such as wild boar, have degenerative joint lesions. The role of growth hormone is unclear.

Nutrition

Nutrition is closely related to growth. Calcium : phosphorus ratios and vitamin A levels are normal. High-energy diets have *no* consistent adverse effect on bone quality.

Genetic factors

Suspicions of genetic predisposition have *not* been confirmed, and leg weakness has been shown to have quite a low hereditability. Leg conformation may be important, and animals with very upright legs may be more likely to develop the disease.

Flooring

A very hard or irregular floor surface may have an adverse effect on the incidence of leg weakness, but there is *no* consistent association. The effects of bad flooring may exacerbate the lesions of osteochondrosis.

The pathology of porcine osteochondrosis

An understanding of this helps explain how the disease develops and how the observed clinical signs occur. The pathology can be explained simply as follows.

- Growing cartilage is found in the growth plates (physeal cartilage), where it contributes to bone length, and in the joints, where it contributes to the articular cartilage and to epiphyseal growth.
- In osteochondrosis, some areas of cartilage growth do not proceed in an orderly fashion. Focal areas in the physeal, articular, and epiphyseal cartilages show failure of normal cartilage growth. Some areas show reduced growth and others increased growth in the same animal.
- As a result of the pathological changes, abnormalities may be seen in various parts of the bone and its joints. Joint 'mice' may develop.
- Physeal cartilage defects may lead to bending and deformity of the bones.
- Articular and epiphyseal cartilage defects may lead on to sterile joint abnormalities. Some areas of the articular cartilage are thickened, and others are thinly covered. There may be some in-folding of cartilage between the various areas.
- In piglets, articular cartilage increases in thickness until about 5 weeks of age. After that age, there is a reduction in the thickness of articular cartilage.
- Clinical signs of abnormality may not be seen until the pig is 6 months of age or more, even though the pathology has been present from a very early age.
- Lesions of articular osteochondrosis are seen mainly on the weight-bearing medial condyles of the long bones, such as the femur and humerus (Fig. 16.20). They can also affect the intervertebral articulations, causing disc pressure or spondylitis.
- Long-term articular cartilage problems can lead on to loss of the cartilage and the establishment of degenerative joint disease; initially there is some increase in sterile synovial fluid, but there is later loss of fluid, loss of residual cartilage, and the establishment of a dry joint.

Clinical signs

Clinical signs are seen in about 30% of animals with bony lesions. Animals show pain when asked to walk or stand. The signs can be quite variable. Pigs may walk on their knees with carpi flexed. The animal may seem unwilling to extend its carpi, while the fetlock joints may be overextended. The toes of the hind legs may be turned in. The animal may adopt a wide hind leg stance. The pig may be ataxic and have a swaying gait with swaying movements of the hindquarters. When such animals get to their feet, they may stand with their hind legs held further forward than normal. Some pigs find that getting to their feet after resting is difficult, and assistance may be needed in some cases. Viewed from in front, the pig may appear to have knock knees.

Diagnosis

Diagnosis is based on the history, the clinical signs, and the elimination of other causes of lameness. Osteochondrosis can be detected radiographically, showing irregularity of the articular cartilages and sometimes the presence of joint mice.

Synovial fluid increases in the early stages of the disease but later is reduced in quantity. The cellular content of the synovial fluid is normal and is not increased as it is in cases of septic arthritis.

Differential diagnosis

The clinician must be sure that no other causes of lameness are present, and a comprehensive clinical examination is required in every case. The clinician must check that there are no foot lesions, no infectious diseases of the joints, and no injuries (including fracture). Body temperature is normal in leg weakness cases but often raised in infectious arthritis.

Treatment

Treatment is not effective, as it is impossible to reverse the abnormalities present. In valuable breeding animals, non-steroidal anti-inflammatory drug (NSAID) therapy can be used for pain relief.

Control

The lack of a definite cause of the condition makes prevention difficult. Careful breeding from unaffected animals with monitoring of the performance of groups of siblings may be helpful.

Slipped epiphysis of the femoral head

Incidence

This condition (Figs 3.7 and 3.8) is not uncommon and is most frequently seen in young boars and less commonly in gilts at the beginning of their breeding life.

Aetiology

An existing defect in the epiphysis, possibly the result of dyschondroplasia, may result in a weak link between the

Fig 3.7 – Slipped epiphysis of the hip joint: lameness of the left hind leg.

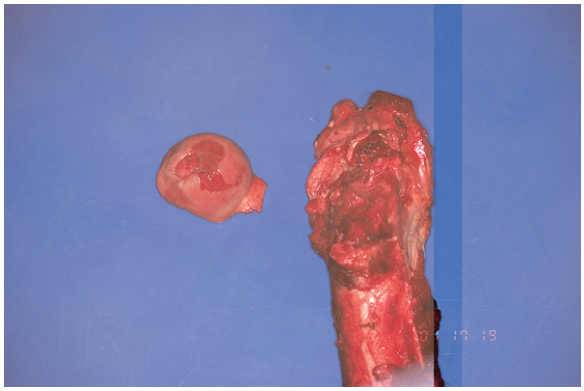

Fig 3.8 – Slipped epiphysis: separation of femoral head in post-mortem specimen.

femur and its femoral head epiphysis. Trauma sustained in attempts to serve or through fighting may cause the separation to occur. The epiphyseal growth plate does not fuse until the pig is about 3 years of age.

Clinical signs

Lameness involving one hind leg is seen. The severity of the lameness may increase over the course of a few days. The leg may just touch the ground or be carried when possible. Muscle wasting in the ipsilateral gluteal region occurs quite quickly. It may be possible to detect crepitus around the hip joint by touch or via auscultation using a stethoscope.

Diagnosis

The history and clinical signs are suspicious, but radiography provides a definitive diagnosis. Heavy sedation or general anaesthesia is required to restrain the animal for a good dorsal–ventral radiograph of the hip joint. The clinician must discuss the costs and practicalities of radiology with the owner.

Fig 3.9 – Pig polyarthritis: *Mycoplasma hyorhinis* infection. Note distension of left hock joint.

Differential diagnosis

Other causes of lameness, including the foot, should be considered. Femoral shaft fracture should also be considered. Such animals are usually totally non-weight bearing, and crepitus may be detected along the femoral shaft – although the heavy muscle mass of the upper limb makes this difficult.

Treatment

Euthanasia is required.

Infectious causes of lameness in pigs

Mycoplasma hyorhinis

Only lameness is considered here.

Incidence

The exact incidence of this condition (Fig. 3.9) is unknown, but it is quite common. It mostly affects pigs aged 3–10 weeks, often just before or just after weaning. *Mycoplasma* species were responsible for arthritis in 5.22% of the pigs suffering from musculoskeletal problems seen by the VLA in 1996–2003.

Aetiology

Mycoplasma hyorhinis is the cause.

Epidemiology

The infection may originate from an older pneumonia case. The organism is found in the nasal tracts of 60% of

pigs. The organism gains access to susceptible piglets and septicaemia develops. It settles in the joints, causing a polyarthritis.

Clinical signs

Affected pigs are dull, lame, and anorexic. Body temperature may rise to 40°C, and appetite is depressed. Growth rate is markedly depressed. Joints are swollen and warm to the touch; the hock, stifle, shoulder, and elbow are most frequently affected. The distended joint capsule is readily palpable on the anterior aspects of the hock joints.

Diagnosis

Diagnosis is based on the clinical signs. Joint fluid is serosanguineous or serofibrinous; there is an increase in numbers of plasma cells and lymphocytes.

Post-mortem – Post-mortem examination reveals joint changes, including villous hypertrophy of the synovial membranes.

Differential diagnosis

Other causes of lameness including *Haemophilus parasuis*, *Streptococcus suis*, *Actinobacillus pleuropneumoniae*, and *Actinobacillus suis*. Culture of joint fluid is required to confirm the exact cause. *M. hyorhinis* can be difficult to culture, and the help of a skilled laboratory may be required to confirm the diagnosis.

Treatment

Treatment consists of antibiotic therapy, for example oxytetracycline or tiamulin given for 5 days by intramuscular injection or in food or water if piglets are eating. If untreated, some mild cases may show self-resolution in 4 weeks, but treatment of the whole group is preferable to encourage complete resolution.

Mycoplasma hyosynoviae

Lameness only is considered here.

Incidence

This infection (Fig. 3.10) is quite common in the UK and in other pig-keeping areas. Mycoplasmas of various species causing arthritis were found to be responsible for 5.22% of the musculoskeletal problems diagnosed by the VLA in 1996–2003.

Aetiology

Mycoplasma hyosynoviae infection often follows stress.

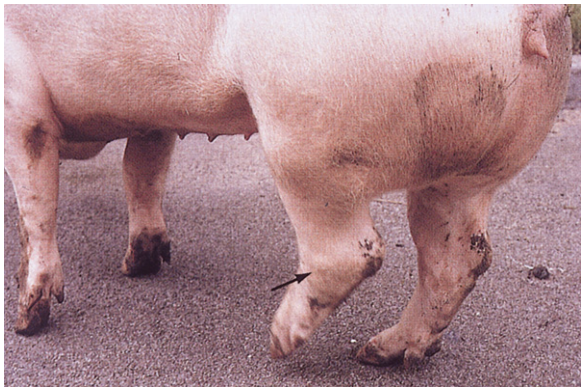

Fig 3.10 – Pig polyarthritis: *Mycoplasma hyosynoviae* infection. Note swelling of left hock joint and non-weight-bearing left leg.

Epidemiology

The infection mostly affects older pigs than those affected by *M. hyorhinis*. Pigs are affected in the 35- to 120-kg body weight range at approximately 10–26 weeks of age. Less commonly, the condition may also affect newly purchased breeding stock. The causal organism is carried in tonsils of many pigs.

Clinical signs

Clinical signs vary from slight to acute lameness. Body temperature is usually normal. Apparent self-cure of mild cases and regression of symptoms may be seen after a few days. Affected joints are swollen and may be warm to the touch.

The condition may affect all limbs or either the forelimbs or hind limbs. If the hind limbs are painful, pigs may adopt a 'dog sitting' posture.

Diagnosis

Clinical signs are suggestive of the condition, but to confirm one must isolate *M. hyosynoviae* from joint fluid. Joint fluid in affected pigs is yellow-brown and clear.

Differential diagnosis

Differential diagnosis includes osteochondrosis in breeding stock and other causes of infectious arthritis. *M. hyosynoviae* is more likely to respond to treatment than osteochondrosis is.

Treatment

Parenteral treatment by intramuscular injection with tiamulin, tylosin, or lincomycin for 3–5 days is used.

Glasser's disease

For full discussion of Glasser's disease, please see Chapter 4 (*Diseases of the respiratory system*). Only lameness is considered here.

Incidence

Glasser's disease occurs worldwide. The disease is not uncommon.

Aetiology

Haemophilus parasuis is responsible. This organism can cause polyarthritis. It affects pigs from weaning to 4 months of age.

Clinical signs

There is a sudden onset of lameness, and central nervous system and respiratory signs may also be seen. Body temperature may rise to 41°C. Joints are swollen, and affected pigs walk with short strides. Polyserositis is present in some pigs, which may develop pleurisy, peritonitis, and pericarditis.

Diagnosis

Diagnosis is by isolation of the organism from joints and complement fixation tests on serum.

Treatment

Treatment is by intramuscular injections of penicillin–streptomycin, oxytetracycline, or trimethoprim-sulpha for 3 days. In-contact pigs may be given oxytetracycline in their drinking water.

Prevention

Vaccination is used. A combined vaccine against Glasser's disease and enzootic pneumonia is available.

Streptococcus suis type 1

For full discussion, please see Chapter 7 (*Diseases of the nervous system*). Lameness only is considered here.

Incidence

Streptococcus suis type 1 is a common infection affecting pigs in the 10- to 14-day age group. It is carried by the sow and enters the piglet's body via tonsillar crypts. A septicaemia develops, localizing in the joints and other organs.

Clinical signs

One may see an occasional sudden death in the group. Several members of the litter are lame with swollen joints and are reluctant to move. Elevated body temperature, rising to 40–41°C, may be seen. Central nervous system signs such as fitting may be seen, as well as occasional cases of endocarditis.

Diagnosis

Diagnosis is by isolation of the organism. The joint fluid is serosanguineous.

Treatment

Immediate treatment with parenteral penicillin, ampicillin, or trimethoprim-sulpha is given for 3–5 days. NSAIDs may help in painful cases.

Control

See Chapter 7.

Streptococcus suis types 2 and 14

For full discussion, please see Chapter 7 (*Diseases of the nervous system*). Lameness only is considered here.

Type 14 is a potentially zoonotic organism.

Incidence

This is a common and important disease usually affecting weaned or finishing pigs. The organism usually affects older animals than those that are affected by *S. suis* type 1. Infection often follows stresses such as mixing and moving. Type 2 occurs worldwide. Type 14 is seen in the UK.

Clinical signs

There is an occasional sudden death, and then several pigs in the group show signs of lameness. One may see acute polyarthritis accompanied by severe discomfort. The body temperature is 40–41°C. Central nervous system signs may also be seen.

Diagnosis

Diagnosis is by history and clinical signs, and isolation of the organism.

Treatment

A course of antibiotics such as penicillin, ampicillin, or trimethoprim-sulpha for 3–5 days should be given. NSAIDs may help in painful cases.

Control

The infection is controlled by strategic medication (see Chapter 7).

Actinobacillus suis

Only lameness is considered here. For further discussion, please see Chapter 8 (*Diseases of the cardiovascular, haemopoietic, and lymphatic systems*). *Actinobacillus pleuropneumoniae* occasionally causes arthritis.

Incidence

The exact incidence is unknown, but the infection is seldom diagnosed.

Epidemiology

This is one of a number of organisms causing septicaemia in susceptible piglets. It is an opportunist pathogen.

Clinical signs

The clinical signs are sudden death, endocarditis, and polyarthritis. Only polyarthritis is discussed here. Affected piglets are usually <3 months of age. Some of the litter have elevated temperatures and are reluctant to move. Affected joints are warm and swollen.

Diagnosis

Clinical signs are not diagnostic, and isolation of the organism is essential to confirm the diagnosis.

Treatment

The whole litter or group should be treated with parenteral penicillin, ampicillin, or oxytetracycline. Enrofloxacin or trimethoprim-sulpha can also be used.

Swine erysipelas

For full discussion of this disease, please see Chapter 11 (*Polysystemic diseases*). Lameness only is considered here.

Incidence

Swine erysipelas was diagnosed as the cause of arthritis in only 1.04% of pigs suffering from musculoskeletal problems examined by the VLA in 1996–2003. Chronic arthritis caused by erysipelas is probably less common than that caused by other organisms such as *Arcanobacter pyogenes*. In many cases, the cause of arthritis is not determined, but the clinical signs of erysipelas listed below are quite specific. The condition mostly affects pigs at >12–16 weeks.

Aetiology

The cause is *Erysipelothrix rhusiopathiae*.

Epidemiology

Chronic erysipelas may follow the acute form of the disease, but often there is no history of this. The changes in the joint are of a *non-suppurative proliferative arthritis*. Damage to the articular cartilages can result in their loss, and the affected joints become dry, ankylosed, and immobile.

Clinical signs

Lameness caused by erysipelas may affect an individual or group of pigs. Affected animals are reluctant to get to their feet or take weight on their legs. They often stand with all four legs held close together under their bodies (Fig. 3.11). The carpi and hocks are most commonly affected, but other joints including those of the vertebrae may be involved. Body temperature is often normal. The affected joints may be warm and initially painful, but the joint capsule is seldom distended as in many other joint conditions. Later the joints are not warm but are enlarged, especially around the epiphyses. Joint movement is reduced, and affected animals are in severe discomfort.

Diagnosis

Diagnosis is by clinical signs and progression of the untreated disease.

Post-mortem – The joint capsule is thickened and has areas of granulation tissue. In advanced cases, there is loss of articular cartilage. A definitive diagnosis is based on culture of the organism from the joints.

Fig 3.11 – Chronic arthritis caused by erysipelas. Note lack of visible joint distension.

Differential diagnosis

Other causes of lameness must be eliminated by careful clinical evaluation and culture of joint contents and synovial membranes. Specific conditions that should be considered include Glasser's disease, *S. suis* type 2, *Mycoplasma* infections, the leg weakness syndrome, and diseases of the foot.

Treatment

Treatment can be unrewarding. In cases where there are chronic pathological changes in the joints, euthanasia may be advisable as the chance of recovery and normal growth is poor. In early cases, parenteral injections of penicillin for at least 5 days are advised. NSAIDs help to control pain.

Control

Control is by effective treatment of acute cases. A programme of vaccination is important to reduce the incidence of acute disease and the level of infection in the herd. Vaccination may not prevent the development of chronic lesions following acute infection.

Joint ill in neonatal piglets

Incidence

This condition (Fig. 3.12) is common. Piglets are affected in the first 10 days of life.

Aetiology

The causes are *Arcanobacter pyogenes*, *Streptococcus* species including sometimes *S. suis* type 1, *Mycoplasma* species, and *Haemophilus* species. In many clinical cases, the actual cause is unknown and may not be determined.

Fig 3.12 – Joint ill in neonatal piglet. Note distension of hock and carpal joints.

Epidemiology

Infection gains access through the navel soon after birth. Colostrum-deprived piglets are especially at risk. Hypothermic and hypoglycaemic piglets are also vulnerable. Infection spreads haematologically through the body.

It can also gain access through dirty syringes and needles, and through superficial skin lesions such as abrasions. Roughened floor surfaces or sharp mesh edges increase the risk of superficial damage in piglets.

Clinical signs

Affected piglets fail to come out of the creep to feed when called by their sow. If encouraged to stand, they are reluctant to take weight on all four legs. They feel hot to the touch, and body temperature is elevated to 40–42°C. One or more joints are distended, warm, and painful. The hock, carpus, and stifle joints are most commonly affected. Central nervous system signs rarely occur but occasionally there are signs of meningitis, in which case *S. suis* type 1 may be the specific cause (see below).

Diagnosis

Diagnosis is based chiefly on the age of the piglets and their clinical signs. Joint fluid is increased in volume and may be turbid and hypercellular.

Differential diagnosis

When possible, the cause should be ascertained by culture to determine whether a specific infection, such as *S. suis* type 1, is present.

Treatment

All members of the litter, affected and unaffected, should be treated. A 3- to 5-day course of antibiotics should be given by intramuscular injection. Penicillin–streptomycin, ampicillin, clavulanic acid, and amoxycillin are usually effective. Small doses of steroids or NSAIDs speed recovery in severely affected animals. The progress of the litter should be monitored to avoid the development of chronic cases (see *Chronic septic arthritis* below). Euthanasia may be necessary for non-responsive cases. Joint lavage can be considered in valuable animals.

Control

Check farrowing house hygiene and improve where necessary. Ensure piglets receive adequate colostrum.

Chronic septic arthritis

Incidence

The incidence of chronic septic arthritis (Figs 3.13–3.16) is quite high and likely to increase in difficult economic times when treatment for acute cases can be delayed or incomplete.

Aetiology

A range of organisms, especially *Arcanobacter pyogenes*, may be involved, although this may not have been the primary organism. Streptococci and possibly the mycoplasmas may have contributed to the primary pathology.

Epidemiology

Many are chronic cases from a previous episode of joint ill when young piglets are affected. The farmer's treatment of an affected litter may be a single injection of an antibiotic such as oxytetracycline; the majority of the litter appear to recover and no follow-up treatment is given.

Clinical signs

These are lameness and deformity. One or more joints are affected, and there is often severe loss of muscle mass

Fig 3.15 – Chronic arthritis in stifle joint. Note muscle wasting above joint.

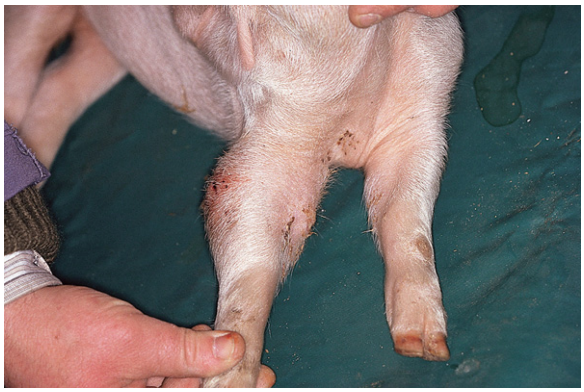

Fig 3.13 – Chronic arthritis in piglet: elbow joint (anterior view).

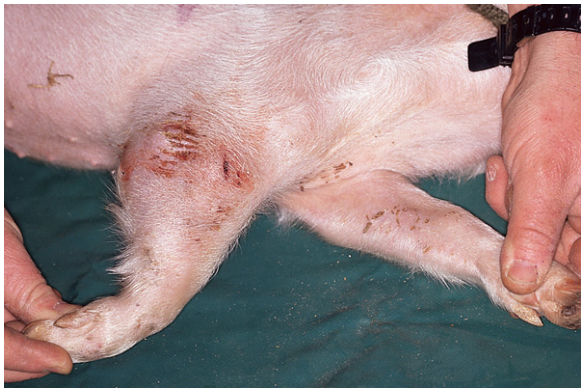

Fig 3.14 – Chronic arthritis in piglet: elbow joint (lateral view).

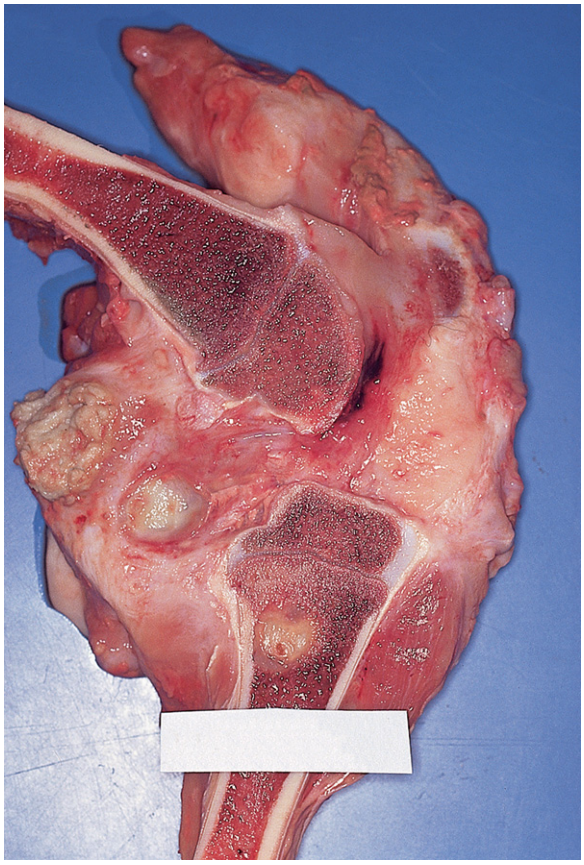

Fig 3.16 – Chronic arthritis in stifle joint: post-mortem appearance.

caused by the atrophy of disuse. The affected joints are swollen and sometimes abscessed. They may have opened spontaneously, in which case thick pus exudes from within. In many cases, the range of joint movement is greatly reduced and there may be total ankylosis. Affected animals are often in very poor condition and are unable to compete for food and water.

Diagnosis

Diagnosis is based on the history, clinical signs, and orthopaedic evaluation of the patients. Affected animals present a diagnostic challenge, but economic considerations preclude the ideal case evaluation. Chronically swollen joints may have severe bony damage including osteomyelitis, destruction of epiphyses and articular surfaces, and pathological fractures with some spontaneous repair. Others have little bony damage. An x-ray is helpful if treatment is contemplated and is economic to check that irreversible pathology is not already present.

Treatment

Treatment is seldom economical, and euthanasia may be the kindest option.

Chronic joint infection can be treated, with some success. Antibiotic therapy, NSAIDs, joint drainage, and lavage are used. Treatment may also include nursing care and what physiotherapy is possible.

Control

The unacceptable pathological changes that occur in untreated animals can easily be prevented by effective individual pig care. Early treatment of properly diagnosed disease and completion of courses of treatment are effective in dealing with most joint infections in pigs. The condition is largely preventable.

Foot and mouth disease

This is an acute **notifiable** viral disease that causes pyrexia and lameness. Lameness only is discussed here. The disease is considered further in Chapter 11 (*Polysystemic diseases*).

Incidence

A major outbreak of the disease occurred in the UK in 2001.

Aetiology

Apthovirus is responsible.

Epidemiology

The disease is highly infectious and has an incubation of 3–7 days. Direct and windborne aerosol spread occurs, and large amounts of virus are present in affected animals.

Clinical signs

There is a sudden onset of severe lameness. Vesicles are found on the coronet, interdigital space, and around supernumerary digits, less commonly on the tongue and snout. Body temperature is elevated, at 41°C. Vesicles rupture and the horn of hooves may slough, leaving exposed cuticle.

Diagnosis

Diagnosis is by enzyme-linked immunosorbent assay (ELISA) test on serum, and by polymerase chain reaction and complement fixation tests for virus on tissue.

Differential diagnosis

Differential diagnoses are other causes of lameness and swine vesicular disease.

Treatment and control

Treatment and control currently comprise a slaughter policy and Department for Environment, Food and Rural Affairs (Defra) restrictions.

Swine vesicular disease

Lameness only is considered here.

Incidence

This disease is **notifiable** but has not occurred in the UK for a number of years. It presents clinical signs that are very similar to those of foot and mouth disease. Unlike foot and mouth disease, swine vesicular disease affects only pigs.

Aetiology

A picornavirus is the cause.

Clinical signs

Lameness and high fever are the clinical signs. Body temperature may rise to 41°C, and affected animals show signs of severe discomfort if encouraged to move. Vesicles develop at the coronary bands of the feet, and separation of the hooves of the digits and supernumerary digits may occur. A small number of animals may show vesicular lesions elsewhere, including on the snout and tongue.

Epidemiology

The disease can be introduced by feeding infected pork products and also from pigs incubating the disease.

Diagnosis

The clinical signs suggest that the pigs are suffering from either swine vesicular disease or foot and mouth disease. Because both conditions are notifiable, Defra must be notified and will make a definitive diagnosis. ELISA tests are used for initial screening.

Differential diagnosis

Differential diagnoses are other causes of lameness, especially foot and mouth disease.

Treatment

The disease is currently subject to a slaughter policy. See Table 3.2.

Other bone and muscle problems

Fractures

Incidence

Fractures are quite common, especially in units where handling facilities for numbers of pigs are poor.

Aetiology

Fractures are caused by *trauma* – accidental, injuries inflicted by other pigs, falling during mating, and falling over brick rubbish in poorly maintained fields. Humeral fractures are also seen in cases of *electrocution*. Fractures may also develop as a result of bone disease such as osteomalacia (see below) and osteoporosis. The latter condition may occur in second-litter gilts, who may be particularly susceptible to fractures.

Clinical signs

There is a sudden onset of severe and acute lameness. The pig is usually unable to take weight on the affected limb, although it may attempt to do so. Muscle tone in the limb is reduced. The pedal withdrawal reflex is non-responsive. In lower limb fractures, there may be obvious deformity of the limb. In upper limb fractures, there may be soft tissue swelling and evidence of haematoma formation around the fracture.

Clinical examination

Clinical examination is difficult in suspected fracture cases. Sedation or general anaesthesia may be necessary to enable an effective examination to be carried out humanely. Upper limb fractures such as those involving the femur and humerus are difficult to evaluate. The heavy musculature and swelling of the affected limb make detailed bony palpation almost impossible. Abnormal bone mobility, crepitus palpated or auscultated, and the dramatic decrease in muscle tone around the fracture are often diagnostic. Radiography can be helpful but can be impractical and costly under field conditions.

Diagnosis

Despite the difficulties the case can present, a full and detailed clinical examination should be attempted. One must ensure that no other possible cause of lameness, such as severe foot pathology and leg weakness, has been overlooked.

Table 3.2 Summary of infectious causes of porcine arthritis[a]

Cause	Age affected (weeks)	Joint involvement	Other system involvement
Mycoplasma hyorhinis	3–10	++	Polyserositis, pneumonia
Mycoplasma hyosynoviae	10–26	++	–
Glasser's disease[b]	5–16	++	Polyserositis
Streptococcus suis type 1	1–2	++	Central nervous system meningitis, endocarditis
S. suis types 2 and 14	10–26	++	Central nervous system meningitis, pneumonia
Actinobacillus suis[c]	<12	++	Skin abscesses, pericarditis, pneumonia
Erysipelas	>12	+	Unusual to see other signs in erysipelas arthritis cases
Joint ill[d]	1–2	++	–
Chronic septic arthritis	>4	+++	–

[a]Laboratory help is usually necessary to confirm diagnosis. In practice, many cases are treated without full diagnosis of cause. Outbreaks must be investigated.
[b]*Haemophilus parasuis*.
[c]And occasionally *Actinobacillus pleuropneumoniae*.
[d]*Streptococcus*, *Staphylococcus*, *Arcanobacter pyogenes*, etc.

Treatment

Lower limb fractures can be successfully plastered using a resin-impregnated bandage. Management usually requires the pig being kept in a hospital pen on its own. Upper limb fractures have a very poor prognosis. Internal fixation does not work well in pigs because of their poor bone density. Transport to a slaughterhouse is forbidden on welfare grounds. Euthanasia may be required.

Muscle injuries

Muscles can be damaged as a result of fighting or in association with bone damage, for example in fractures. It can also occur when pigs fall or do 'the splits'. Deep bite wounds penetrating the skin may extend deep into muscle. Such wounds may require surgical repair and may become infected.

Muscular dystrophy

Incidence

Muscular dystrophy (Figs 3.17 and 3.18) is uncommon.

Fig 3.17 – Acute muscular dystrophy in a gilt. Note gross swelling of the left longissimus dorsi muscle.

Fig 3.18 – Acute muscular dystrophy showing involvement of longissimus dorsi muscle.

Aetiology

Vitamin E and selenium deficiency are the most important causes of muscular dystrophy, sometimes known as white muscle disease. Deficiencies cause a number of other clinical conditions in pigs, including mulberry heart and hepatosis dietetica. For further discussion, please see Chapter 8 (*Diseases of the cardiovascular, haemopoietic, and lymphatic systems*). Only muscular dystrophy will be discussed here.

Epidemiology

As in other species, the onset of this problem can be sudden.

Vitamin E deficiency can arise if the diet is rich in unsaturated fats. Barley treated with propionic acid can also be low in vitamin E. The vitamin is an essential dietary antioxidant and is involved in membrane protection.

Clinical signs

Sudden onset of pain and swelling occurs in one or more muscle groups.

Pigs in the group may have shown other signs of vitamin E deficiency, including sudden death. If muscles in the limbs are affected, the animal is reluctant to move. The longissimus dorsi muscles of the back can also be involved. Affected animals resent getting to their feet, moving, and turning. One or both sides may be involved, and painful firm swellings are palpable in the lumbar region.

Diagnosis

Diagnosis is based on history, feeding (low vitamin E and selenium levels in diet), and clinical signs.

Differential diagnosis

Differential diagnoses are injury by other pigs or by the boar mounting gilts and damaging their backs with his feet, as well as back muscle necrosis – part of the porcine stress syndrome (see above). Overlying skin damage may be seen in cases of swelling caused by injury, and an ultrasonographic scan will clearly demonstrate lesions such as haematomata or abscesses. Muscle biopsy might help confirm the diagnosis. Back muscle necrosis is never fully resolved, and areas of fibrosis in the affected muscles are present after the acute phase. Muscular dystrophy resolves completely following treatment.

Treatment

Pigs are treated with intramuscular injection of vitamin E/selenium. Dietary deficiencies are corrected. NSAIDs may be needed in the acute phase, which can be very painful.

Rickets

Incidence
The condition may arise through inadequate levels of calcium, phosphorus, or vitamin D, especially in home-mixed diets.

Aetiology
The cause is a deficiency of calcium, phosphorus, or vitamin D in the diet. Some cereal-based rations have very high phosphorus levels but are low in vitamin D and calcium.

Epidemiology
When seen, the condition usually involves rapidly growing young animals.

Clinical signs
The clinical signs are lameness and reluctance to move. Epiphyseal regions of long bones are enlarged, and there may be bending of the diaphysis. Enlargement of the costochondral junctions may be seen or palpated (rickety rosary). Tooth development is delayed or inhibited.

Diagnosis
The condition is diagnosed by low levels of calcium, phosphorus, or vitamin D in the diet. Radiography shows very poor ossification of the bones.

Differential diagnosis
The rarity of rickets means that the animals must be carefully checked to make sure no infectious causes of lameness, such as *M. hyorhinis* or *M. hyosynoviae* and the bacterial causes of arthritis, are present. Analysis of the diet will confirm if any related elements are deficient.

Treatment
Treatment comprises correct diet and parenteral injections of vitamin D.

Control
Ensure adequate supplementation of diet.

Osteomalacia and osteoporosis

Incidence
The condition is uncommon but is seen occasionally in individual or groups of mature animals. It should not occur on well-run farms using a correctly balanced diet.

Aetiology
The cause is a deficiency of calcium, phosphorus, or vitamin D in the diet of animals in which endochondral ossification is complete. Osteoporosis occurs, and the bones are no longer strong enough to provide an effective endoskeleton. Bone is replaced with unspecialized osteoid.

Epidemiology
The condition may arise through inadequate dietary supplies of calcium, phosphorus, and vitamin D. It may occur in sows with a prolonged lactation and an inadequate diet. The sow's demand for calcium cannot be met by her diet, and the element is removed from the bones to supply the piglets in their milk.

Clinical signs
The owner may find a sow – often after weaning and mixing – with a femoral fracture involving the shaft or the neck. Radiography confirms the presence of the fracture and poor calcification of bone. Abnormal bending of bones is seen in some cases, but a fracture is the most common outcome.

Diagnosis
Diagnosis is based on the clinical signs, diet analysis, radiography, and bone analysis.

Treatment
Treatment of femoral fractures is unsatisfactory (see above); euthanasia may be necessary.

Control
Ensure adequate supplementation of diet.

Osteodystrophia fibrosa

This condition has some similarities to osteomalacia. In this condition, the bone depleted of calcium is replaced with flexible fibrous tissue. It occurs when pigs are fed on diets very high in phosphorus. The condition may develop if the calcium : phosphorus ratio is >1 : 2. The higher the phosphorus levels, the more rapid the onset.

Clinical signs
These include lameness and a reluctance to move. Pain on standing is seen, and many of the long bones become bent and deformed.

Treatment

Treatment is not possible, and affected animals must be euthanized.

Prevention

The condition is prevented by ensuring an adequate balance in dietary supplements.

Osteitis and proliferative osteitis

Incidence

The exact incidence is unknown. Lameness has been reported in gilts shortly after weaning, involving either the forelegs or the hind legs. Proliferative osteitis has been found on the medial condyle of the humerus and on the greater trochanter of the femur (Fig. 3.19). In both cases, the gilt is reluctant to stand and take weight on the affected limb.

Aetiology

This is not known, but there is an apparent bony response – at the point of attachment of muscle masses – to trauma on exertion. This may be exacerbated by osteoporosis. Bony enlargements may be palpated on the distal medial humerus, and osteophyte growth with poor radiolucency has been found adjacent to the femoral trochanter. Fractures may occur in the affected bones.

Treatment

Treatment is not possible, and humane slaughter of affected animals is recommended. Calcium may have been lost from bones during lactation, and poor flooring may exacerbate the problem.

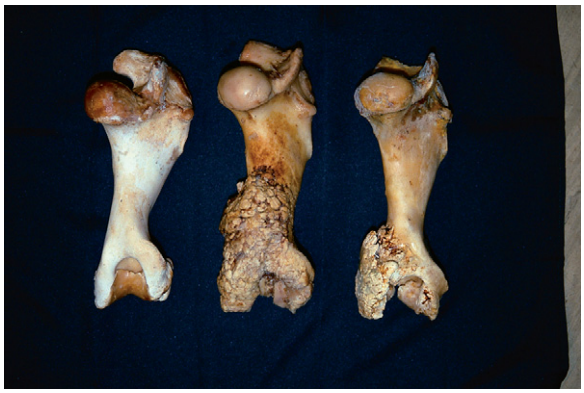

Fig 3.19 – Pig humeri showing lesions of osteitis. (Courtesy of R.W. Blowey.)

Prevention

Ensure good feeding in pregnancy and lactation, and maintain satisfactory calcium : phosphorus ratio. Avoid uneven or slippery flooring.

Limb lesions caused by pressure and unsuitable bedding

Incidence

These lesions (Fig. 3.20) are very common.

Aetiology

These lesions are caused by unsuitable or inadequate bedding to prevent damage to the skin and deeper tissues, especially over the pressure points of the body.

Epidemiology

Metal mesh flooring, rough concrete surfaces, lack of straw, and slatted floors are among surfaces likely to predispose to these lesions. Wet and dirty conditions, poor ventilation, and overcrowding exacerbate the problem.

Clinical signs

The knees and hocks, and less commonly the anterior surfaces of the fetlock joints, are most frequently affected. The severity of the lesions depends on the severity of the damage sustained and the length of time the pig is exposed to it. Areas of *excoriation of the skin* with *local skin necrosis*, and in some cases *abscess formation*, occur in adjacent tissue. *Hygromata* – subcutaneous bursae filled

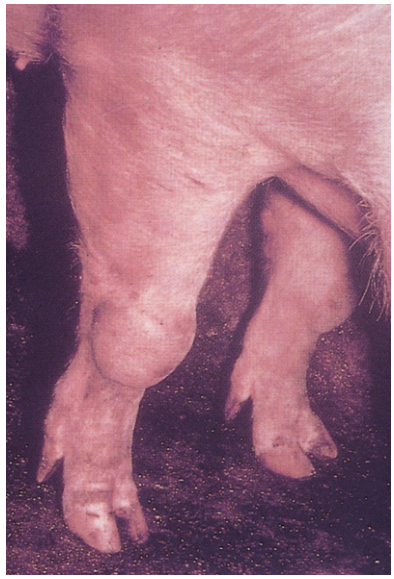

Fig 3.20 – Bursal swelling over the carpal joints.

with synovia-like fluid – develop, and these sometimes also become infected. In other cases, chronic irritation of the skin causes hyperplasia and the development of *calluses*. Lameness associated with this group of lesions is relatively uncommon.

Treatment

In the absence of infection, no treatment is normally given. Pigs should be moved to better accommodation or better bedding provided. Abscesses are drained in the normal way, and superficial areas of infection may be treated by application of topical oxytetracycline spray.

Prevention

Good husbandry with adequate bedding will eliminate most of these problems.

Foot lesions in pigs

Foot lesions in piglets

Surveys have shown that foot lesions in baby pigs are very common in many systems. A survey of 356 piglets found that 100% of piglets showed evidence of *sole bruising* by 24 days of age, many showing signs by 4 days of age. Forty-nine per cent of piglets had *sole erosions* (Fig. 3.21). Heavier piglets were more likely to be affected than lighter piglets. Sixty-one per cent of piglets showed *carpal abrasions*, on the anterior aspect of the carpus, and 71% had healed wounds. These mostly healed within a few days.

Round weld mesh and lack of bedding increased the incidence of solar bruising. Similar weld mesh, an uneven floor, and sparse wood shavings predisposed to skin abrasions.

These minor injuries can allow entry of opportunist pathogens that can cause local or systemic infection. Cushioned floors have been used successfully under experimental conditions. Applying plasters to abrasions helped healing but would not be popular commercially.

Local infection and crushing injuries by the sow are likely to cause lameness on one or more limbs. The various causes of polyarthritis in baby pigs were discussed above. Early treatment with antibiotic injections will normally resolve local foot infections in piglets.

Foot lesions in adult pigs

These are very common, and examples are seen in almost any group of adult pigs. Cases are usually presented for treatment only if the animal is lame.

Examples of foot lameness are given below.

Overgrown claws

One or both claws of both forelegs and hind legs may be affected (Fig. 3.22). Unsuitable diet, lack of exercise, and lack of exposure to firm surfaces can all predispose to the problem. Affected claws may crack and possibly become infected. They can be trimmed using cattle hoof shears in a feeding pen or farrowing crate. Sedation may be necessary if offering of food does not facilitate examination and trimming of the feet.

Broken claws

These may require tidying up or treatment if infected. If many cracks are present, biotin deficiency may be involved (please see below).

Bush foot or 'bumble foot' ('foot rot')

This lesion (Fig. 3.23) is very common and causes affected pigs considerable distress. The animal is reluctant to walk

Fig 3.21 – Solar erosion on piglet feet. (Courtesy of R.W. Blowey.)

Fig 3.22 – Overgrown toes in a sow.

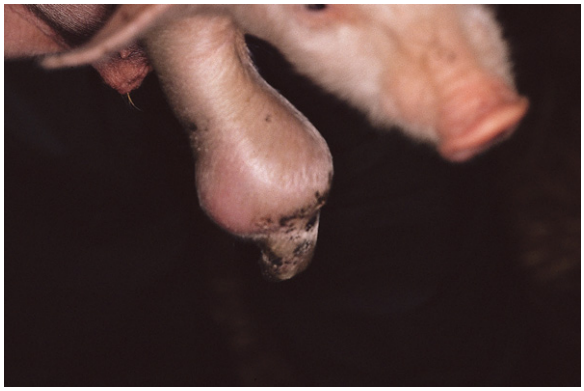

Fig 3.23 – 'Bumble foot'.

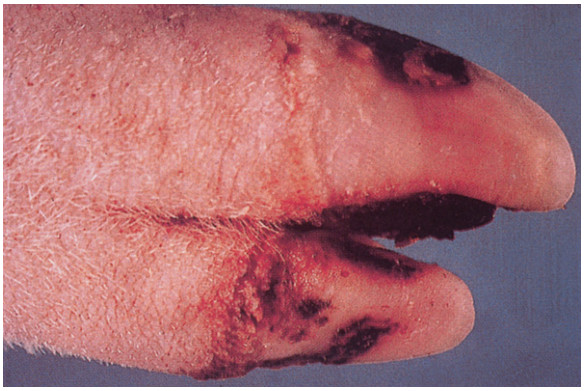

Fig 3.24 – Biotin deficiency in a pig. Note cracks and poor hoof quality.

or place the infected foot on the ground. The animal may try to put the foot down and then tap it repeatedly on the floor as it tries unsuccessfully to put it down comfortably. The term *foot rot* has been used, but the problem is really one of pedal sepsis, with infection in a variety of soft tissues and occasionally bone. One or both toes may be enlarged, warm, and very painful to the touch. Once established, infection may spread upwards and break through at the coronary band. Ideally, the sow should be examined quietly in lateral recumbency. Please see Chapter 1 (*Investigation of clinical problems on pig farms*). Sedation or light anaesthesia may be necessary to carry out a diagnostic clinical examination.

Infection usually gains access through a puncture wound or crack in the hoof sustained when walking over rough ground etc. A variety of organisms may be involved, including *Fusobacter necrophorum*, *Arcanobacter pyogenes*, and spirochete species.

Treatment

Animals are treated by aggressive antibiotic and NSAID therapy – good results can sometimes be obtained even in chronic cases – but a full course of antibiotic must be given. Occasionally, surgical drainage under sedation may be required. Other techniques, such as poulticing, have been used. Radiography of the foot, if economically justified, may be necessary if a *pedal bone fracture* or *osteomyelitis* is suspected. Amputation of the digit may be necessary in non-responsive cases. Complications such as *solar proliferation* and *corn formation* may occur; uneven wear of a claw, caused by injury or conformation, may result. Careful use of a foot knife can be helpful in such cases.

Laminitis

Laminitis may occasionally follow post-parturient infection, including mastitis–metritis–agalactia.

The affected sow shows signs of painful forefeet, and a digital pulse can be detected in the fetlock. The hooves are very warm to the touch and may show pain on percussion. The affected animal may be reluctant to rise, and walking is very uncomfortable. Treatment may be attempted by hosing the feet with cold water and prescribing NSAID therapy.

Biotin deficiency

Biotin has been found to be essential for the maintenance and integrity of the hooves in pigs and other animals. Deficiency can lead to lameness and the development of cracks in the walls and soles of the hooves (Fig. 3.24).

Incidence

Modern diets should contain adequate biotin (>220 g/kg), but problems can arise with home-mixed food. Biotin levels can be marginal in some cereals.

Epidemiology

Prolonged biotin deficiency is required to produce symptoms.

Clinical signs

Several sows in a herd may show signs of lameness and reluctance to take weight on their feet. Mating can be difficult, as the additional weight of the mounting boar increases the foot discomfort of the sow. Litter size may also fall in biotin-deficient herds. The feet are sensitive to the touch, and affected animals show resentment if their toes are squeezed. Cracks are seen in the walls (originating near the coronary band) and also in the soles. The feet

must be thoroughly cleaned and the pig properly restrained to allow lesions to be seen. The pads of the soles are softer than normal.

Diagnosis

The condition is diagnosed by clinical signs and low blood biotin levels (<700 ng/L).

Differential diagnosis

Differential diagnoses are other causes of foot lameness (see above), including laminitis and infected feet. Laminitis is not normally accompanied by cracks in the horny tissues of the feet. Careful clinical examination should detect other abnormalities.

Treatment

Supplement biotin levels in diet. It may take some months for the full benefit of this to be seen.

4

Diseases of the respiratory system

Introduction

Respiratory diseases can cause great distress to individual animals. They also have a serious effect on performance and production in affected groups of pigs. They are a major problem to the pig industry, and the potential cost of outbreaks of respiratory disease is high. A number of pigs may die, and there may be a reduction in food conversion efficiency and a decrease in meat quality in those that survive. Pigs that survive but are left with pathological lesions may also have a greater risk of condemnation at slaughter.

Aetiology of respiratory disease

Various causes of respiratory disease have been identified, including viruses, mycoplasmas, bacteria, and parasites. Secondary bacterial infection of lung tissue already compromised by primary pathogens frequently occurs. Poor biosecurity predisposes to the introduction and spread of respiratory diseases. In many cases, the causal agents are highly infectious. They may spread by direct or aerosol contact between pigs and be transmitted by birds and vehicles in addition to airborne spread between units.

Epidemiology of respiratory disease

A large number of diseases can affect the respiratory system. More than one respiratory disease can affect a unit at the same time. The porcine respiratory system has physical and immunological defence mechanisms against disease. These defences can be overcome by overwhelming levels of infection, poor immunity, poor management, adverse environmental factors, and the presence of other diseases. Poor ventilation and insulation, poor diet, an inadequate water supply, overcrowding, and high levels of dust and ammonia are particularly important environmental risk factors. Experimental work has shown that exposure to high levels of dust and ammonia do not necessarily result in an increase in respiratory disease. A failure to recognize and treat respiratory disease at an early stage predisposes to severe outbreaks of respiratory disease. These exacerbate the negative effects of respiratory diseases on growth and production. Failure to segregate the various age groups on the unit may also predispose to outbreaks of disease, including those affecting the respiratory system. This is especially likely to occur where there is common airspace or airflow for all ages of pig.

Clinical signs of respiratory disease

One of the first signs of respiratory disease is coughing, which may be particularly noticeable when pigs get up from rest. Coughing is especially severe in cases of enzootic pneumonia when large numbers of pigs in a group are affected. Dyspnoea accompanies a number of respiratory diseases. If breathing is slow and accompanied by snuffling sounds it may indicate the presence of nasal obstruction, which often accompanies rhinitis.

Dyspnoea with hyperpnoea may indicate severe compromise of lung function. In cases of severe respiratory distress, affected pigs may breathe through their open mouth and may have a 'respiratory line' caused by tightening of the abdominal muscles as they attempt to force air from inelastic lung tissue (Fig. 4.1).

Heart failure and cor pulmonale may develop in animals suffering from severe or chronic respiratory disease. Pleurisy with adhesions between lung tissue and the chest wall may further compromise respiratory function and efficiency.

Only a limited physical clinical examination may be possible in pigs suffering from respiratory disease. Brief auscultation of the chest may be possible, and increased lung sounds and specific sounds of pneumonia and pleurisy may be audible. Wheezing sounds may indicate narrowing of the airways; bubbling sounds may indicate intermittent blocking of bronchioles. High-pitched squeaking sounds may indicate the presence of pleuritis, and harsh rubbing sounds may occur in cases of pleurisy. In some cases, it may be possible to identify which parts of the lungs are affected and whether there are signs of cardiac disease or pleurisy. Any attempt to restrain the pig may increase the severity of the clinical signs, and it may be life-threatening in severe cases.

Animals with respiratory disease are often anorexic and soon have an empty appearance. An ocular discharge may be present, and a nasal discharge may be seen in cases of rhinitis. Death of untreated pigs and sudden deaths may occur with a number of respiratory diseases. Post-mortem examination can be extremely helpful in confirming the diagnosis of the cause of the respiratory disease. The distribution and nature of gross lesions can be observed and samples taken for diagnostic purposes.

Some respiratory diseases, such as enzootic pneumonia and porcine reproductive and respiratory syndrome

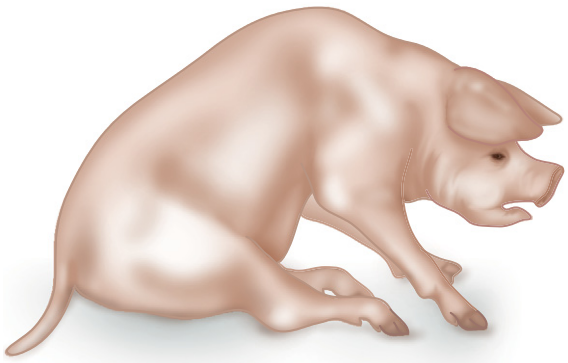

Fig 4.1 – Pig in severe respiratory distress. (Redrawn from Jackson PGG, Cockcroft PD. Clinical Examination of Farm Animals. Oxford: Blackwell. © 2002 Blackwell.)

(PRRS), readily become endemic in affected herds. When first introduced, they may be quite acute but later become chronic. Such diseases have an adverse effect on welfare and production. Other diseases, such as swine influenza and some outbreaks of pleuropneumonia, are more acute and may have a shorter course on affected units.

Porcine respiratory diseases remain a challenge to the pig industry. Outbreaks of such diseases should be investigated quickly and thoroughly. Treatment and preventive measures must be put in place to ensure that the adverse effects of diseases are kept to a minimum.

Diagnosis of respiratory disease

Accurate diagnosis of the cause is essential in outbreaks of respiratory disease. The history of the unit, clinical examination, and observation may provide a tentative diagnosis. This must be confirmed by specific laboratory tests and a full post-mortem examination of dead or very sick pigs. Information from abattoir surveillance of slaughtered pigs may indicate the nature and level of any existing respiratory disease. More than one respiratory infection may be present on a unit at the same time. It is essential if all potential causes are identified to ensure that effective and specific control methods can be instituted.

Treatment of respiratory disease

Once the diagnosis has been determined, treatment can commence and plans for future prophylaxis discussed with the owner. In recent years, effective vaccines have been developed for many of the major porcine infectious respiratory diseases. New antibiotics have been developed that have improved penetration into lung tissue, permitting more effective treatment of primary and secondary infections. Whenever antibiotics are used, care must be taken to ensure that meat withdrawal times are observed.

Antibiotic therapy is usually prescribed and can be administered in the water or food to affected and at-risk animals. Sick pigs are usually treated parenterally, as they will often be too ill to either eat or drink. Severely ill animals may be euthanized on humane or economic grounds.

Control of respiratory disease

Ideally, infectious respiratory diseases should be eliminated from the unit or their incidence and severity reduced to controllable levels. Once eliminated or controlled, future levels of disease should be monitored. This can be done by careful observation, clinical examination of sick pigs, and post-mortem examination of any pigs that die. The respiratory systems of slaughter pigs should be examined and monitored at the abattoir. All infections present should be identified.

Elimination of respiratory disease may be attempted by a number of methods. Some of these are discussed below in relation to specific diseases. All require determination and dedication, especially by farm owners and staff. Potential elimination methods include the establishment of a specific pathogen-free (SPF) herd from hysterectomy-derived stock. Alternatively, early weaning and segregation or strategic medication can be used. Once high health status is achieved, it must be maintained by high herd biosecurity. Vaccines are available for some but not all respiratory infections.

Comparative notes on some infectious diseases of the porcine respiratory system are presented in Table 4.1.

Veterinary Investigation Diagnosis Analysis sample submissions: respiratory diseases, 1996–2003

See Table 4.2.

Enzootic pneumonia

This is a very important disease of pigs that causes severe economic loss through clinical disease and an adverse effect on food conversion and weight gain.

Incidence

This disease has widespread occurrence throughout the world. It is probably the most important and most common porcine respiratory disease in intensive pig units, and has low mortality but high morbidity.

Aetiology

The cause is *Mycoplasma hyopneumoniae* with frequent superimposed secondary infection, especially by

Table 4.1 Comparative notes on some infectious diseases of the respiratory system

Disease	Incidence	Age affected (weeks)	Severity	Special diagnostic points
Enzootic pneumonia	+++	3–26	+	Typical post-mortem: ventral consolidation ELISA serum PCR nasal swabs
Pleuropneumonia lesions	++	8–24	+++	Typical post-mortem: diaphragmatic lobe lesions ELISA serum Culture organism
Glasser's disease	+	5–16	++	Post-mortem: polyserositis ELISA serum Culture organism
Pasteurella multocida	++	10–20	+++	Culture organism Eliminate other causes
Swine flu	++	All ages	+	Rapid onset, rapid disappearance ELISA PCR nasal swabs
Porcine reproductive and respiratory syndrome	+++	After weaning	++	ELISA serum PCR virus

ELISA, enzyme-linked immunosorbent assay; PCR, polymerase chain reaction.

Table 4.2 Veterinary Investigation Diagnosis Analysis sample submissions: respiratory diseases, 1996–2003

Cause	Incidence (%)
Pneumonia not specific	28.19
Pasteurella multocida	24.68
Pleuropneumonia	17.03
No diagnosis	10.58
Swine influenza	7.42
Mycoplasma hyopneumoniae	4.23
Haemophilus parasuis	2.30
Rhinitis	1.04
Other diseases	0.89
Progressive atrophic rhinitis	0.89
Pleurisy	0.86
Inclusion body rhinitis	0.75
Haemophilus sp.	0.58
Bordetella bronchiseptica	0.37
Parasitic pneumonia	0.20

Pasteurella multocida (type A). The disease can also be worsened by the simultaneous presence of atrophic rhinitis and ascariasis. *Mycoplasma hyopneumoniae* alone can cause lung damage, but this will usually resolve spontaneously. Environmental problems and secondary infection are responsible for the more chronic signs and poor performance. Infection can be induced by the administration of aerosol cultures of *Mycoplasma hyopneumoniae*.

Epidemiology

The organism lives in the pig's respiratory system, where it can compromise epithelial function. On entering the body, it becomes attached to the ciliated cells of the trachea, bronchi, and bronchioles. The organism survives for a short time only in the environment out of the body. Spread is mostly by pig to pig contact, by aerosol transmission, and also by wind. Poor housing, variable temperatures, high humidity, mixing of pigs from different sources and ages, overcrowding, and continuous throughput systems all predispose to a greater severity of disease. The numerous contributory aetiological factors make assessment of each factor difficult. Immunity can be short-lived, and there is no transfer of colostral immunity from sow to litter. Infection can pass from the sow to her piglets in the first few days of life.

Infection of a naive herd may lead to signs of pneumonia in all ages, from 10-day-old piglets to sows. In *chronically infected herds*, the growing (post-weaning) pig is most often clinically affected. Up to 90% of pigs in the latter herds may show lung pathology at slaughter. Daily weight gain may fall by up to 17% and feed efficiency by 14%. Between 23 and 37 g in weight gain is lost per day for every 10% of lung affected by pneumonia. The appearance of lungs at slaughter, although useful, is not necessarily a good indication of the true extent of a herd infection.

Clinical signs

Prolonged non-productive coughing, worsened by exercise, is the main sign of the disease in affected herds. Individual animals, especially those with secondary infection by *P. multocida*, may show signs of severe respiratory distress. Such animals are sometimes called panters, with temperature 40–42°C and possibly open-mouthed breathing. A more productive cough may be heard in such animals. Increased lung sounds may be heard if the

clinician is able to auscultate the chest. Pleurisy and pericarditis may complicate some cases.

Diagnosis

Diagnosis is based on the herd history, clinical signs, and specific laboratory tests. A diagnostic serum enzyme-linked immunosorbent assay (ELISA) test and polymerase chain reaction (PCR) are also available. *Mycoplasma hyopneumoniae* can be detected in tissue (e.g. by an antigen ELISA test, culture, and PCR). *Mycoplasma hyopneumoniae* is, however, very difficult to culture.

Post-mortem – Areas of consolidation of the ventral parts of apical, cardiac, and diaphragmatic lung lobes adjacent to normal tissue are seen (Figs 4.2 and 16.12).

Differential diagnosis

Differential diagnoses include other porcine respiratory diseases, especially the following.

- *Actinobacillus pleuropneumoniae*. This tends to be more acute and has a high mortality rate, and the lungs have a characteristic gross pathological appearance at post-mortem. It affects the dorsocaudal parts of the lung lobes rather than the craniioventral areas.
- *Metastrongylus apri* infestation in outdoor pigs; parasites are found in the bronchi.
- *Swine influenza*. This has a short course and is less common, and the pathological changes are chiefly in the upper respiratory tract.
- *Glasser's disease*. There is sudden onset of polyserositis, with joints affected. Culture *Haemophilus parasuis*.

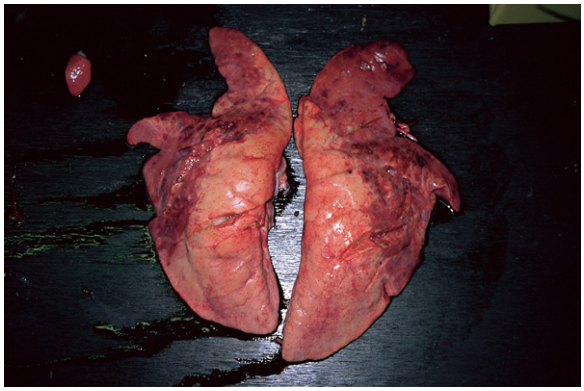

Fig 4.2 – Enzootic pneumonia: ventral consolidation of lungs. (Courtesy of R.W. Blowey.)

Treatment

Acute cases may respond to antibiotic therapy (e.g. tylosin, tiamulin, and enrofloxacin). Florfenicol and tulathromycin have recently (2003–2004) been licensed for use in pigs. Early treatment is essential, and a full course of treatment must be given. Tilmicosin is given in food for 15 days. Valnemulin premix is an option. Lincomycin is also effective. Steroids or non-steroidal anti-inflammatory drugs (NSAIDs) may aid resolution of acute individual cases.

Strategic dosing of growing pigs may be necessary on some farms if signs of respiratory disease appear.

Control

Various strategies are available depending on the severity of disease and economic considerations. These include the following.

- Improve environment and management. Make sure all aspects of husbandry, such as ventilation and the separation of different age groups, are as good as possible.
- All in, all out management of growers. After each batch, the building is pressure washed, fumigated, and rested for as long as possible before the next batch of pigs is admitted.
- Depopulate infected herd and – after thorough cleaning, fumigation, and resting – repopulate with 'disease-free pigs', but great care must be taken to ensure that they are really enzootic pneumonia-free.
- Repopulate with hysterectomy-derived pigs. Piglets obtained in this way are reared artificially away from their mothers. Such closed SPF herds are costly to produce and difficult to maintain.
- Partial depopulation and treatment. In this system, an attempt is made to keep the original breeding stock but remove (to other premises) all other pigs in the unit. The breeding stock pigs are treated with tiamulin in their water or food for 10 days. Future offspring should be relatively free of enzootic pneumonia.
- Medicated early weaning. In this system, farrowing sows are fed on a ration containing tiamulin until their piglets are 5 days old. The piglets are then removed and reared in isolation. They are kept on oral tiamulin in their food until they are 10 days old. They are then grown on away from other pigs on the farm.
- Vaccination (see below).

Monitoring the incidence of enzootic pneumonia

This includes examination of batches of lungs at slaughter and serum profiling of the herd using an ELISA test.

Vaccination

Inactivated, adjuvanted vaccines against *Mycoplasma hyopneumoniae* have helped control enzootic pneumonia very effectively in some herds. In some cases, a single dose of vaccine given at about 1 week of age is used, and in some cases a second dose is used 3 weeks later. Data sheets should be consulted before use. Trials of the vaccines have shown reduced mortality, better food conversion, and reduced costs. An inactivated vaccine is also available for the control of both enzootic pneumonia and Glasser's disease. Trials have been held to compare the efficacy of vaccination against enzootic pneumonia with herd medication. Both have been shown to help, but there appears to be little benefit in using both methods at the same time.

Progressive atrophic rhinitis

This is an important disease of pigs that adversely affects their welfare, food conversion, and growth rate. Progressive and non-progressive forms of the disease have been described depending on whether nasal turbinate damage is present.

Incidence

The disease has worldwide distribution, adversely affecting production and the welfare of affected pigs in intensive units. Growth rate can be reduced, and the predisposition to pneumonia is increased. Up to 50% of pigs in an infected herd may show evidence of snout deformity in abattoir surveys. Poor housing, overcrowding, and insufficient ventilation can increase the incidence of the disease.

Aetiology

Toxigenic strains of *Bordetella bronchiseptica* and *P. multocida* type D cause this disease. In the progressive form, toxigenic *P. multocida* is present.

Epidemiology

The infection can spread by direct or droplet contact between pigs. The main route of spread is between an infected sow and her litter in their first week of life. The effects of infection are worse in pigs affected at this early age than in older piglets. Further spread occurs when batches of piglets are mixed, for example in flat deck systems after weaning.

Bordetella bronchiseptica is a common inhabitant of the pig's nasal cavity. Infection with this bacterium results in mild, reversible, and temporary turbinate damage and eventual elimination of infection. Occasionally, rhinitis, tear staining, and pneumonia are seen in suckling piglets. The organism produces a cytotoxin that assists *P. multocida* gain access to the turbinate bones. This latter organism produces an *osteolytic toxin* that has a predilection for the turbinate bones. The turbinates are progressively destroyed with secondary involvement of the nasal mucosa. Growth rate may fall by up to 13%; this is thought to be due to the nasal irritation and obstruction that interferes with suckling and prehending food.

Clinical signs

In most cases, these are first seen at 3–9 weeks of age. Piglets sneeze and have a clear or purulent nasal discharge. They may rub their blocked noses on the floor. Once infection by *P. multocida* is established, nasal haemorrhage may be seen. Later, evidence of facial deformity (Figs 4.3 and 4.4) caused by underlying turbinate damage (Figs 4.5–4.7) is seen. If both nasal cavities are affected, the snout may be 'dished' and concave. In unilateral infection, lateral deviation of the snout is seen. Malocclusion of teeth may occur, and the pig has difficulty in prehending its food. There may be reduced pheromone recognition in breeding animals.

Diagnosis

Causal organisms can be cultured from the nose. Serological tests are available for *B. bronchiseptica* but not for *P. multocida*.

Post-mortem – This reveals a degree of destruction of the turbinates. The snout is sectioned at the level of the second premolar, and damage is estimated on a scale of 0 (no turbinate damage) to 5 (almost total loss of turbinates). The nasal septum may be twisted or displaced.

Fig 4.3 – Progressive atrophic rhinitis: deviation of the snout.

Fig 4.4 – Progressive atrophic rhinitis: deviation of snout.

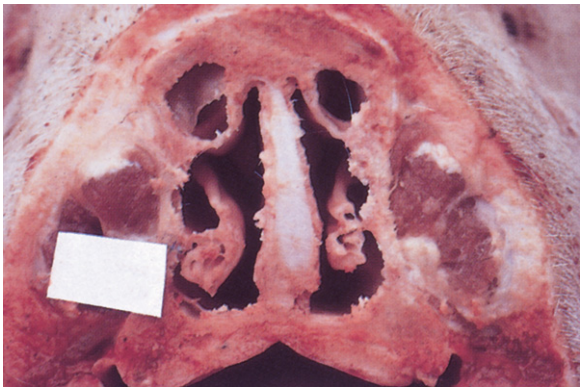

Fig 4.5 – Progressive atrophic rhinitis: early turbinate damage.

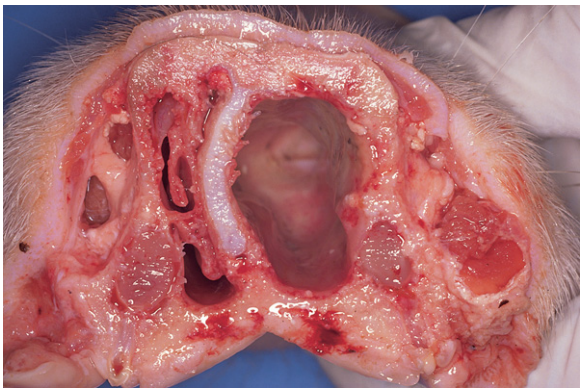

Fig 4.6 – Progressive atrophic rhinitis: unilateral turbinate destruction.

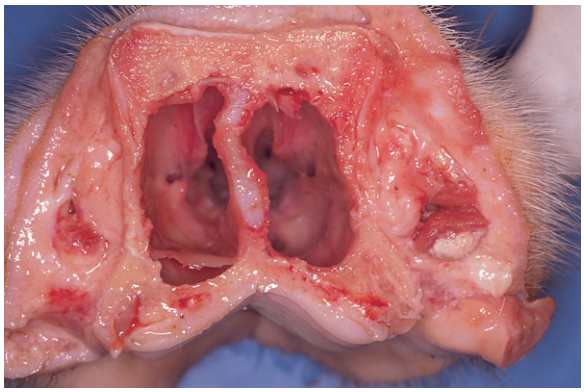

Fig 4.7 – Progressive atrophic rhinitis: bilateral turbinate destruction.

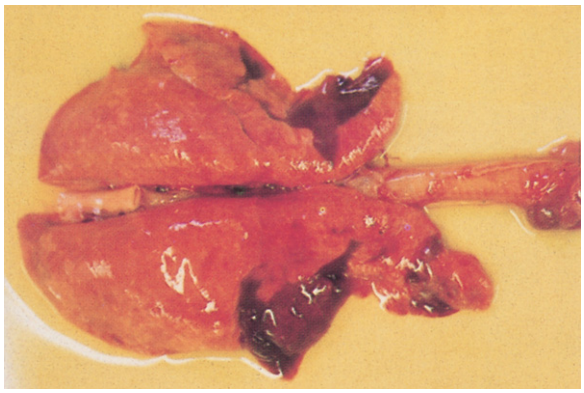

Fig 4.8 – *Bordetella bronchiseptica* pneumonia. (Courtesy of R.W. Blowey.)

Differential diagnosis

Differential diagnoses are other forms of rhinitis.

- *Bordetella bronchiseptica* infection by itself causes catarrhal rhinitis, tear staining, and occasionally pneumonia in pigs >1 week but usually no signs of snout deviation in older pigs. Culture of the organism and a serological ELISA test are used to confirm the diagnosis (Fig. 4.8).
- *Inclusion body rhinitis* (please see detailed description below). This is a milder disease usually having only a transient effect and no turbinate damage. A specific ELISA test and the finding of inclusion bodies (by microscopy and histopathology) in nasal discharges confirm the diagnosis.
- *Swine influenza* (please see detailed description below). This usually affects older pigs, with acute respiratory signs and no turbinate damage. Rapid resolution of the outbreak occurs, with little residual damage.

- *Necrotic rhinitis* is uncommon but may accompany atrophic rhinitis. It is caused by *Fusobacterium necrophorum*, with resulting rhinitis and facial swellings. Necrotic lesions are also found in the mouth and on the skin. The disease carries a high mortality.
- *Inherited prognathia*. Individual piglets have excessively long lower jaws.

Treatment

Antibiotic therapy may help reduce the severity of clinical signs shortly after infection, but there is no cure for facial deformity. Tylosin and trimethoprim-sulpha given parenterally at intervals may reduce problems in growing piglets. Tilmicosin can be fed in creep feed continuously for 6 weeks, but it may be difficult to get piglets to eat enough to obtain a therapeutic dose.

Control

Ideally, depopulate and restock with atrophic rhinitis-free pigs.

Strategic medication may be used, especially for pigs showing signs of rhinitis. Improving housing and reducing stocking rates may help. Screening herds can be done with an ELISA test for *B. bronchiseptica*. Tonsillar swabs or swabs from the posterior nares can be cultured. At least 18 negative swabs are required to say that a herd is free.

Vaccination

Vaccination of sows and gilts may help prevent the establishment of disease in their piglets. Primary vaccination is by two intramuscular doses 6 weeks apart. Then vaccinate sows twice yearly 2–6 weeks before each farrowing is due. The vaccine contains *P. multocida* dermonecrotic toxoid and inactivated *B. bronchiseptica*. Great care is required when using the vaccine, which can cause severe reaction in people if accidentally injected.

Pleuropneumonia of pigs

This is an important respiratory disease that can be associated with severe economic loss. It is highly contagious and can be fatal.

Incidence

There is widespread evidence of infection in North America, Asia, and Europe.

The infection is found frequently when there is no evidence of clinical disease. It is responsible for 17.03% of cases in the Veterinary Investigation Diagnosis Analysis list for 1996–2003.

Aetiology

The cause is *Actinobacillus pleuropneumoniae*. There are 12 serotypes of variable virulence. The organism produces a number of pathogenic toxins that lower defences by killing macrophages and neutrophils.

Epidemiology

The disease has relatively low clinical incidence but high mortality. It is usually brought into a herd by a carrier (often recovered) pig. The carrier pig may have infected foci in necrotic areas of the lungs. The severity of the diseases may be exacerbated by the presence of other diseases, such as PRRS or swine influenza. Sudden outbreaks of the disease are occasionally seen when no other pigs have been introduced into the herd. Infection is exacerbated by poor housing and ventilation. It chiefly affects pigs from 2 to 6 months of age and is seen occasionally in finishing pigs approaching slaughter weight. High condemnation rates may be seen at slaughter. Piglets aged <10 days may have colostral protection. Weight gain losses of up to 20% have been reported in some herds.

Clinical signs

The clinical signs have a sudden onset. Typically, one or two sudden deaths occur and other pigs are severely ill, anorexic, and depressed with pneumonia. In acute cases, temperature may be 41°C but may fall as a pig becomes terminally ill. There is dyspnoea with jerky breathing and coughing. Blood-stained foamy mucus may be seen coming from the nose and mouth. Pigs may die within a few hours of showing clinical signs. Some pigs are cyanotic as a result of cardiac failure. Abortion may occur in sows. Other pigs in the group may be less severely affected, showing subacute or chronic signs. Coughing, weight loss, and a poor appetite are seen. Meningitis occurs occasionally.

Diagnosis

Diagnosis is by serological ELISA test and culture of the causal organism from nasal swabs or lung tissue.

Post-mortem – There is acute, fibrinous, haemorrhagic pleuropneumonia. In peracute cases, the lung is dark red and firm to the touch, with blood running from the cut surfaces. Lesions are black-red raised areas, especially in dorsal parts of both lungs including the diaphragmatic lobes (Figs 4.9 and 16.13). Many cases also have fibrinous pleurisy and pericarditis. In chronic cases, a high incidence of pleurisy may be seen at the abattoir. Pathology can occasionally be confined to one lung.

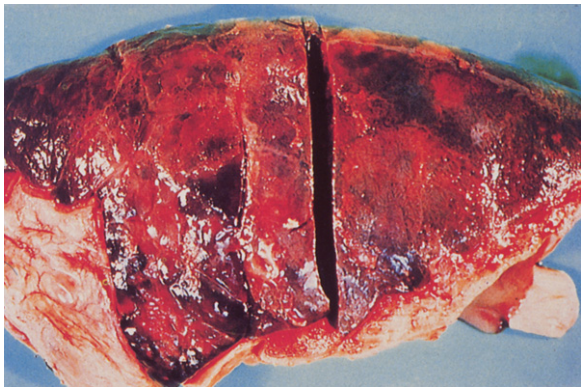

Fig 4.9 – Pleuropneumonia. Note raised red-black lesions in dorsal lung.

Differential diagnosis

- *Enzootic pneumonia*. This is usually a more chronic infection with greater ventral consolidation of the lungs at post-mortem.
- *Pneumonic pasteurellosis*. Culture of the organism and post-mortem examination may show haemorrhagic and necrotizing bronchopneumonia.
- *Swine influenza*. Younger pigs are worst affected, with upper respiratory tract signs and post-mortem findings.
- *Glasser's disease*. Polyserositis including arthritis usually accompanies respiratory signs.
- *Mulberry heart disease*. Sudden death frequently occurs, with abnormal appearance of the heart but no pericarditis or pneumonia.

Treatment

Parenteral antibiotic treatment is given for severely affected pigs; spectinomycin, tilmicosin, oxytetracycline, and ceftiofur can all be used. Sensitivity tests may help decide which antibiotic to use. Early treatment is essential before intractable pathology is established. Mass medication by injection is used for severely affected pigs, or in water or feed for in-contacts if infection is spreading. Isolate clinically affected animals; this may help reduce spread within a group. NSAIDs may aid recovery in acute cases.

Control

Ideally, keep infection out by having a closed herd and purchasing any replacements only from repeatedly serologically negative herds. Try to either eradicate or control the disease.

Eradication – This involves depopulation, thorough cleaning, and repopulation with serologically negative (perhaps hysterectomy-derived) pigs. Piglets may be weaned at 10 days (they are immune to disease until then), medicated (as above), and moved to a separate unit. Rear pigs in isolation and move to a separate finishing unit.

Vaccination – The large number of serotypes of *A. pleuropneumoniae* make effective vaccination difficult, although autogenous vaccines have been used. A commercial vaccine is not currently available in the UK.

Glasser's disease

This is an acute infection causing polyserositis, arthritis, and meningitis. Respiratory signs are seen chiefly in older pigs.

Incidence

It occurs worldwide.

Aetiology

The cause is *Haemophilus parasuis*, which has many serotypes.

Epidemiology

The causal organism(s) is found in the nasal cavity of many pigs. The onset of disease may be associated with exposure to stress such as transport or movement to new pens. The disease may follow other infections such as swine influenza. Although thought to be chiefly a secondary invader, the organism may be a serious primary pathogen in SPF herds. Piglets may have colostral immunity, and Glasser's disease is seen chiefly in pigs from weaning to 4 months of age. The disease can be a complication of other respiratory diseases such as enzootic pneumonia and PRRS.

Clinical signs

These usually have sudden onset, and several pigs are affected at the same time. Affected animals show pyrexia (temperature, 41°C), anorexia, cough, and dyspnoea. Lameness is also seen, with swollen joints and pigs walking with short strides and on tiptoes. Central nervous system

signs may develop in untreated cases, and discoloration of the skin may precede death. *Chronic cases* may follow initial infection: chronic arthritis, intestinal obstruction caused by peritonitis, and heart failure. Deaths and abortion have been reported in sows suddenly exposed to Glasser's disease in SPF herds.

Diagnosis

Diagnosis is based on history and clinical signs, PCR, ELISA, complement fixation tests on serum, and culture of the organism from joints and other tissues (e.g. the pericardium). Culture is difficult and requires special media.

Post-mortem – Polyserositis, polyarthritis, and fibrinous meningitis are found (Fig. 4.10).

Differential diagnosis

- *Swine erysipelas*. Mostly chronic lameness is present, with epiphyseal enlargement rather than swelling of joint capsules. Culture the organism.
- *Mycoplasma hyosynoviae*. This is a milder disease. Culture serology is used.
- *Streptococcal infections*. These often lead to meningitis. Culture to confirm diagnosis.

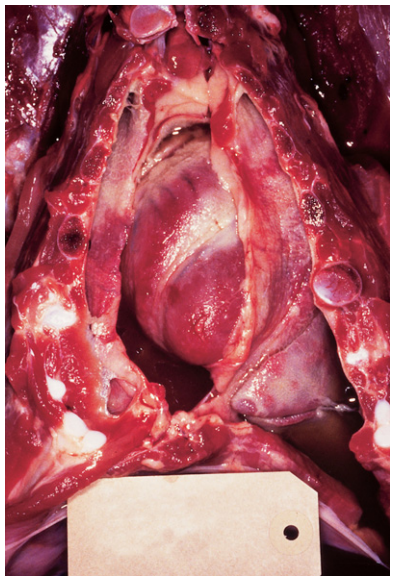

Fig 4.10 – Glasser's disease. Note early signs of pericarditis and pleurisy. (Courtesy of W.D. Strachan.)

Treatment

Injections of penicillin–streptomycin, oxytetracycline, or trimethoprim-sulpha are given to sick pigs. In-contacts are given oral medication in water or food (if eating). Sensitivity tests are advisable if large numbers of pigs are affected or if there is a poor response to treatment. Early treatment is essential to avoid the establishment of pathological problems that might prevent an effective cure.

Control

Avoid stress. Strategic medication may be used at times of high risk, for example after moving if there has been a recent history of disease.

Vaccination – An inactivated vaccine is available for the prevention of Glasser's disease and enzootic pneumonia. The first injection (2 mL i.m.) is given at >1 week of age, the second dose 2–3 weeks later. The complete course is given by <10 weeks of age. Experimental vaccination of both gilts and their piglets may produce better results.

Porcine pasteurellosis

Strains of the organism *Pasteurella multocida* are important as *secondary invaders* in a number of porcine respiratory diseases including enzootic pneumonia, atrophic rhinitis, and pleuropneumonia. The organism can also act as a *primary pathogen*, producing either pneumonic pasteurellosis or *Pasteurella* septicaemia.

Pneumonic pasteurellosis

Incidence

This is a common respiratory disease of pigs. Cases of acute pasteurella pneumonia are seen, sometimes secondary to other infections. Pneumonic pasterellosis has worldwide distribution.

Aetiology

Pasteurella multocida is the cause; a number of different strains are recognized.

Epidemiology

Cases are mostly sporadic in 10- to 20-week-old growing pigs, but several pigs may be affected. The causal organism is found in many pigs, and stress may predispose to acute infection. The organism may also require prior lung damage by another pathogen, such as *Mycoplasma hyopneumoniae* or *A. pleuropneumoniae*, to become established and cause pneumonia.

Clinical signs

These are pyrexia, anorexia, and dyspnoea; one may see open-mouthed breathing, coughing, and some sudden deaths. Some treated cases may become chronic, with reduced growth rate and tendency to relapse.

Diagnosis

Diagnosis is by clinical signs and isolation of the organism.

Post-mortem – Acute necrotizing and fibrinous pneumonia are seen. Grey-pink consolidation is present, especially in anterior lung lobes. There is an absence of other active infectious causes of pneumonia (Fig. 4.11).

Differential diagnosis

Differential diagnoses are other causes of pneumonia.

Treatment

Treatment is by antibiotic therapy: penicillin–streptomycin or oxytetracycline, trimethoprim-sulpha, and ampicillin. A sensitivity test is advisable if several cases are seen or if there is a poor initial response to treatment.

Control

Improve management and try to reduce predisposing stress. Segregated early weaning can be very effective in controlling the disease.

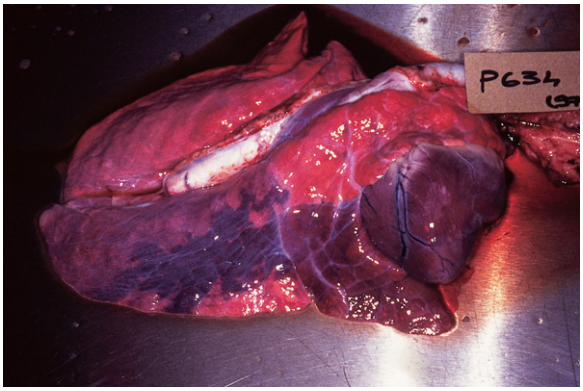

Fig 4.11 – *Pasteurella* pneumonia. Note severe consolidation of lungs. (Courtesy of W.D. Strachan.)

Septicaemic pasteurellosis

This is a cause of sudden death in young and growing pigs. A sudden onset of depression may precede some deaths. Post-mortem reveals a septicaemic carcass with congested lymph nodes. The skin of the abdomen and ears may be cyanotic. The organism is found in impression smears of lymph nodes and the sectioned spleen. Differential diagnoses are other causes of sudden death including clostridial infection.

Swine influenza

Swine influenza is an acute respiratory infection with rapid onset, spread, and resolution.

Incidence

The disease occurs in North America, the Far East, and Europe. The first UK case was seen in 1986.

Aetiology

The cause is influenza A virus, an orthomyxovirus with a number of subtypes. The H1 N1 strain has been seen in the UK and elsewhere. Other human influenza strains have also been found in pigs not showing signs. Aquatic and other birds may also carry the swine influenza virus.

Epidemiology

Direct pig to pig transmission via infected droplets is the most important method of spread, but airborne infection can also occur. There may be a sudden onset of disease, often after a few days' bad weather. Young pigs are most frequently affected, but infection can spread throughout a whole herd. Severity may be worsened by the presence of other respiratory pathogens and also by migrating ascarid larvae. Other respiratory pathogens may gain access to the respiratory system that has been weakened by swine influenza infection. There is no evidence of carrier pigs, and the organism often disappears from the herd after an outbreak of acute infection.

Clinical signs

Clinical signs are incubation over 2–7 days, rapid spread through pigs, temperature of 41.5°C, anorexia, jerky breathing, sneezing, painful (sometimes paroxysmal) cough, and sometimes prostration. The ocular conjunctivae are inflamed, and some ocular discharge is present. Muscular movement is painful and stiff (as in human flu). Severe weight loss occurs despite the short course of the disease. Usually, rapid recovery occurs in 4–6 days.

The disease has high morbidity but low mortality. Convulsions may occur before death. Sows may abort

following infection, and surviving piglets may be born with deformed or infected lungs. Increased numbers of pigs with middle ear disease may be seen. These may result from bacterial infection following viral damage.

Swine influenza infection is occasionally asymptomatic but may facilitate the access of other diseases such as Glasser's disease.

Diagnosis

Diagnosis is by ELISA on serum. PCR is used for virus detection from nasal swabs.

Post-mortem – Severe congestion of the upper respiratory tract is seen. Bronchial or mediastinal lymph nodes are enlarged and hyperaemic. The cervical and mediastinal lymph nodes are also enlarged. Thick exudate may be found in the bronchi. Localized red-purple areas of lung collapse in apical and cardiac lobes, the right lung worse than the left. Emphysema may surround collapsed areas. A necrotizing bronchiolitis may be present.

Differential diagnosis

- *Enzootic pneumonia* is more chronic and insidious.
- *Classical swine fever*. Other body systems are also involved.
- *Atrophic rhinitis*. Characteristic bony changes are seen in the turbinate bones.
- *Inclusion body rhinitis* can look very similar in young pigs, but detection of inclusion bodies in this disease should confirm its presence.

Treatment

Oral or parenteral antibiotics may help control secondary infection with *H. parasuis* and *A. pleuropneumoniae*. Oxytetracycline in water or food may help. Nursing care with warmth and good ventilation may help recovery.

Control

Maintain a closed herd. Vaccination with an oil-adjuvanted vaccine has been used in some countries. Protecting pigs from adverse weather may help reduce the severity of the disease.

Inclusion body rhinitis

Incidence

The disease has worldwide incidence; >90% of UK herds are affected.

Aetiology

The cause is porcine cytomegalovirus (a herpesvirus).

Epidemiology

Transmission is by direct pig–pig contact or aerosol. Virus may be present in the urine. Inclusion body rhinitis mostly affects young pigs but one may get severe herd-wide infection in naive (e.g. minimum disease) herds. The virus can cross the placenta and thus could appear in hysterectomy-derived herds. The virus may predispose to secondary infection with *B. bronchiseptica* and other respiratory pathogens.

Clinical signs

These are mostly in pigs <3 weeks of age. Sneezing may follow play fighting. There may be serous nasal discharge (occasionally bloody) and brown ocular discharge. High morbidity and low mortality occur, especially in chronically affected herds. *In naive herds, signs may be much more severe*. Piglets may have diarrhoea followed by anaemia, rhinitis, oedema, and death. Adult pigs may show respiratory signs. Stillbirth and abortion may occur in sows.

Diagnosis

Diagnosis is by ELISA test for serum antibodies and by the finding of inclusion bodies from nasal swabs and tissues including nasal inflammation and discharge from killed piglets (Fig. 4.12).

Differential diagnosis

- *Atrophic rhinitis*. Check herd history. Bony changes occur.
- *Necrotic rhinitis*. Necrotic tissues and facial swelling are present.
- *Swine influenza* has sudden onset and affects older pigs too.
- *Bordetella* rhinitis is seen occasionally.

Treatment

Antibiotic therapy may help control secondary infection.

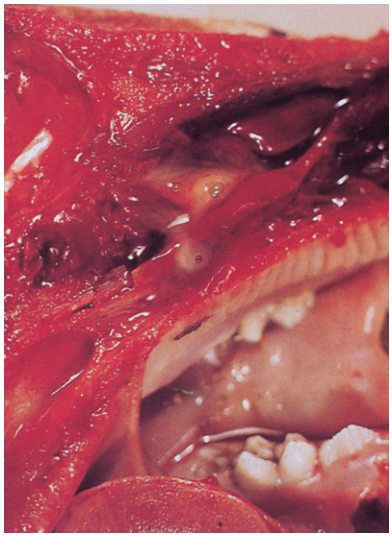

Fig 4.12 – Inclusion body rhinitis. Note severe inflammation of nasal mucosa.

Control

In a herd outbreak, it is beneficial to protect suckling piglets from exposure to infection. Maintain a closed herd.

Porcine reproductive and respiratory syndrome

For a full discussion of this disease, please see Chapter 11 (*Polysystemic diseases*). Only respiratory aspects will be discussed here. The disease is also known as blue-eared pig disease and 'purrs'.

Incidence

The disease occurs in most pig-keeping areas of the world.

Aetiology

The cause is a virus (the PRRS virus) from the Arteriviridae family. There are US and European strains.

Epidemiology

The disease was first identified in the UK in 1991 and is now endemic in many herds. Respiratory disease may persist in an infected herd for years. A wide range of clinical signs have been associated with this disease. Infection may enter a herd with a carrier pig and spread by pig to pig contact. Flies may carry and spread the virus. Aerosol transmission is less likely than is the case with enzootic pneumonia. PRRS virus can be a primary or secondary pathogen.

Clinical signs

Respiratory disease, pyrexia, anorexia, cough, dyspnoea, skin discoloration, and ill thrift are present (Fig. 11.13). About 25% of affected young pigs may die through secondary infection with *Mycoplasma hyopneumoniae*, *H. parasuis*, and other respiratory pathogens. There is lower mortality in older pigs. Respiratory signs may precede abortion in gilts. As disease becomes more endemic, respiratory signs and poor growth can be seen in weaners for several months.

Diagnosis

Diagnosis is based on history.

Post-mortem – Interstitial pneumonia is found. Serology involves ELISA and PCR tests.

Differential diagnosis

Differential diagnoses are other causes of respiratory disease, including enzootic pneumonia, *H. parasuis*, *A. pleuropneumoniae*, and *P. multocida*.

Treatment

Antibiotic therapy and good management may help overcome secondary respiratory infection.

Control

Segregate and rear young pigs off site. Success has been achieved in the USA with testing (ELISA and PCR) and culling from herds.

Vaccination – A freeze-dried vaccine with an adjuvanting diluent is available for use in pigs 6–9 weeks of age in an infected herd. After a single dose of vaccine, protection against the virus should be present for 14 weeks. For a fuller discussion on the control of this disease, please see Chapter 11 (*Polysystemic diseases*).

Porcine respiratory coronavirus

Incidence

Evidence of this infection has been found in many parts of the world.

Aetiology

A *coronavirus* very similar to the transmissible gastroenteritis virus but distinguishable only by monoclonal antibody tests is the cause. Other serological tests do not distinguish the two conditions and may complicate testing for transmissible gastroenteritis.

Clinical signs

Experimentally, the virus has produced signs of bronchopneumonia, pyrexia, coughing, anorexia, and delayed growth. No gastrointestinal signs have been seen. The exact role of the virus in the field is not known, but it may predispose to other respiratory diseases.

Diagnosis

The condition is diagnosed by virus isolation and monoclonal antibody serology.

Post-mortem – Catarrhal bronchopneumonia is present.

Control

An antibody-free herd may be maintained. However, a good level of antibodies will help protect the herd against transmissible gastroenteritis.

Postweaning multisystemic wasting syndrome

Respiratory distress is seen in some affected pigs, caused chiefly by pulmonary oedema. For a full discussion on postweaning multisystemic wasting syndrome, please see Chapter 11 (*Polysystemic diseases*).

Metastrongylosis

Incidence

Metastrongylosis is relatively uncommon, but the return to outdoor methods of pig keeping may increase the incidence.

Aetiology

The causal organisms are *Metastrongylus apri* and *Metastrongylus edentatus*.

Epidemiology

The organism's life cycle requires the earthworm as the intermediate host, and hence the parasite occurs only where pigs have access to earth in an outside environment. Worms are found in the lungs 20–24 days after the pig consumes the thick-walled egg containing the lungworm first-stage larva. The disease mostly affects young pigs – adults appear to be immune.

Clinical signs

Coughing and dyspnoea occur in piglets or growing pigs.

Diagnosis

Diagnosis is based on clinical signs, history, and management. It is confirmed by finding thick-walled eggs containing a larva in the faeces.

Post-mortem – Adult worms 45 mm long are found in or can be squeezed out of the bronchi or bronchioles of the diaphragmatic lobes of the lungs. Areas of pulmonary emphysema may be seen in caudal parts of the diaphragmatic lung lobes (Fig. 4.13).

Treatment

Anthelminthic therapy is used, for example ivermectin, doramectin, fenbendazole or flubendazole.

Control

Avoid access to the intermediate host if practical. Routine dosing of piglets is used.

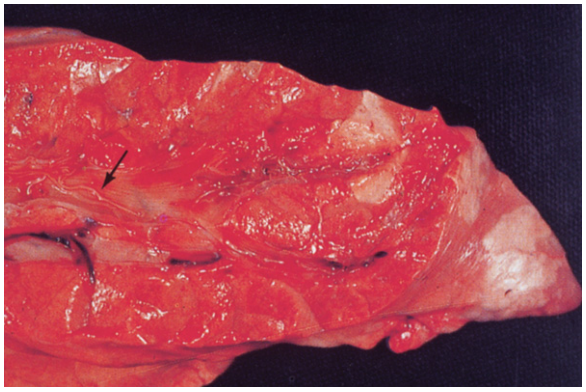

Fig 4.13 – *Metastrongylus apri*. Note small worms in bronchioles.

Diseases of the gastrointestinal system

Introduction

Gastrointestinal disease in pigs is very common and a major cause of economic loss to the pig industry. A large number of infective agents cause disease in the porcine gastrointestinal tract. Some affect a particular age group such as neonatal pigs, while others affect the whole herd. Losses caused by this group of diseases can be direct or indirect. Death and ill thrift can occur, and the farmer has the additional expense of treating sick animals whose welfare can also be severely compromised. Weight loss is a common consequence of these diseases. Slow growth and delays in reaching target weights may be a feature of the recovery phase. A rapid and accurate diagnosis is very important in outbreaks of gastrointestinal tract disease. In many cases, samples will be submitted to a laboratory to confirm a tentative diagnosis. This will ensure that the treatment prescribed is effective and enables a programme of preventive medicine to be planned and introduced.

Aetiology of gastrointestinal diseases

This group of diseases may have infectious or non-infectious causes. Infectious agents are a very important cause of gastrointestinal disease in pigs. Viruses, bacteria, and protozoa are commonly involved in producing clinical signs related to this body system.

Examples are given in each category. Some pathogens, such as *Escherichia coli*, are frequent causes of enteric disease in younger pigs. Other conditions, such as the porcine reproductive and respiratory syndrome virus, may be associated with some outbreaks of diarrhoea, although their main pathological effect is on the respiratory and reproductive systems.

Helminth parasites can also be involved, especially in older pigs. A sudden change in diet in pigs of all ages can be accompanied by diarrhoea. For this reason, care should always be taken when changing from one diet to another. In baby piglets, problems with maternal milk supply may predispose to hypoglycaemia and diarrhoea.

The sudden loss of milk at weaning renders piglets particularly susceptible to enteric disease. Less commonly, diarrhoea may be associated with exposure to toxic substances in the food, for example in aflatoxin poisoning. In some diseases, such as swine dysentery, clinical signs may be limited to the gastrointestinal system. In other conditions, such as classical swine fever, many body systems including the gastrointestinal system are affected by the disease.

Epidemiology of gastrointestinal diseases

Consideration of this is of particular importance in cases of infectious disease. Gastrointestinal disease is especially likely to be a problem if animals are exposed to either pigs carrying disease or a contaminated environment where levels of infection are high. The effects of disease may be influenced by the immunity of the pigs exposed to infection. Infectious agents usually spread rapidly through a litter or a group of piglets. In older pigs, some individual pigs may not show signs of disease. Some infections, such as colibacillosis, rarely affect older pigs, while in outbreaks of transmissible gastroenteritis (TGE) pigs of all ages may show clinical signs. Whenever possible, pigs suffering from infectious gastrointestinal disease should be isolated. This will help to prevent spread to other at-risk animals and reduce further spread of disease.

Clinical signs of gastrointestinal diseases

Pig faeces are normally quite well formed, but diarrhoea is seen in many gastrointestinal diseases. Signs of diarrhoea may be immediately obvious when a group of pigs are observed. Faeces may be passed all over the pen rather than in the dunging area usually located in one corner of a pen. In some diseases, the faeces are foul-smelling; in others, dysentery may be present. Dehydration and loss in condition occur quite rapidly in pigs suffering from diarrhoea. Loose faeces may cause perineal staining in affected animals, which may also show tenesmus. Prostration and death are features of some conditions, such as clostridial infection in unweaned piglets.

Vomiting accompanies enteritis in some conditions, such as TGE. In other diseases, such as subacute swine erysipelas, vomiting may occur in the absence of diarrhoea. The nature of the faeces may give some indication of the location of the pathology within the gastrointestinal tract. In cases in which the cause of diarrhoea is located within the small intestine, the faeces may contain some undigested food and have an acid pH. Diarrhoeic faeces associated with large intestinal disease may have an alkaline pH.

Diagnosis of gastrointestinal diseases

This considers the history of the farm and of the pigs involved. The clinical signs may suggest either a single disease or a group of diseases. Specific diagnosis is greatly aided by the results of a post-mortem examination from dead or euthanized pigs. Culture of causal organisms from faecal samples is another important aid to diagnosis. In general, faecal samples are of greater help diagnostically than rectal swabs. In some but not all conditions, serological tests can provide useful evidence of recent infection.

Treatment of gastrointestinal diseases

This will depend chiefly on the cause. Dehydration and loss of essential electrolytes can be life-threatening in pigs of all ages and should be corrected. Oral electrolyte solutions can be offered to those pigs that are willing to drink. Food should be withdrawn from weaned pigs for a period of 12 h and then carefully reintroduced before being fully restored 24 h later. Water should be freely available. Withdrawal of milk from unweaned piglets is not normally a practical option, but they can be offered additional electrolytes to drink.

There is no specific treatment for viral diseases other than the provision of supportive care and access to electrolytes. Antibiotic therapy, ideally based on sensitivity testing, should be given to pigs with bacterial infections of the gastrointestinal system. A number of piglet dosers are available for the administration of oral antibiotic treatment to young piglets. The whole litter should always be treated, even if only one or two members are showing signs of disease. If there are any signs of systemic infection, for example in some cases of colibacillosis, parenteral antibiotic treatment should also be given.

Antibiotic therapy is often used in cases of viral infections either before a firm diagnosis of cause is available or in an attempt to prevent secondary bacterial infection becoming established. In older pigs, antibiotic treatment can be given in either the water or the food. Water-soluble antibiotics are available and are mentioned below under the treatment of specific diseases. Sick pigs will often drink even if they are unwilling to eat, and they can be medicated while on a very restricted diet.

Control of gastrointestinal diseases

This will depend on whether the disease is associated with infection or is related to problems such as overfeeding or a sudden change of diet. As already mentioned, pigs are susceptible to dietary changes, and these should always be made carefully and slowly. Precautions such as the maintenance of a closed and biosecure unit are important in the control of all porcine disease.

In the case of infectious diseases, the level of environmental contamination and the immunity of the pigs both play important roles. Cleaning, disinfection, and proper resting of pens after each batch of pigs are very important in reducing the amount of environmental infection. Immunity is often boosted by exposure to pathogens. At one time, sows were exposed to biological material from pigs that had died. This is a potentially dangerous procedure and may expose recipients to other diseases than the one it is hoped to prevent. Faecal material can be used with fewer risks. Older sows that have been exposed to a farm's pathogens are often better able to pass on immunity to their piglets than are young gilts. In the cases of some diseases, such as colibacillosis, vaccination of the sow or her piglets will provide a useful boost to immunity in the face of potential infection. On farms where the control methods described are not practical, antibiotic therapy may be given prophylactically before a disease is likely to occur.

Veterinary Investigation Diagnosis Analysis sample submissions: gastrointestinal diseases, 1996–2003

See Table 5.1.

Table 5.1 Veterinary Investigation Diagnosis Analysis sample submissions: gastrointestinal diseases, 1996–2003

Condition	Incidence (%)
Enteric colibacillosis	33.54
No diagnosis	20.85
Swine dysentery	12.01
Rotavirus infection	7.35
Intestinal adenomatosis	4.41
Coccidiosis	3.53
Non-specific colitis	3.03
Torsion of small intestine	2.70
Colitis (*Brachyspira pilosicoli*)	2.24
Clostridium perfringens type C	1.88
Other	1.61
Gastric ulceration	1.42
Intestinal haemorrhage syndrome	1.06
C. perfringens type A	0.98
Helminthiasis	0.96
Cryptosporidiosis	0.52
Hepatosis dietetica	0.47
Torsion of spleen and stomach	0.47
C. perfringens type B	0.38
Porcine epidemic diarrhoea	0.27
Rectal stricture	0.24
Transmissible gastroenteritis	0.08
Vomiting and wasting disease	0.02

Congenital and hereditary conditions affecting the gastrointestinal tract

Atresia ani (imperforate anus)

Incidence

Atresia ani (Fig. 5.1) is quite common; it may occur in 0.3% of all piglets born in the UK.

Aetiology

It is thought to be caused by a recessive gene with incomplete penetrance.

Epidemiology

Atresia ani may involve just one or (more commonly) several piglets in the litter. Both males and females are affected. In the male, total rectal obstruction is present. In the female piglet, faeces are sometimes passed through a rectovaginal fistula.

Clinical signs

Absence of the anus may be spotted within a short time of birth.

The perineum may be distended by the obstructed rectum just beneath the skin. On other occasions, the rectum terminates at the pelvic inlet or within the pelvis. In such cases, dullness, progressive inappetance, and a distended abdomen may be seen. In female piglets with a fistula, small faecal pellets may be seen emerging through the vulval lips. Piglets with a totally imperforate anus may survive 3–10 days after birth.

Treatment

If the rectum is immediately beneath the perineal skin, a cruciate incision can be made through the perineal skin and into the rectum. Local anaesthesia is placed in the skin before the incision is made. Faecal material escapes, and usually the anal opening remains patent. Treatment is not normally necessary for female piglets with a rectovaginal fistula. Please see also Chapter 15 *(Analgesia, anaesthesia, and surgical procedures in the pig)*.

Control

Control is by careful breeding to identify and avoid the use of breeding stock thought to be carrying the recessive gene responsible for the condition.

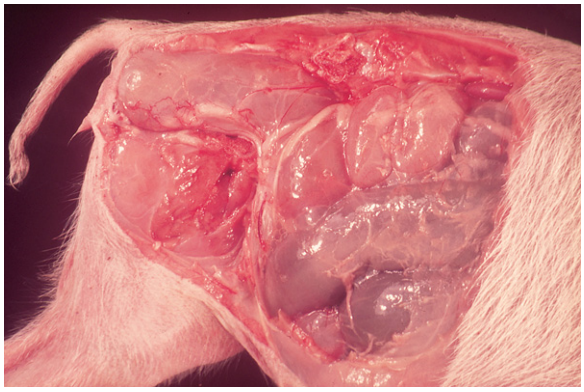

Fig 5.1 – Atresia ani.

Cleft palate

Incidence

Cleft palate is uncommon.

Aetiology

It is thought to be inherited in some cases, possibly through an autosomal recessive gene. It may also follow hemlock poisoning in the sow during pregnancy.

Clinical signs

The piglet is soon seen to be failing to thrive. It is unable to swallow milk from the mother; the milk may run down the nasal passages when the piglet tries to swallow.

Diagnosis

The clinical signs are suggestive of the problem, which can be confirmed by careful examination of the hard palate.

Treatment

Treatment is not attempted. Affected animals are euthanized.

Macroglossus

Macroglossus (Fig. 5.2) is uncommon. The cause is unknown but probably genetic. One or more piglets in a litter are born with very large tongues. Affected animals are unable to close their mouths or swallow. The defect is incompatible with life. Euthanasia and investigation of the genetic background is required.

Short mandible

Short mandible is uncommon but is seen in the humpy-backed pig syndrome, which has been described in pigs of the Large White breed (Fig. 3.5).

Fig 5.2 – Macroglossus.

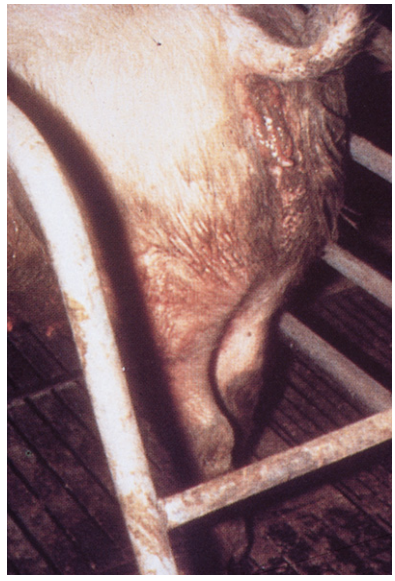

Fig 5.4 – Transmissible gastroenteritis in a sow. This is one of the few causes of diarrhoea in adult pigs. (Courtesy of W.A. Noble.)

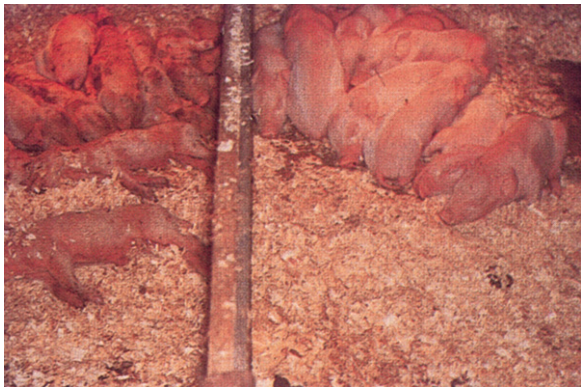

Fig 5.3 – Transmissible gastroenteritis in piglets. (Courtesy of W.A. Noble.)

Infectious diseases of the gastrointestinal tract

Transmissible gastroenteritis

Incidence

The incidence of transmissible gastroenteritis (Figs 5.3 and 5.4) is currently quite low in Europe. It was a major epidemic when first seen in the UK in the late 1950s to early 1960s. The last major outbreak in the UK was in 1981; please see under *Epidemiology* below. The disease is widespread in the USA.

Aetiology

The cause is the TGE virus, a coronavirus.

Epidemiology

A closely related virus, porcine respiratory coronavirus (PRCV), was identified in 1986. Believed to be a mutant of the TGE virus, the PRCV is thought to be widespread among the pig population, providing some immunity against the devastations of TGE.

Transmissible gastroenteritis is a highly contagious disease affecting, in a naive herd, pigs of all ages. It has a high morbidity and mortality. The virus can enter the body orally and by aerosol. It can be carried by birds, vehicles, and carrier pigs between adjacent farms. The disease can become endemic on farms with continuous production of pigs after an earlier epidemic. Piglets receive immunoglobulin (Ig) A-protective immunoglobulins in their mother's milk but after weaning have little immediate immunity and are at risk of infection – this allows the disease to become endemic (see below). The incubation period is 18 h to 3 days. The virus is heat-labile but survives in cold weather, and most outbreaks occur in the winter months.

Clinical signs

- *Epidemic TGE*. This involves a sudden explosive outbreak of disease affecting almost all the pigs on the farm. The youngest pigs are worst affected, and there is almost 100% mortality for piglets <10 days of age. Acute diarrhoea and vomiting occur. Temperature: mostly normal or slightly subnormal. Diarrhoea: watery, foul-smelling, and greenish yellow. Vomitus: yellow. Severe diarrhoea, depression, dehydration, prostration, and death occur in neonatal piglets. Diar-

rhoea in adult pigs lasts a few days. Diarrhoea is occasionally seen in adult sows and boars; TGE is one of the few conditions in which this occurs. Adult pigs receiving wet or dry food should be given extra water and electrolytes to prevent dehydration.

- *Endemic TGE*. Recurrent outbreaks of diarrhoea occur in piglets >6 days of age. It is seen in partially immune large herds in which animals with poor immunity were exposed to the virus and developed diarrhoea. In some such herds, further acute epidemics have occurred after about 9 months.

Diagnosis

Diagnosis is based on virus isolation from the faeces of affected animals, using fluorescent antibody test (FAT) and an enzyme-linked immunosorbent assay (ELISA) antigen test. An ELISA test is also available for detection of specific serum antibodies. A specific monoclonal antibody test is available to differentiate between TGE and PRCV infection.

Post-mortem – Piglets are in poor and dehydrated condition. The stomach is empty, and the gut wall is thin and transparent with green fluid clearly visible within the lumen. Villous atrophy is visible histologically but is also seen in other enteric diseases.

Differential diagnosis

Few conditions produce such high morbidity and mortality as TGE. All ages of pigs on the farm, including adults, are affected.

Specific differential diagnoses include the following.

- *Enteric colibacillosis*. Diarrhoea is mostly responsive to treatment, with no vomiting.
- *Coccidiosis* affects piglets 5–15 days old, with high morbidity and low mortality. Oocysts are present in faeces.
- *Clostridium perfringens* type C. Acute haemorrhagic diarrhoea occurs. Post-mortem needed.
- *Rotavirus enteritis* carries high morbidity and low mortality.
- *Porcine epidemic diarrhoea* (*coronavirus*) is similar to TGE but much less severe.
- *Vomiting and wasting disease* usually has no diarrhoea. Piglets are mainly affected.
- Diarrhoea in adults: classical swine fever, salmonellosis, swine dysentery, and proliferative enteritis.

Confirmation of diagnosis may take some days; in the meantime, attempt treatment as if the case was one of enteric colibacillosis – lack of response is suggestive of TGE or another virus.

Treatment

None is effective. There is little chance of success with neonatal piglets. In older pigs, electrolyte solutions for oral consumption may aid recovery. Some success was experienced with oral administration to neonatal piglets of whole blood or serum from recovered sows in a herd. IgA immunoglobulins may have been responsible. Antiviral drugs have been considered too expensive for routine use.

Control

Good biosecurity, for example by maintaining a closed herd, is essential to prevent TGE access to the farm. In the face of outbreak, planned exposure of sows >14 days from farrowing to piglet or faecal material may give immunity and hence IgA to piglets at birth. The divisional veterinary manager's permission for this is required. Isolate sows <14 days to go to avoid exposure. Vaccination has been attempted in some countries but with dubious effectiveness.

Vomiting and wasting disease

Incidence

A single case was reported by Veterinary Investigation Diagnosis Analysis (VIDA) in 2002.

Aetiology

The cause is a *coronavirus* – a haemagglutinating encephalomyelitis virus (HEV) (not to be confused with the enterovirus cause of viral encephalomyelitis in Teschen's and Talfan's diseases). HEV causes both vomiting and wasting disease and an encephalomyelitis.

Epidemiology

Although clinical disease is quite uncommon, serological surveys show a wide serological response to the virus, suggesting natural challenge. Transmission is by oral and respiratory routes. Several litters are affected in outbreaks. The encephalitic form affects younger pigs. Vomiting and wasting may be seen in slightly older unweaned piglets. One may see both disease forms in the same outbreak. The disease carries high morbidity and mortality. The cause of vomiting is possibly neurological.

Clinical signs

- The *vomiting and wasting form* mostly affects piglets <3 weeks of age. Vomiting with yellow-green vomitus, anorexia, and thirst is seen. Upper respiratory signs are occasionally seen. Attempts to drink and swallow can

be unsuccessful. A transient pyrexia is seen in some animals. Dehydration and emaciation follow. Affected piglets look pale and huddle together with hunched backs. Tooth grinding may be audible. Vomiting may continue for some weeks. Faeces are hard, dry, and scant.

- The *encephalitic form* involves depression followed by hyperaesthesia, muscular tremors, and some vomiting. Terminally, there are convulsions and death.

Diagnosis

This is based on clinical signs, virus isolation, FAT, and serology.

Differential diagnosis

Other enteric diseases are unlikely, except TGE. Check other causes of central nervous system (CNS) lesions in young pigs; please see Chapter 7 (*Diseases of the nervous system*).

Treatment

None is effective.

Control

Avoid entry of infection on to the farm. General principles are as for TGE.

Porcine epidemic diarrhoea

Incidence

Porcine epidemic diarrhoea has a worldwide incidence. The disease was first seen in the UK in 1972. There were seven cases in 2000 and one in 2002 according to the VIDA report.

Aetiology

A coronavirus is responsible.

Epidemiology

There are two distinct clinical forms: type 1 and type 2. They carry high morbidity and low mortality (much less severe than with TGE). The disease may start in finishing pigs on the farm and then spread to breeding stock and neonatal piglets. It can be transmitted by carrier pigs, vehicles, and other contacts. It may be present with other pathogens and may persist on the farm for some time.

Clinical signs

- *Type 1*. Diarrhoea occurs in piglets <5 weeks old only. There is profuse watery diarrhoea. Type 1 carries high morbidity but low mortality. Abdominal pain may be seen in older pigs.
- *Type 2*. Diarrhoea occurs in pigs of all ages. There is often an explosive outbreak affecting most herd members. Some deaths occur in the youngest piglets, much less than in TGE.

Growth rate falls, and pigs take an extra 2 weeks on average to reach finishing weight.

Diagnosis

This is by virus isolation and FAT serology – an ELISA test for antibodies.

Differential diagnosis

Other causes of enteritis in young and older pigs are the differential diagnoses. These include enteric colibacillosis, coccidiosis, *Clostridium perfringens* type C, rotavirus, and vomiting and wasting disease. For discussion, see under *Transmissible gastroenteritis* above.

Treatment

None is effective. Electrolytes in water and keeping the piglets warm and comfortable may help.

Control

Biosecurity is needed. The general principles are as for TGE.

Rotavirus infection

Incidence

Rotavirus infection is common. It is fourth in the VIDA list, accounting for about 7% of gastrointestinal diagnoses in the 1996–2003 survey.

Aetiology

Group A rotaviruses are the most important in pigs.

Epidemiology

Rotavirus can be involved in piglet enteritis at a number of ages, especially in the first 5 weeks of life. It peaks at 3–5 days, 1–3 weeks, and 3–5 weeks (weaning). Subclini-

cal infection is also common, and rotavirus can be involved in enteric diseases at the same time as other organisms, such as *Escherichia coli*. The sow is the source of infection and sheds virus from 5 days before to 2 weeks after farrowing. Sows have IgA in their milk, which is highly beneficial in ensuring some immediate piglet immunity. At weaning, the supply of IgA from the sow stops and piglets become susceptible to infection. This accounts for the last peak in incidence mentioned above.

Rotavirus can be a particular problem in intensive systems where there is a constant throughput of farrowing sows with no resting of accommodation between batches. Infection may be endemic on some farms. The incubation period is short, at 12–24 h.

Clinical signs

Diarrhoea: very fluid to semiformed faeces, sometimes pale yellow. Piglets are often quite bright and may recover spontaneously within a few days. Clinical signs are more severe in cold conditions. Deaths from dehydration may occur.

Diagnosis

This involves virus isolation from piglet faeces, using electron microscopy and FAT. ELISA tests are available for both antigen and antibodies. Positive serological tests may suggest herd exposure rather than a current infection.

Post-mortem – There are fluid-filled small intestines, some milk in the stomach, and evidence of carcass dehydration. Villous atrophy is evident in the small intestine.

Differential diagnosis

The cause must be identified to eliminate a number of other infections, including TGE, porcine epidemic diarrhoea (coronavirus), enteric colibacillosis (may occur with rotavirus), coccidiosis, and *Clostridium perfringens* type C. For full discussion of differential diagnoses for neonatal diarrhoea, see under *Transmissible gastroenteritis* above.

Treatment

There is no specific treatment. Oral electrolytes can be offered if piglets will drink. The removal of milk for 24 h is seldom practical. It is probably best to prescribe oral antibiotics (e.g. neomycin and apramycin) in case *Escherichia coli* infection is also present. One can seldom be sure of the diagnosis until the results of culture and virus tests are available.

Control

Control involves general improvement in hygiene. All in, all out systems are recommended, with thorough cleaning and resting between batches. Ensure good colostrum uptake. No commercial vaccine is available.

Viral enteric diseases in piglets are compared in Table 5.2.

Classical swine fever

For full discussion, see Chapter 11 (*Polysystemic diseases*).

Diarrhoea and occasionally vomiting are seen in some cases of classical swine fever. Other signs, especially CNS signs, are usually present. Affected pigs normally have very high temperatures that are non-responsive to antibiotic therapy. The disease is **notifiable** in the UK and must be reported to the divisional veterinary manager.

Clostridium perfringens type C infection (haemorrhagic enterotoxaemia)

Incidence

Clostridium perfringens type C infection (Fig. 5.5) occurs sporadically and more commonly in outdoor pigs. It is much less common than *Escherichia coli* but usually much more virulent. It is mostly seen in the first 7 days of life, and made up 1.88% of VIDA cases in 1996–2003.

Table 5.2 Comparison of viral enteric diseases in piglets

Disease	Cause	Age affected	Signs	CNS affected?	Mortality	Diagnosis
Transmissible gastroenteritis	Coronavirus	All ages	Vomiting and diarrhoea	No	Very high	FAT, ELISA
Vomiting and wasting disease	Coronavirus	Under 3 weeks	Vomiting and CNS signs	Yes	High	FAT, serology
Porcine epidemic diarrhoea	Coronavirus	All ages	Vomiting and diarrhoea	No	Low	FAT, ELISA
Rotavirus infection	Rotavirus	Under 5 weeks	Diarrhoea	No	Low	FAT, ELISA

CNS, central nervous system; ELISA, enzyme-linked immunosorbent assay; FAT, fluorescent antibody test.

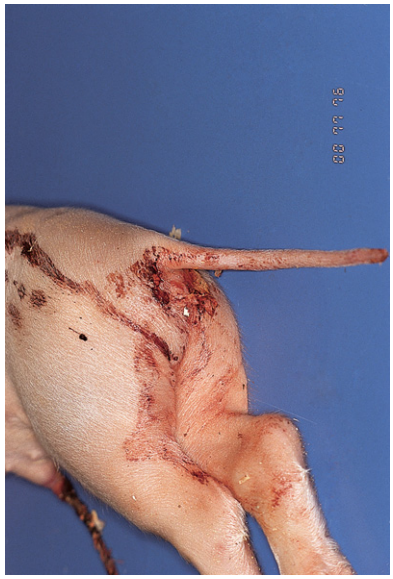

Fig 5.5 – *Clostridium perfringens* type C: dead piglet showing blood staining around the anus.

Aetiology

Clostridium perfringens type C is the cause. Type B is seen occasionally as cause of piglet enteritis. Type A is much less pathogenic (see below).

Epidemiology

Infection is carried on the skin of some sows and also in the faeces of the sow and affected piglets. It may also be present in the environment and can be carried on to the farm on boots or clothing. It is usually seen as an outbreak with a number of litters affected. It occasionally becomes endemic on a unit, but there may be many months between batches of cases. Without preventive measures, the disease may persist for up to 2 months. The organism produces severe damage in the small intestine, causing necrosis and haemorrhage. The production of a β toxin causes further tissue necrosis.

Clinical signs

The first sign is often one or more piglets being found dead. They are often in good condition and may have evidence of bloody faecal staining on the perineum.

Other piglets are dull, depressed, and diarrhoeic; faeces may initially be pale but are often dark red and contain blood and necrotic debris. The anus can be slightly swollen and reddened. Temperature may be briefly elevated but soon becomes subnormal. Piglets can appear normal and then be found dead an hour or so later. Several pigs in a litter are usually affected, and several litters involved. Older piglets may be less severely affected.

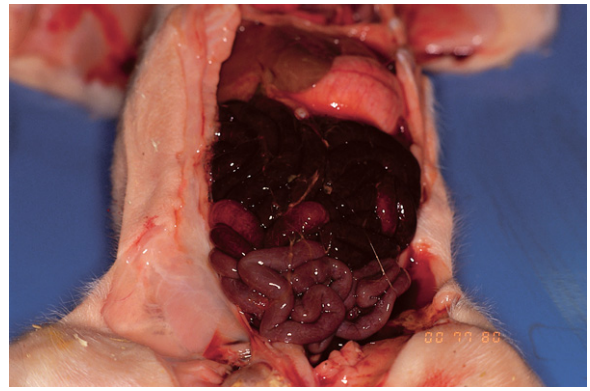

Fig 5.6 – *Clostridium perfringens* type C: at post-mortem, note the dark small intestine and the full stomach.

Diagnosis

There is sudden onset of clinical signs and bloody diarrhoea.

Post-mortem – Piglets are in good condition. The stomach is often full of milk, and the small intestine is dark red and necrotic (Fig. 5.6). One may see localized peritonitis, but the piglet usually dies before this can develop. Fibrin tags may be seen in the lumen of the abnormal bowel and emphysema in sections of bowel. To confirm the diagnosis, direct or cultural evidence of the causal organism is needed. Polymerase chain reaction (PCR) is now available for decoding the various toxins. An ELISA test for the β toxin is available.

Differential diagnosis

Other causes of neonatal diarrhoea are differential diagnoses. *Coccidiosis* may show haemorrhagic diarrhoea, but oocysts are found in faeces.

Treatment

The prognosis for severely ill piglets is poor. Always treat the whole litter and preferably others in the farrowing house. Administer oral ampicillin and parenteral ampicillin for each piglet for 3–5 days. In severe cases, immunity can be boosted by an injection of lamb dysentery antiserum. (Lamb dysentery is caused by *Clostridium perfringens* type B, but the type B toxin provides cross-protection from type C toxin.)

Control

Vaccinate sows to ensure good colostral immunity. A vaccine containing *Clostridium perfringens* types B, C, and D toxoids and a number of *Escherichia coli* antigens is commercially available. The sow is vaccinated 6 weeks before farrowing, with a second dose 2 weeks before farrowing. Sheep clostridia vaccines have been used in emergency. Neonatal piglets can be dosed with oral and parenteral antibiotics if there has been insufficient time for effective use of vaccine.

Clostridium perfringens types A and B

Incidence

These serotypes were less common than type C, but a slight increase in incidence was reported in the VIDA report for 1996–2003.

Aetiology

Clostridium perfringens type A or B is responsible.

Epidemiology

This is a much less severe disease than haemorrhagic enterotoxaemia. It carries high morbidity but low mortality, and mostly affects pigs <3 weeks of age.

Clinical signs

Affected piglets show signs of yellow, watery diarrhoea.

Diagnosis

This is based on clinical signs and identification of the organism by PCR.

Treatment

Administer parenteral and oral antibiotic (e.g. ampicillin) for 3–5 days.

Control

Improve hygiene. Strategic dosing of neonatal piglets may be necessary.

Enteric colibacillosis: enteric diseases caused by Escherichia coli

Incidence

Enteric colibacillosis (Figs 5.7 and 5.8) is very common and the most important cause of enteric disease in pigs. It is responsible for >30% of all gastrointestinal problems in neonatal piglets.

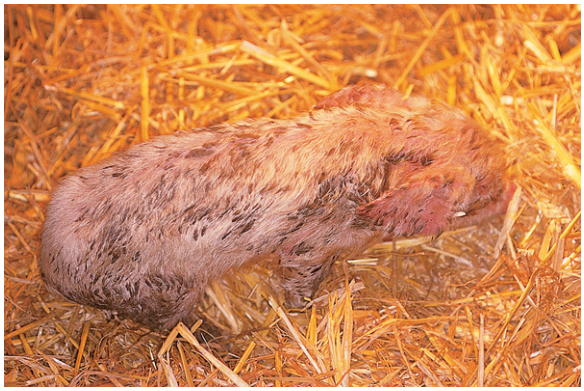

Fig 5.7 – Neonatal piglet showing severe depression caused by *Escherichia coli* enteritis.

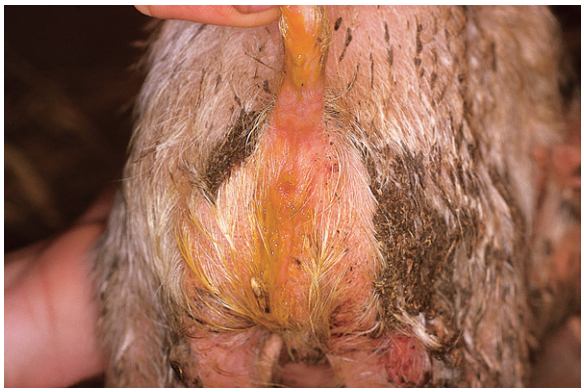

Fig 5.8 – Perineum of neonatal piglet suffering from *Escherichia coli* enteritis.

Aetiology

It is caused by various serotypes of *Escherichia coli*. Important pilus antigens include K88, K99, and K987P. The majority of serotypes do not penetrate the gut wall. They adhere to the gut wall and produce *enterotoxins* that in turn give rise to diarrhoea, dehydration, and sometimes death. Some produce verotoxins.

Members of serogroup 078 are invasive – organisms pass the gut wall, enter the body, and produce *endotoxins*. These in turn contribute to the signs of endotoxic shock, causing severe illness and often death within a few hours.

Disease syndromes: pathogenic *Escherichia coli* produces signs of disease in two ways:

1. *Septicaemic colibacillosis*. The organism invades the piglet's body. It is less common than enterotoxigenic colibacillosis.

2. *Enterotoxigenic colibacillosis*. The organism remains in the bowel. It is the most common kind of colibacillosis.

In both conditions, either a few or many members of the litter may be affected.

Epidemiology

The problem is very widespread, and few units are free from this group of diseases. The litters of older sows with better immunity to *Escherichia coli* experience fewer problems than do the litters of gilts. The organisms are found in the bowel and faeces of sows and other pigs, and also in the uterus and vagina.

Large numbers of organisms can build up in heavily used farrowing crates. Affected litters pass very large numbers of potentially pathogenic organisms, increasing the risk of infection of other litters in nearby accommodation. Infection normally gains access through the mouth but can occur through the respiratory tract or the umbilicus.

Predisposing factors are very important in the epidemiology of enteric colibacillosis. They include the following:

- poor hygiene;
- lack of colostrum;
- adverse temperatures;
- cold, damp, dirty buildings;
- sows coming into the farrowing house unwashed and in a very dirty condition; and
- lack of sow's milk for neonatal piglets.

In older pigs taking solid food, sudden changes of diet may predispose to infection. These include the sudden introduction of creep feed or a major change of diet after weaning. Some strains of *Escherichia coli* are much more pathogenic than others.

Clinical signs

These are discussed under three main age groups:

1. neonatal piglets,
2. unweaned older piglets <3 weeks old, and
3. acute postweaning enteritis.

Neonatal piglets (first 3 days of life)

- *Septicaemic colibacillosis*. Piglets may be found dead or very weak, with low temperature; they feel cold to the touch. They may be slightly cyanotic and occasionally experience terminal convulsions. There is often no diarrhoea at this stage.
- *Enterotoxigenic colibacillosis*. Piglets have yellow-green diarrhoea.

Faecal staining is present on the perineum; contaminated tails hang down with reduced tone. Temperature is subnormal or just normal. Loose faeces are distributed all over the pen. Piglets may attempt to feed from the sow but may be too weak to reach her udder or hold their position in a row of feeding piglets. They become progressively weaker as they become hypothermic, hypoglycaemic, and dehydrated. If untreated, many will die.

Diagnosis

Diagnose by clinical signs and the disease history of the farm. There may be a history of poor colostral uptake, but this may not have been observed.

Post-mortem – Few specific signs are seen in septicaemic cases. In enterotoxigenic colibacillosis cases, the carcass is dehydrated. The stomach is often empty or contains a little dirty fluid. The intestines are thin-walled and fluid-filled (Fig. 5.9). Confirm by isolation and possible typing of the causal organism.

Differential diagnosis

Differential diagnoses are other infectious causes of neonatal enteritis, especially TGE.

- *Transmissible gastroenteritis* is more acute, with 100% mortality, and also affects older pigs.
- *Rotavirus infection* can look very similar to *Escherichia coli* cases, and both conditions may be present at the same time. Virus isolation is needed.
- *Clostridium perfringens* type C has very sudden onset, with haemorrhagic diarrhoea and characteristic post-mortem.
- *Coccidiosis* is usually less acute, with lower morbidity and mortality. Diagnose by identification of oocysts in faeces.
- *Vomiting and wasting disease*. Vomiting is unusual in colibacillosis, diarrhoea unusual in vomiting and wasting disease. Virus isolation is needed.
- *Porcine epidemic diarrhoea*. A range of ages are often affected. It is like a mild TGE infection. Virus isolation is used.

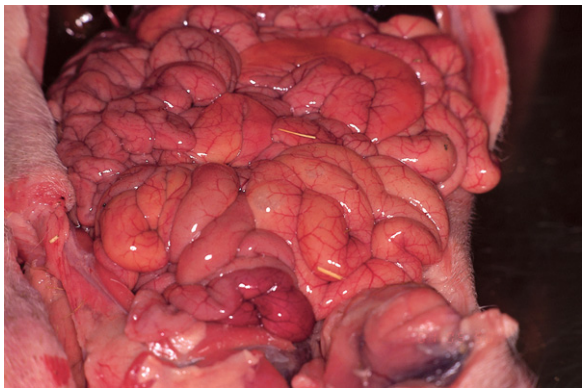

Fig 5.9 – Post-mortem appearance of piglet suffering *Escherichia coli* enteritis. Note the fluid-filled, inflamed small intestine.

Treatment

Oral antibiotics should be given using piglet doser preparations. Parenteral antibiotics are also advisable, as it is not possible to be sure which piglets are septicaemic. Parenteral therapy may help to reduce the risk of septicaemia. Nursing care is important. Piglets must be kept warm and hydrated. It is best not to take them off the sow, but fluid intake may be supplemented by allowing piglets access to electrolyte and glucose solutions in a low dish. One should give antibiotics for 5 days. Beware overdosing with injectable neomycin, which can be toxic. Steroids and non-steroidal anti-inflammatory drugs (NSAIDs) may help reduce mortality in severely ill piglets. For further discussion on practical aspects of diagnosis and treatment, see below under *Dealing with an outbreak of enteritis on the farm*.

Control

- Maximize colostral intake by supervising suckling by neonatal piglets.
- Strict cleanliness in the farrowing accommodation; an all in, all out policy is best. Rest the farrowing house between batches.
- Wash sows before moving them into farrowing quarters.
- Boost piglet immunity by sow vaccination during pregnancy – a number of killed vaccines are available.

Escherichia coli vaccines are available; most contain pilus antigens K88, K99, and K987P. Sows usually receive two injections during pregnancy; the last is administered 2 weeks or so before farrowing. Piglets receive boosted colostral immunity against *Escherichia coli* and hence colostral management must be good. Some vaccines contain *Escherichia coli* antigens and *Clostridium perfringens* toxoids, boosting immunity to both diseases.

Unweaned older piglets <3 weeks old

Epidemiology

This is not always clear; the disease may result from an older litter on the unit being exposed to faeces from younger scouring pigs. The older litter's immunity may be waning, and clinical signs occur. Several litters may be affected, and the problem may persist on the unit for some time. Creep feed may have been changed, increased in quantity, or introduced later than normal. In some cases, no change in management has occurred.

Clinical signs

There is usually a sudden onset of diarrhoea – pale yellow to grey. A variable percentage of the litter are affected. Occasional sudden death occurs, but usually loose foul-smelling faeces are found in the pen and sick piglets spotted. Temperature is usually normal or slightly subnormal. Some animals develop chronic diarrhoea and ill thrift.

Diagnosis

Diagnose by history and clinical signs; diagnosis is confirmed by isolation and possible typing of *Escherichia coli* from faeces or the intestine of a fatality.

Post-mortem – Post-mortem shows severe inflammation of the bowel and sometimes dehydration.

Differential diagnosis

One must eliminate all other possibilities, especially virus infection. See details under *Neonatal piglets (first 3 days of life)* above.

Treatment

Treatment is as for neonatal piglets. It may also help to introduce some clumps of earth into the pen – chewing these appears to speed recovery of the piglets.

Control

Control can be difficult, as the predisposing causes can be difficult to elucidate.

Reducing the environmental contamination is essential. Prophylactic antibiotic treatment may help but is not a long-term solution. Careful introduction of creep and using a creep with a lower protein level may help empirically. Vaccination of piglets early in life has been used, but insufficient immunity is produced to overcome the infectious challenge.

Acute postweaning enteritis

Incidence

This is common. It was especially common after weaning at 8 weeks, and on some units the condition has displaced bowel oedema as a major cause of postweaning illness. The condition occurs after weaning at 3 weeks or at 5 weeks.

Aetiology

Escherichia coli is the cause. A common serotype is K88 antigen Abbottstown, but other serotypes are also seen.

Epidemiology

As with other *Escherichia coli* infections, the level of infection overcomes an inadequate or absent immunity. Mixing of litters and stress (e.g. of fighting) may predispose. Change from creep feed on to an ad lib weaner ration can also be involved. Moving pigs into a dirty, cold pen from the warmth and comfort of their farrowing quarters may also predispose to the problem. Sudden loss of maternal milk in the diet may produce an environment that is conducive to massive multiplication of bacteria within the bowel.

Clinical signs

There are often one or more very sudden deaths, sometimes within hours of weaning. Other pigs have severe enteritis, with temperature normal or low. Flanks are tucked in and the pig has a dehydrated, empty appearance. Eyes are severely sunken. Faeces are watery, greenish grey, and foul-smelling. Tail is straight, cold, and soiled. Vocalization is a thin reedy squeal.

Diagnosis

Diagnosis is based on history of weaning, possible change of food, and clinical signs.

Post-mortem – Examination shows a congested, dehydrated carcass, with stomach often very full of dry meal contents. Small intestine is reddened, thin-walled, and fluid-filled (Fig. 5.10). Confirm by culture of profuse pure growth of (often haemolytic) *Escherichia coli* from faeces or carcass.

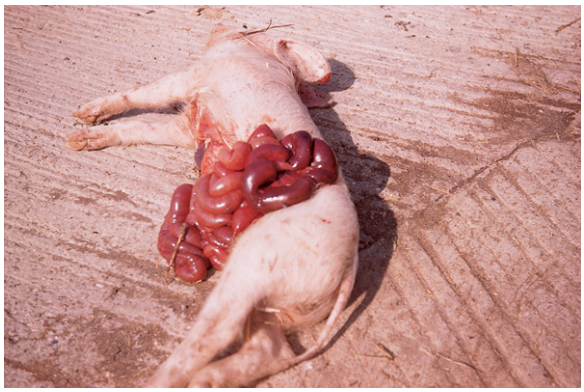

Fig 5.10 – Post-mortem appearance of a growing pig suffering from acute postweaning enteritis. Note severe inflammation of the fluid-filled small intestine.

Differential diagnosis

- Signs could be part of a wider problem (e.g. *TGE*). Virus isolation.
- *Salmonella* infections may cause diarrhoea in this age group; pigs are usually pyrexic. Bacterial culture.
- *Swine dysentery* is usually pyrexic, with dark dysenteric faeces. Bacterial culture.

Treatment

Treatment must be quick and supportive or more pigs will die. Pigs must be carefully handled to avoid more stress. Parenteral antibiotic injection (e.g. neomycin or ampicillin) is used. NSAIDs or steroid injections are used for shocked, dehydrated animals. Offer electrolyte solution and antibiotic (e.g. neomycin) in warmed water. Reduce food drastically and then reintroduce it carefully and slowly.

Control

Reduce predisposing causes, especially sudden dietary changes. Improve hygiene and avoid stress of all kinds. Strategic antibiotic therapy around weaning may help if there is high incidence in weaned litters. See also the general farm approach to enteric problems (below). Some success has been claimed with using zinc oxide in the diet at 2600 ppm for 2 weeks. This is not permitted in some countries.

Bowel oedema (oedema disease)

Incidence

Bowel oedema (Fig. 5.11) is probably less common than it was when pigs were weaned at 8 weeks, but there is some evidence that the incidence may be rising in early weaned groups.

Aetiology

The causes are strains of *Escherichia coli* including 1039 K81, 0139 K12, and 0141 K58. The presence of fimbria type F18 antibody is believed to be significant. In non-immune pigs, the pathogenic organisms adhere to the jejunal mucosa and produce a verotoxin. This in turn causes hypertension and angiopathy of arterioles in the gut wall. Local oedema develops as a result of these changes, which may also affect nerve plexuses, which in turn may be responsible for the CNS signs that are seen in some cases.

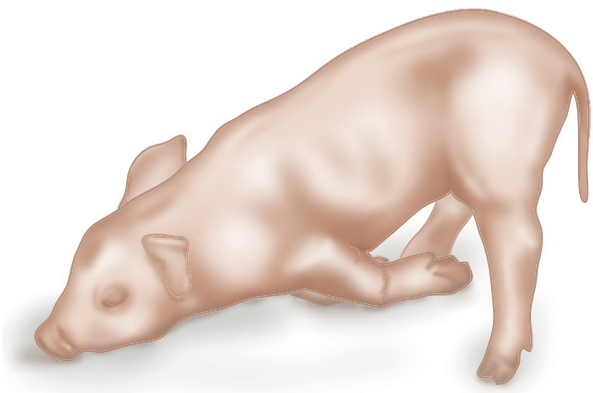

Fig 5.11 – Pig bowel oedema. Note the flexion of the forelimbs and the eye closed by an oedematous upper eyelid. (Redrawn from Jackson PGG, Cockcroft PD. Clinical Examination of Farm Animals. Oxford: Blackwell. © 2002 Blackwell.)

Epidemiology

The disease usually occurs within 10 days of weaning. One or more animals in the litter are affected. Predisposing factors are thought to include excessive feeding, sudden change of diet, too fine particle size in the food, and sudden loss of maternal milk. These factors are thought to aid the establishment of the causal organism in the gastrointestinal tract.

Clinical signs

Affected pigs are dull and less interested in food than the others. Temperature is mostly normal or subnormal. Mild ataxia may be seen, and animals appear to lose control of their forelegs, and they try to move with their shoulders flexed and forelimbs being pushed along the floor. Bilateral eyelid oedema is seen, and pigs may appear blind and deaf. Faeces are mostly normal, but very occasionally diarrhoea is seen. If handled, pigs have a muffled 'bubbly' squeal probably caused by laryngeal oedema. Deterioration is rapid, and within 12 h affected pigs may be in lateral recumbency, breathing with a forced expiration, and unconscious. Death follows in most cases. Very occasionally, pigs recover but may be left with a mild CNS defect such as a head tilt or mild ataxia. In most cases, only one or two pigs in a litter are affected. In rare cases, the majority of the litter show clinical signs.

Diagnosis

Base diagnosis on history of weaning and clinical signs.

Post-mortem – Examine ideally shortly after death; marked oedema of gastric wall as well as laryngeal and mesenteric oedema are present (Fig. 16.15).

Differential diagnosis

Differential diagnoses are other CNS diseases such as *Streptococcus suis* type 2; meningitic signs, including nystagmus, are not seen, and there is no joint involvement with bowel oedema.

Prognosis

Prognosis is very guarded, as affected pigs very rarely recover.

Treatment

Nothing is very effective. Parenteral antibiotics (e.g. neomycin, diuretics, and NSAIDs) may help. For the rest of the litter, try to prevent cases developing. Reduce food intake; possibly change back to original diet, feeding bran with meal. Dosing with magnesium sulphate – oral Epsom salts – is thought to help. Prevention with a verotoxin toxoid vaccine has been reported to be successful, as has the administration of oral oregano powder.

Control

Care at weaning time is needed, and try to avoid predisposing factors.

Salmonellosis

Incidence

Salmonellosis (Figs 5.12 and 5.13) is a widespread and important problem in the pig industry. VIDA reported 91 outbreaks of *Salmonella typhimurium* and 23 cases in

Fig 5.12 – Piglet acute salmonellosis. Note the cyanosis of the ear tips.

which other salmonellae were involved in 2000. The condition is reportable by the Veterinary Laboratories Agency to the environmental health authorities. Some salmonellae are capable of causing zoonotic infections. A recent survey of 2500 slaughter pigs showed the presence of salmonellae as follows: caecal carriage of salmonellae, 23%; surface contamination, 5%. Much lower figures are reported in cattle and sheep. *Salmonella typhimurium* predominated. Surface contamination is very low in well-run abattoirs, and human risk should be quite low – there are few accounts of human infection from this source. In another survey, scalded and singed carcasses were free from salmonella contamination.

Aetiology

Salmonella cholerae-suis (Fig. 5.14) is now rare in the UK and Europe; it was much more common when classical swine fever was endemic in these countries. But *Salmonella cholerae-suis* is a major cause of disease in the pig-keeping areas of the USA.

Fig 5.13 – Piglet acute salmonellosis. Sloughing of the ear tips may occur in some recovered cases.

Salmonella typhimurium is the serotype most frequently found in pigs now. A wide range of other serotypes, including *Salmonella heidelberg* and *Salmonella derby*, have been reported.

Epidemiology

Salmonellosis can have direct and indirect spread. Food, rodents, people, birds, and vehicles can all be involved in the spread of infection. Carrier animals are common, and the organisms may be found in their tonsils or bowel. Piglets can acquire infection from a carrier sow. *Stress* plays a major part in predisposition, as it does in other animals – overcrowding, transport, and poor hygiene. *Risk of human infection* from pigs or contaminated carcasses is important.

Clinical signs

These vary from acute sudden deaths from septicaemia to enteritis and subclinical carriers. One set of signs predominates in most outbreaks, but one may see a spectrum of signs in an outbreak. Signs are chiefly seen in pigs 6–26 weeks of age.

- *Septicaemic form*. A pig or pigs may be found dead; others in the group are dull and pyrexic, with a temperature of 41–42°C. Pigs are anorexic, bury themselves under the straw of their pen, and are reluctant to move. Ear tips, lower limbs, and flanks may show purple discoloration. CNS signs are occasionally seen. Cyanosis was very frequently seen in *Salmonella cholerae-suis*.
- *Acute enteritis*. Pigs are dull and anorexic. Temperatures are 41–42°C. Pigs produce profuse watery, yellow diarrhoea. Some deaths occur. Some pigs may show respiratory and CNS signs. Skin discoloration is present in some pigs, as in the septicaemic form. One may see fresh blood in the faeces.

Fig 5.14 – *Salmonella cholerae-suis*: dead pig showing areas of cyanosis on the skin.

- *Chronic diarrhoea*. Severe weight loss and chronic scour occur. Pigs have intermittent pyrexia. Some develop *rectal stricture* (see below). Pigs may become emaciated and totally anorexic, and death may ensue.

Diagnosis

With high temperature in scouring pigs, always suspect salmonella.

Purple skin discoloration is suspicious but occurs with other septicaemias.

Faecal culture may help confirm the diagnosis, but repeated culture may be necessary as the organism is often secreted intermittently. ELISA tests are available to identify antibodies in sera in individual animals and in the herd.

Post-mortem

- *Septicaemic form*. Multiple haemorrhages, including petechiae, in kidneys are found; the disease can resemble classical swine fever.
- *Enteritis*. There is severe bowel inflammation with infarcts and ulcers. Mesenteric lymph nodes are enlarged.
- *Chronic enteritis*. One may see necrotic enteritis with destruction of much of the mucosal surface. The organism must be cultured to confirm the presence of infection; one may need to do this repeatedly, as the organism may be excreted intermittently.

Differential diagnosis

Differential diagnoses include the following.

- *Classical swine fever*. An important differential. Pigs are also very pyrexic and septicaemic.
- *Acute swine erysipelas*. Pyrexia and anorexia occur, but no diarrhoea is found.
- *Swine dysentery*. This is less pyrexic, with more dysentery.
- *Escherichia coli* infections. These are usually non-pyrexic, and no haemorrhage into bowel or ulceration occur.

Treatment

A range of antibiotics can be used; sensitivity test if time allows. Drugs include trimethoprim-sulpha, apramycin, ampicillin, neomycin, and enrofloxacin.

Parenteral treatment is used for ill and pyrexic animals. Severely ill animals may be helped by steroids or NSAIDs. Fluid therapy is not really practical if large numbers are involved, but oral electrolytes can be provided. Antibiotics are administered in the water for sick and in-contact animals. Food medication is possible. For drugs, see comments on the general approach to enteric diseases in practice (below).

Control

- Identify and (if possible) eliminate the source of infection, including rodents.
- Improve hygiene.
- Maintain a closed herd.
- Use strategic medication for at-risk animals.
- Avoid stress.

Vaccines are not very effective.

The Department for Environment, Food and Rural Affairs (Defra) has published a *Code of Conduct on Salmonella Control in Pigs*, covering prevention, control, and hygiene. All deaths and cases of illness should be investigated to ensure the absence of salmonellosis.

Prevention

Prevention covers unit siting and access, vehicle hygiene, staff training, source of breeding stock, bedding, and water and food. Hygiene involves effective cleaning of the whole unit, including food storage areas.

Swine dysentery

Incidence

Swine dysentery (Fig. 5.15) is very common. It is the second most common enteric disease reported by VIDA, accounting for 12% of cases in 1996–2003. The disease mostly affects pigs aged 8–16 weeks but occasionally adults in a naive herd. Severe economic losses occur through poor food conversion and losses through death of pigs.

Aetiology

The cause is *Brachyspira hyodysenteriae* (formerly *Treponema* and *Serpulina*).

Epidemiology

Direct pig to pig or indirect spread occurs. The organism can live >40 days in damp faeces. It passes through the

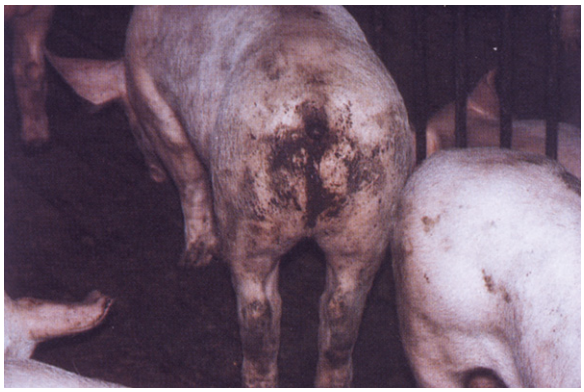

Fig 5.15 – Swine dysentery. Note depression and perineal staining with dark tarry faeces.

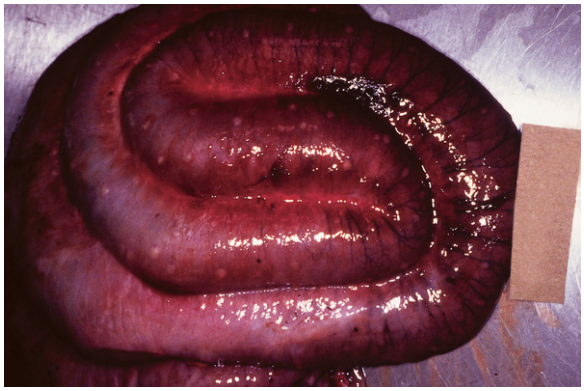

Fig 5.16 – Swine dysentery: post-mortem showing gross appearance of the inflamed and discoloured spiral colon. (Courtesy of W.D. Strachan.)

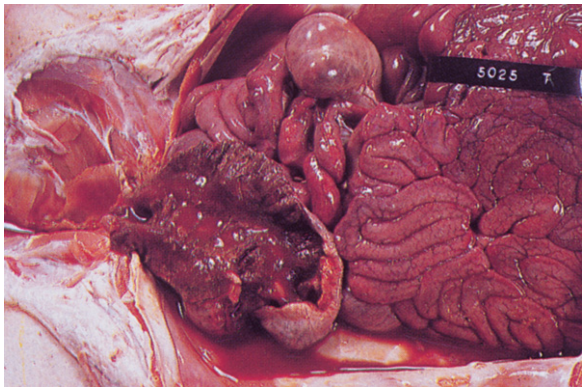

Fig 5.17 – Swine dysentery: opened colon at post-mortem.

stomach unaffected by gastric pH. It can be carried by rodents. Stress – overcrowding, mixing, and moving – predisposes. Morbidity can be 75%. Mortality can reach 25%.

Clinical signs

Mucohaemorrhagic diarrhoea occurs (i.e. dysentery but not always at first). Temperature is 39–40°C. Pigs have poor appetite and rapid weight loss. Abdominal discomfort is experienced, and there are loose faeces all over the pen. Perineal staining is present. Diarrhoea is greyish black, with blood and mucus flecks. Occasional deaths occur early in an outbreak. If untreated, some animals recover spontaneously; others develop chronic, irreversible bowel damage.

Diagnosis

Base diagnosis on herd history, clinical signs, and motile spirochaetes in faeces diluted in buffered saline – but the causal organism must be identified. Slide agglutination test, FAT, and culture are used. ELISA on serum is not very specific.

Post-mortem – Lesions are mainly in the large bowel; the small intestine is mostly unaffected (Figs 5.16, 5.17 and 16.18). The carcass is emaciated and later chronic gut wall damage is seen.

Differential diagnosis

Differential diagnoses include the following.

- *Classical swine fever.* Pigs have higher temperatures. CNS and respiratory signs are present. Dysentery is rare.
- *Porcine colonic spirochaetosis* is milder, and *B. hyodysenteriae* is not present.
- *Escherichia coli* infections are non-pyrexic. The small intestine is involved. Culture.
- *Salmonellosis*. Pigs usually have higher temperature. Yellow diarrhoea is seen.
- *Yersinia* infection. Low-grade diarrhoea and ill thrift occur.
- *Intestinal haemorrhagic syndrome.*
- *Gastric or oesophageal ulceration*. See below.
- *Trichuris* infestation with heavy worm burden.

Treatment

Start as soon as possible. It is advisable to treat the whole group. Parenteral injection of, for example, tiamulin, tylosin (some resistance), and lincomycin is used.

Water medication is given for in-contacts (e.g. tiamulin 45–60 mg/L). In-contacts can receive tiamulin in food (20 ppm for 4 weeks). Valnemulin can be used in the food.

Control

- Depopulation and repopulation is necessary if the outbreak is very severe.
- Improve hygiene.
- Use strategic medication.
- Feed highly digestible food – this reduces the incidence of swine dysentery.

No vaccine is available. Eradication by depopulation and repopulation is the preferred method of control if practically and economically possible.

Spirochaetal colitis and diarrhoea

This condition (Fig. 5.18) is an enteric disease of pigs caused by spirochaetes other than *B. hyodysenteriae*.

Incidence

The disease has worldwide incidence and is possibly increasing. It is mostly seen in young weaned pigs but occasionally in adults.

Aetiology

Spirochaetes, including *B. pilosicoli*, are responsible.

Epidemiology

This is as for swine dysentery, including the rodent carrier state.

Clinical signs

Diarrhoea and very occasionally dysentery occur. Temperature is 39–40°C. Poor weight gain and some chronic cases of diarrhoea are seen. Many pigs recover without treatment.

Diagnosis

Diagnosis is by clinical signs and isolation of a spirochaete other than *B. hyodysenteriae* from the faeces. This is normally a much milder disease than swine dysentery.

Treatment and control

These are as for swine dysentery.

Campylobacter coli

Incidence

This is quite common, but the presence of the causal organism is not always associated with disease.

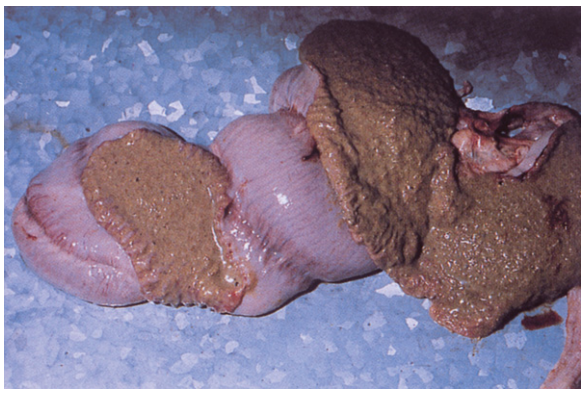

Fig 5.18 – Spirochaetal dysentery. Opened bowel at post mortem.

Aetiology

Campylobacter coli and also other group members – *Campylobacter hyointestinalis* and *Campylobacter jejuni* – are responsible.

Epidemiology

The infection is probably transmitted from pig to pig by the faeco-oral route. The organism is present in many pigs but may cause disease in non-immune animals. It may contribute to clinical signs of other bowel infections. It has been suggested that the organism produces a cytotoxin within the bowel that damages the gut wall. Zoonotic infection may occur.

Clinical signs

Diarrhoea is seen, which is pale and watery with some mucus present. Temperature is 40°C.

Occasional blood flecks are in the faeces. Weight loss and production delay occur, but deaths are uncommon.

Diagnosis

Base diagnosis on clinical signs, isolation of organism, and absence of other infectious agents.

Post-mortem – There is mild inflammation of the small intestine and proximal large intestine. Mesenteric lymph nodes are enlarged, and one may see some thickening of terminal ileum wall.

Differential diagnosis

Differential diagnoses are other causes of diarrhoea including dietary change.

Treatment

Parenteral treatment is rarely necessary. Oral (water) medication with neomycin, enrofloxacin, or oxytetracycline is used.

Porcine proliferative enteritis complex (porcine intestinal adenomatosis)

Synonyms include proliferative enteropathy, necrotic enteritis, proliferative haemorrhagic enteropathy, and regional ileitis. Chronic infection is possible. See Figure 5.19.

Incidence

The disease is widespread. It accounted for 15% of cases in the 2002 VIDA reports. It is seen in postweaning pigs aged 4–6 weeks (mostly as proliferative enteropathy) and

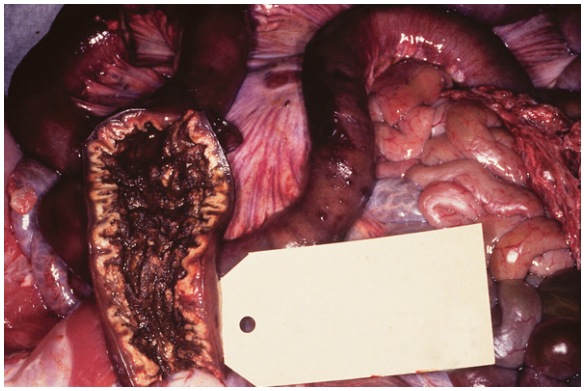

Fig 5.19 – Proliferative enteritis: post-mortem appearance of the terminal ileum.

also in young adults (mostly as proliferative haemorrhagic enteropathy). The incidence has increased since the banning of growth promoters in food. Thirty per cent of UK farms may be affected.

Aetiology

The cause is *Lawsonia intracellularis*.

Epidemiology

Spread is via the faeco-oral route, and infection can be brought in by carrier pigs. The organism is found in other species, including wild boar. The organism is usually intracellular and causes replacement of some gut wall cells by adenomatous tissue. The severity of lesions is dependent on the dose of organisms received.

Clinical signs

Two main syndromes are seen.

1. *Proliferative enteropathy* usually occurs in pigs 4–6 weeks after weaning. Affected pigs are dull, with poor appetite and poor food conversion. Temperature is mostly normal. One may see some pale diarrhoea but not in all animals. Chronically affected animals with thickened terminal ileum ('hosepipe gut') fail to thrive and may die of inanition.
2. *Proliferative haemorrhagic enteropathy* usually occurs in young adults. Sudden death and sometimes passage of haemorrhagic faeces may occur. Animals may be pale due to heavy blood loss into the bowel. Temperature is normal.

Diagnosis

This is based on clinical signs, the age group involved, and identification of organism in faeces or gut wall. PCR test for the organism's DNA in faeces aids diagnosis.

Post-mortem – In proliferative enteropathy, there is inflammation of the bowel with possible terminal thickening and some necrosis of the wall of the terminal ileum. Necrotic enteritis and terminal ileitis are possible.

With the haemorrhagic form in older pigs, large amounts of blood are present in the bowel lumen. This may be in the form of long clots, which have been described as 'ropes'. Some fibrin tags on the bowel epithelium are seen.

Differential diagnosis for intestinal blood loss

Differential diagnoses include the following.

- *Swine dysentery*. This usually develops more slowly.
- *Bleeding from gastric or oesophageal ulcers*. Post-mortem examination confirms this.
- *Intestinal haemorrhagic syndrome* is usually associated with a whey diet (see below).
- *Clostridium perfringens* type C usually occurs in younger pigs and post-mortem signs are seen.

Treatment

For clinically affected animals, use parenteral oxytetracycline or tiamulin. Vitamins help blood replacement.

For in-contact pigs, use oxytetracycline or tiamulin in water. Alternatively, tylosin or lincomycin-spectinomycin can be given in food or water (see data sheets).

Control

Control is difficult, as the full epidemiology is unknown.

- Use strategic antibiotic therapy.
- Improve hygiene.
- Whole building destocking and disinfection help.
- Maintain a closed herd.

It is hoped that an effective vaccine may be developed.

Yersinia infection

Incidence

Yersinia infection is probably uncommon, the main concern being the organism's involvement in human disease, with farm animals as the possible source.

Aetiology

Yersinia enterocolitica and *Y. pseudotuberculosis* have both been involved in porcine and human disease. Pathogenic and non-pathogenic strains are recognized.

Epidemiology

The infection is spread by direct pig to pig transmission. Carrier pigs with the organism in their tonsils may be involved in spread, as are rodents. Carrier pigs may be a source of infection to human abattoir workers. *Yersinia* infection may predispose to rectal stricture in infected pigs (see below).

Clinical signs

Diarrhoea with dark watery faeces is seen, with occasional flecks of blood. Chronic ill thrift, poor food conversion, and wasting may be seen. Temperature is normal or up to 40°C. Some cases recover spontaneously.

Diagnosis

Diagnose by identification of the organism by culture and PCR.

Differential diagnosis

Other causes of enteritis, especially swine dysentery, are differential diagnoses.

Treatment

Give parenteral and oral oxytetracycline or trimethoprim-sulpha.

Control

Control involves attention to hygiene for animals and staff. Rodent control may help.

Colitis (non-specific colitis)

This is a non-specific syndrome causing diarrhoea, in which the definitive aetiology may not be clear. It may predispose to other enteric infections.

Incidence

At least 5% of UK herds may have the problem. The syndrome mostly affects pigs of 8–10 weeks of age. It is more common on farms where conditions are dirty and disinfection is rarely performed.

Aetiology

The cause is unknown but may involve the following.

- *Unsuitable nutrition*. This may include excesses of ingredients such as soya and peas, also too finely ground food. The pelleting process may destroy trace elements. There is evidence for food involvement in the aetiology – changing diet may sometimes reduce the incidence of the problem. Pantothenic acid deficiency has also been suggested, but CNS signs of this are not seen.
- *Chronic parasitism*, including oesophagostomiasis (see below).
- *Chronic infection*, including with *Rotavirus* and chronic TGE.

Clinical signs

Loose faeces with a frothy, grey appearance are seen. Mucus and small quantities of blood may be found in the faeces. Signs are mostly mild, but overall food conversion and growth rates may be reduced.

Diagnosis

Careful investigation of feeding and management is needed. Search for known pathogens in faeces and possibly at post-mortem.

Treatment

A different diet can be tried. Oral antibiotic treatment (e.g. with neomycin or oxytetracycline) may help. Depopulation, repopulation, and thorough cleaning and disinfection may be necessary on problem units.

Necrotic stomatitis

Necrotic stomatitis (Fig. 5.20) is occasionally seen in piglets while on sow. Infection with *Fusobacter necroph-*

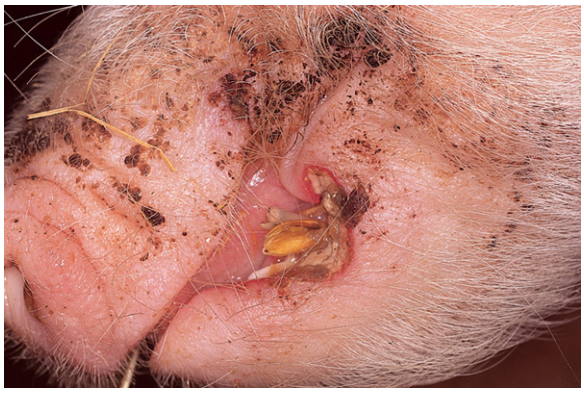

Fig 5.20 – Necrotic stomatitis: after tooth clipping.

orum may gain access to the soft tissues of the mouth through injury, including bites and careless use of tooth clippers. Foul-smelling lesions are seen on cheeks, lips, and tongue. Mild pyrexia is seen. Piglets may have difficulty in feeding. Treatment is by antibiotic injection – oxytetracycline is usually effective. Gentle debridement of accessible necrotic tissue speeds recovery.

Protozoal diseases

Coccidiosis

Incidence

Nine cases of coccidiosis (Fig. 5.21) were reported in the VIDA figures for 2002 (20 in 2000). 3.53% of gastrointestinal submissions in VIDA report for 1996–2003.

Aetiology

A number of species are found in pigs. *Isospora suis* is especially important in suckling piglets. *Eimeria suis* and *Eimeria porci* are among those species found in older pigs.

Epidemiology

Isospora suis is mainly acquired from the faeces of other pigs but rarely from the faeces of the mother. *Eimeria* spp. are, however, contracted from carrier sows. Infestation can build up both on outdoor units and also in damp indoor accommodation, especially that with solid floors.

Clinical signs

Ill thrift, weight loss, and diarrhoea are seen. Piglets from 5 to 21 days are most commonly affected. Diarrhoea may be pale and pasty or yellow and fluid in consistency. Some vomiting of curd-like material is seen. Growth rate is depressed, and piglets look thin and stunted. Young adults may show signs of ill thrift caused by coccidiosis. Mortality can reach 20% in untreated cases.

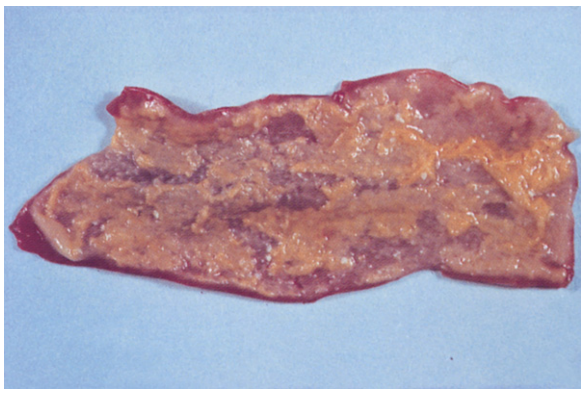

Fig 5.21 – Coccidiosis.

Diagnosis

This is based on clinical signs, possibly poor response to antibiotic therapy, and oocysts in faeces (which should be identified to determine their species).

Post-mortem – In some cases, a fibronecrotic membrane lines parts of the jejunum and ileum. Haemorrhage from damaged gut wall is rarely seen at post-mortem. Development stages of coccidia are found in histological sections of the intestinal mucosa.

Differential diagnosis

Other causes of diarrhoea and ill thrift are differential diagnoses.

Treatment

Some coccidiostatics are not licensed for use in pigs. Trimethoprim-sulpha can be given parenterally or orally. Sulphamethoxypyridazine can also be given parenterally. For other treatments, including toltrazuril used under cascade regulations, please see drug data sheets.

Prevention

Improve hygiene and reduce exposure to oocysts. Prophylactic dosing of piglets may be necessary. Routine treatment of sows does not have great benefit in preventing piglet infection. Steam cleaning or fumigation with ammonia may be used to reduce oocyst contamination in the environment.

Cryptosporidia

Incidence

0.52% of gastrointestinal submissions in VIDA report for 1996–2003.

Aetiology

The cause is *Cryptosporidium parvum*.

Epidemiology

The organism is present in normal pigs and is mostly subclinical. Cryptosporidiosis may be concurrent with other enteric diseases and can be a zoonotic.

Clinical signs

When present, these include diarrhoea and ill thrift.

Diagnosis

Diagnosis is based on oocysts in faeces. Other causes of diarrhoea are eliminated. Histological examination of the intestinal mucosa is done.

Treatment

Antiprotozoal drugs such as halofuginone might be used in an emergency. Supportive treatment, including electrolytes, is given when necessary.

Control

This is as for coccidiosis (see above).

Dealing with an outbreak of enteritis on the farm

1. *The farmer will normally phone to say there are some pigs ill*. The pigs will probably be 'scouring' (suffering from diarrhoea); some may have died already, and others may be very sick indeed. An urgent visit may be requested. The farmer may have attempted treatment already by dosing with an oral antibiotic.
2. *Additional information*. Question the farmer on the phone about the extent of the problem and the age of the piglets or older pigs involved. How long has the problem been going on? Have any pigs died? When did they die? Are they available for post-mortem? Have other litters been affected? Has any treatment been given? What was the result of treatment? Are the pigs from sow or gilt litters? As dietary change is an important cause of diarrhoea in most ages of pig, details of the diet and any recent changes should be ascertained.
3. *First aid*. Advise keeping pigs warm and putting a disinfectant foot dip outside their pen if this is not already present. Do *not* advise dosing with antibiotic until the pigs have been seen. The clinician will wish to take faecal and other samples and does not wish to compromise sensitivity tests. Depending on the age of the pigs, one can suggest offering electrolyte solution. With older pigs suffering from acute postweaning enteritis, immediate removal of food may be advisable.
4. *Equipment required*. Post-mortem facilities: knife, gloves, and sample bottles for faeces and possibly serum and blood. Bacterial swabs. Viral transport media. Antibiotic supplies to treat the approximate numbers of pigs involved. A piglet doser format is needed for neonatal pigs, water-soluble supplies for older sick pigs. Possibly in-feed medication for in-contact pigs.
5. *Arrival at the unit*. Take the usual disease precautions (dipping boots etc. and showering in minimal disease units). Further assessment of case history is done.
6. *General appraisal of the problem*. How extensive is the problem? Are there large numbers of dead pigs? General appraisal of building: ventilation, air quality, and odour. Faeces on the floor? Colour of faeces? Faecal staining on perineum of pigs? Feeding system: ad lib or twice daily, wet or dry? If suckling litters affected, is the sow well? Is she eating? Is she letting her piglets feed?
7. *Initial clinical examination of sick pigs*. A full clinical examination is performed. In many enteric conditions, body temperature may have been briefly elevated but often falls rapidly as pigs become shocked and possibly moribund. Is there any sign of dehydration? Do the affected pigs look very ill? Is any immediate treatment necessary or can the post-mortem of any dead pigs commence?
8. *Post-mortem examination of dead animals*. In some diseases, there may be clear signs of one condition (e.g. *Clostridium perfringens* type C); usually, the piglets' stomachs are full of milk and there is acute necrotic or haemorrhagic inflammation of the intestine. The piglets are in good condition. In cases of enteric colibacillosis, the stomachs are empty and the piglets are in poor condition. A post-mortem of several pigs should be completed and appropriate samples taken. The local divisional veterinary manager of Defra must be consulted if there is any suspicion of notifiable disease.
9. *Samples for laboratory diagnosis*. In many cases, the cause of illness may not be immediately obvious. If a viral infection is suspected, one or more representative living or recently dead piglets should be sent for investigation. *It is very important to remember the cost of the investigations involved and to discuss them in advance with the farmer*. If there is a major problem involving many pigs, there is little option but to investigate fully.

   In emergency, a quick culture can be done in the practice laboratory. If *Escherichia coli* is suspected, one can smear a rectal swab on to blood agar and McConkey agar; in this way, one can confirm the presence of *Escherichia coli* and get a 'crude' sensitivity. For full bacteriology including sensitivity and serotyping, a regional laboratory is needed but the results will take time – if large numbers of piglets are dying, quick results are very helpful. FATs etc. can be done by the Veterinary Laboratories Agency very quickly. As in other cases, *one must have really appropriate samples to get really reliable results*.

   Faecal samples are greatly superior to rectal swabs for bacterial and viral isolation.

10. *Immediate treatment of surviving pigs*. The clinician should encourage the owner to carry this out carefully. Neonatal piglets should be given oral antibiotic. Piglet dosers with pump mechanisms to assist oral treatment are available commercially. If possible, septicaemic, piglets should also receive parenteral antibiotic. Antibiotics include apramycin, amoxycillin, enrofloxacin, neomycin, and spectinomycin. Treat for 3–5 days – this may have to change if there is a poor response or if sensitivity tests suggest a better antibiotic.

    Beware of overdosing, especially with neomycin. This drug is effective for treatment of *Escherichia coli* infections but even a small overdose can kill – piglets should be weighed before dosing. For severely ill piglets, the clinician may advise euthanasia if there is no hope of recovery. The use of steroids (for example betamethasone) or NSAIDs may aid treatment of septicaemic piglets. Reduce food intake until diarrhoea improves. Electrolytes should be added to easily accessible drinking water or be given by injection.

    For treatment of older pigs, treatment in the water is much preferred to in-feed medication for sick pigs. Ill pigs will usually drink even if they will not eat. Food restriction is often advisable in enteric disease, and controlled water medication should ensure that pigs able to drink receive a correct dose. Water proportioners are available in some units, so mains water can be medicated en route to the pigs. If not available, cut off the mains supply and provide medicated water in a trough. Once the daily dose is taken, the farmer can go back to the mains supply. A number of water-soluble antibiotic preparations are commercially available. These include apramycin, lincomycin, oxytetracycline, neomycin, and tylosin. Many *Escherichia coli* serotypes are resistant to oxytetracycline, and it is not the first choice for enteric disease. Oxytetracycline can, however, be very effective in the treatment of enzootic and other pig pneumonias.

    When using any product, the clinician must check the data sheets for the mode of administration and meat withdrawal restrictions for older pigs. In most cases, treatment for 5 days is advised.
11. *Check other pigs on the farm* if the farmer is concerned or if there is a risk that a disease such as classical swine fever or *Salmonella* infection might be affecting whole herd.
12. *Follow-up visit*. This is the next day if there are any very sick pigs; the farmer may cancel if the pigs are fully recovered and losses have ceased. If further pigs have died, they may be submitted for post-mortem to ensure that no other diseases or complications are present.
13. *Discussion with the farmer after laboratory results have been received*. These may confirm the tentative diagnosis. Plan immediate and longer-term prophylaxis. This might suggest prophylactic treatment of the next few litters to be born. Longer-term sow vaccination might be advised. Hygiene and feeding regimens are discussed and amended if necessary. In postweaning enteritis, zinc oxide in the diet may help but is not permitted in some countries. The withdrawal of growth promoters from pig food may increase the incidence of enteric disease.

Other gastrointestinal diseases

Thin sow syndrome

Incidence

Thin sow syndrome (Figs 5.22 and 5.23) is quite common and possibly increasing. Sporadic cases may be seen in the herd, or an unacceptably high proportion of sows are affected. There are serious economic consequences, as affected sows will not breed and cannot be marketed.

Fig 5.22 – Thin sow syndrome (lateral view).

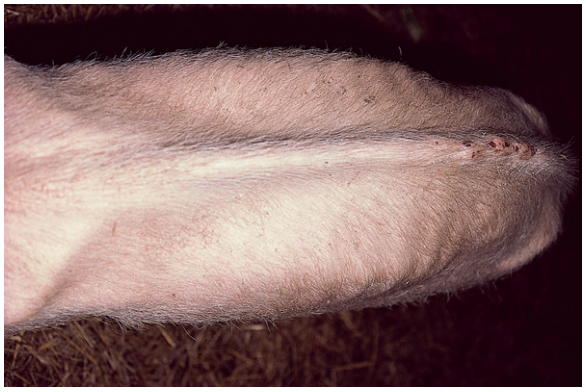

Fig 5.23 – Thin sow syndrome (dorsal view).

Aetiology

Excessive weight loss during pregnancy and especially lactation is seen.

The cause may be multifactorial. It may involve poor husbandry, especially inadequate feeding and parasitism. Important parasites include *Hyostrongylus rubidus* and *Oesophagostomum* spp. Intestinal malabsorption, possibly as a result of parasite or other bowel damage, has been suggested. Cold and draughty housing conditions, inadequate diet and water, and poor management including feeding arrangements can also contribute. In most cases, more than one cause is involved.

Epidemiology

Sows normally lose up to 25% of their body weight during lactation. This weight is restored in most sows during early pregnancy. Thin sow syndrome animals fail to regain their target weight. They seem unable to consume enough food, even if it is available, to restore their proper body weight. Their lack of body fat may mean that they fall into an irreversible catatonic state.

Clinical signs

Affected sows are very thin; ribs and backbone are clearly visible, and condition score is less than 1 (out of 5). Temperature is often slightly low. Apart from severe weight loss, the animal may appear clinically normal. The mucosae may be slightly pale. Appetite is not good, but water consumption may be increased. Return to oestrus after weaning is delayed, and eventual conception rates and litter size may be small. Some fail to conceive.

Diagnosis

A clinical examination should be carried out on each animal to ensure that no other problems, such as pyelonephritis, endocarditis, or other clinical conditions, are present. A faecal worm egg count is performed.

Treatment

Treatment is unsatisfactory. Keeping sows in warm housing with ad lib food including milk may help some cases. Culling with the purchase of replacement stock is preferable.

Control

Prevention is much better than cure. Management must be reviewed and regular weighing instituted to ensure that loss in weight is detected and if possible prevented. Individual feeding and effective parasite control are essential. Regular weighing of sows and condition score assessment may help detect early cases and allow remedial attention.

Intestinal haemorrhagic syndrome

This is also known as 'bloody gut' and 'whey bloat'.

The exact aetiology of the condition is obscure; torsion of the bowel is often present but not in every case. Whey feeding predisposes, but the condition also occurs where it is not fed.

Incidence

Intestinal haemorrhagic syndrome is quite common on some (usually whey-feeding) units. It affects pigs with >35 kg of body weight.

Aetiology

This is unknown; gastric distension with gas production due to fermentation is possible, but the syndrome is also reported after rolling animals during anaesthesia. The condition could be caused by a possible allergy to milk protein. Clostridial toxaemia is a possible cause. Mechanical volvulus may produce the condition in some cases.

Clinical signs

Pigs are mostly found dead with a bloated carcass. The syndrome is occasionally recognized when the pigs are alive; acute abdominal distension and colic are seen. Some pigs recover when no torsion is present. Skin pallor associated with internal haemorrhage is seen.

Diagnosis

History, feeding, and clinical signs are the basis of diagnosis.

Post-mortem – There is gross distension of bowel. Volvulus involving small or large intestine is present in some cases.

Differential diagnosis

Rectal stricture is possible (see below).

Treatment

If torsion is present, it is probably too late to treat successfully – euthanasia is needed. Success has been claimed with use of spasmolytic and antibiotic (e.g. oxytetracycline) therapy.

Control

This is difficult, as the incidence is so sporadic. Altering the diet may not be practical.

Rectal stricture

Incidence

Rectal stricture is quite common; it may affect up to 10% of growing pigs on the unit.

Aetiology

The cause is not fully known. Previous rectal prolapse may predispose, but not all stricture cases have a history of this. Previous long-term medication with tylosin may also predispose. Previous *Salmonella*, *Yersinia*, or *Candida* infection is blamed in some cases, but they are not found in all cases. The *anatomical explanation* is that the site of the stricture is usually approximately 3 cm anterior to the anus. It is at the junction of endoderm and ectoderm, and the terminal point of the blood supplies to each posterior haemorrhoideal artery and the perineal blood vessels are involved. If the area is damaged, repair is mostly by fibrosis, leading to stricture, rather than by first-intention healing.

Epidemiology

The unknown cause makes this difficult, but a high incidence of this unpleasant problem occurs on some farms.

Clinical signs

Abdominal distension (Fig. 5.24) is seen. Pigs are anorexic and dull; they spend much time lying down in sternal recumbency. Despite abdominal enlargement, pigs are in poor condition. Occasionally, they pass large amounts of flatulent liquid faeces – the abdominal distension is relieved but returns in a few days. An attempt to insert a thermometer fully into the rectum is met with an obstruction.

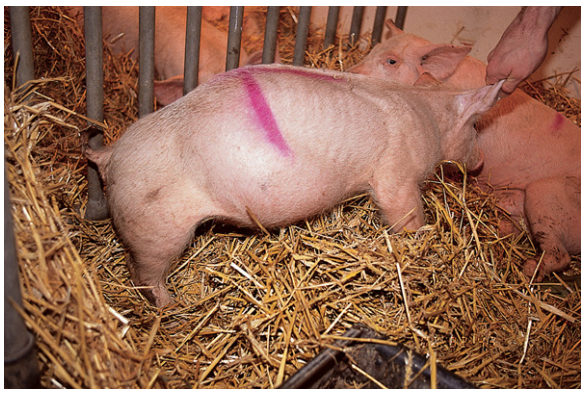

Fig 5.24 – Rectal stricture. Note the gross abdominal distension.

Diagnosis

Diagnose based on clinical signs and farm history. A careful digital rectal examination with a lubricated gloved finger is diagnostically useful if a pig with a distended abdomen is seen (Fig. 5.25). An inability to insert the finger provides immediate confirmation of the problem. At post-mortem, dissection of the rectum reveals the area of stricture (Fig. 5.26). Megacolon caused by chronic rectal obstruction may also be seen (Fig. 5.27)

Differential diagnosis

The sudden onset of distension can occur in intestinal haemorrhagic syndrome and also in ascites following cirrhosis of the liver. Peritonitis may cause abdominal distension.

Treatment

Surgical colostomy has been described but is usually not attempted. Euthanasia of affected animals is the usual course of action.

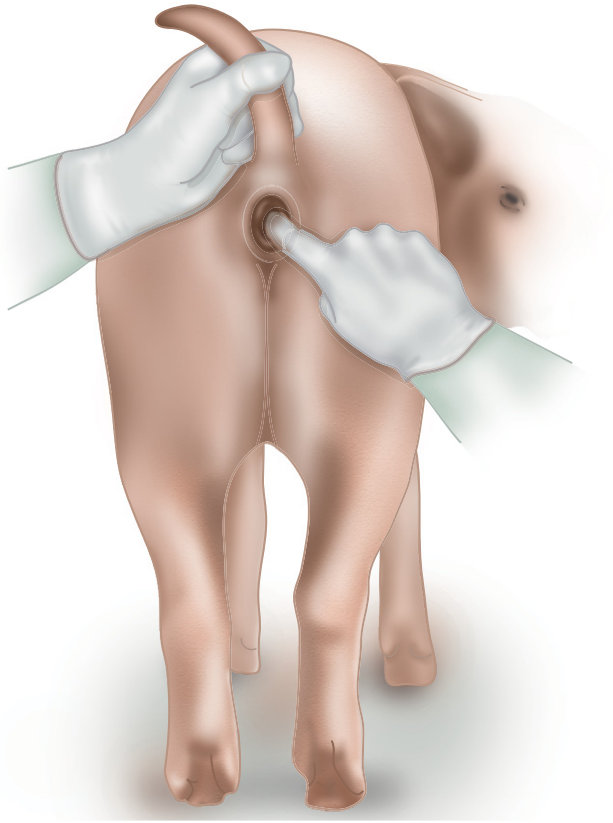

Fig 5.25 – Rectal stricture: digital rectal examination. (Redrawn from Jackson PGG, Cockcroft PD. Clinical Examination of Farm Animals. Oxford: Blackwell. © 2002 Blackwell.)

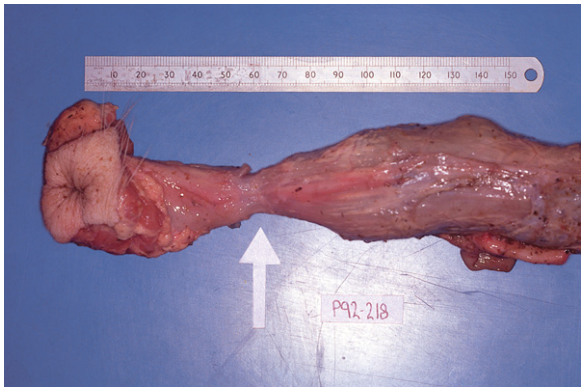

Fig 5.26 – Rectal stricture: post-mortem showing severe reduction of the rectal diameter.

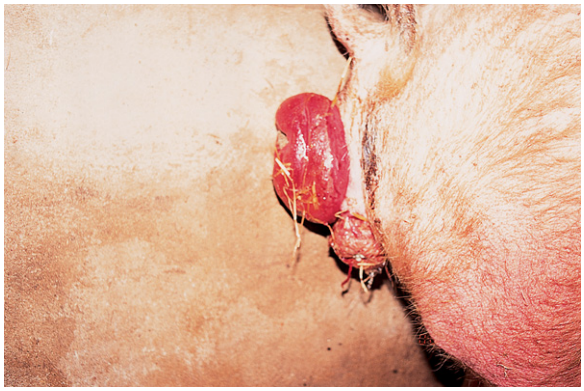

Fig 5.28 – Rectal prolapse (lateral view).

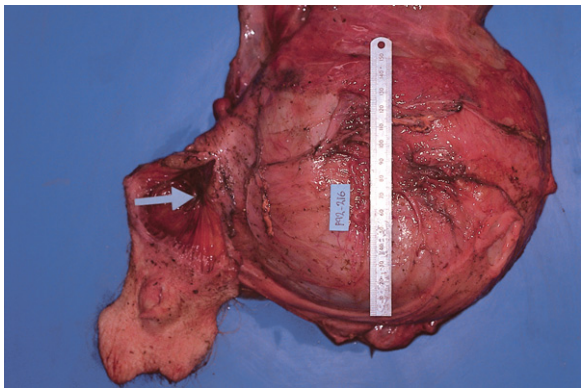

Fig 5.27 – Rectal stricture: megacolon.

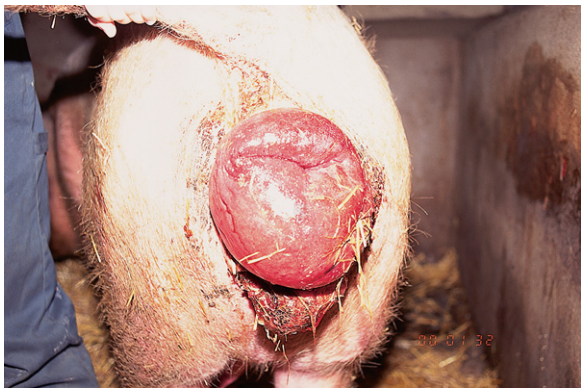

Fig 5.29 – Rectal prolapse (caudal view).

Control

Control is difficult – check the salmonellosis status of the herd. If rectal prolapses are common, investigate their cause.

Rectal prolapse

Incidence

This is quite common. It is mostly sporadic but can be a herd problem. Males are more commonly affected than gilts. It is seen chiefly in pigs aged 12–20 weeks.

Aetiology

The condition is caused by excessive slackening of rectal, perineal, or pelvic tissues.

Chronic diarrhoea and/or cough predispose, also prolonged oral medication with tylosin. Occasionally, rectal prolapse is the result of zearalenone poisoning from oestrogen-producing *Fusarium* spp.

Clinical signs

Intermittent or constant exposure of rectal mucosa is seen. In sows, a vaginal prolapse may also be present (Figs 5.28, 5.29, and 15.3). The prolapse is often damaged by contact with the environment or by biting by other pigs. Severe haemorrhage may be seen from the damaged prolapse.

Diagnosis

Base the diagnosis on clinical signs; the clinician should ensure that the rectum is involved and that the vagina is not prolapsed.

Treatment

Please see Chapter 15 (*Analgesia, anaesthesia, and surgical procedures in the pig*).

Control

Control can be difficult, but if the cause can be identified adjust management to avoid it.

Gastric ulceration (pars oesophagea)

Gastric ulceration is a common finding at post-mortem but is rarely associated with clinical signs. When clinical signs appear, the problem can be life-threatening.

Incidence

Clinical cases are uncommon. The non-glandular oesophageal part of the stomach is mostly involved. Fundic ulcers are discussed below.

Aetiology

Predisposing causes include:

- stress of any kind, including disease and overcrowding;
- parasitism, especially with *H. rubidus*;
- diet, especially too fine particle size, and also high unsaturated fat levels;
- lack of roughage, for example no straw in the diet; and
- porcine multisystemic wasting syndrome.

Epidemiology

The initial lesion may be hyperkeratosis in the pars oesophagea. This leads to mucosal ulceration, exposure of blood vessels, and haemorrhage. The condition is mostly seen in pigs approaching finishing weight.

Clinical signs

There may be sudden death in a pig looking in good condition. One may see skin pallor if the ulcer is bleeding, and pain is detected in the xiphoid region. Melaena is in the faeces, and pigs may vomit blood. Mucous membranes are pale. Temperature is normal or subnormal.

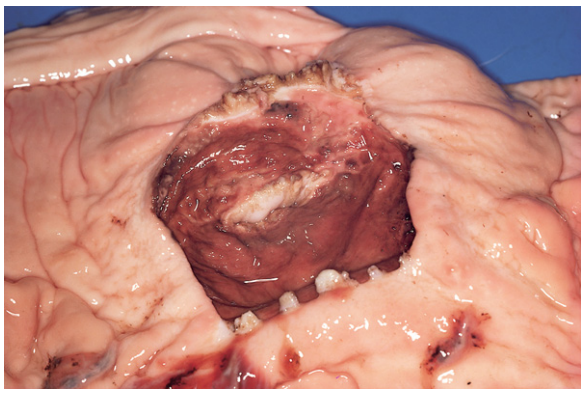

Fig 5.30 – Gastric ulcer.

Diagnosis

This is based on clinical signs.

Post-mortem – An extensive ulcerated area and stomach full of blood are seen.

Differential diagnosis

Differential diagnoses include other causes of blood in the gastrointestinal tract, including swine dysentery – usually many pigs are involved and they have initial high temperature. Damage to the gut wall is also seen in some cases of salmonellosis. The source of the blood is intestinal rather than gastric.

Treatment

Blood transfusion is an option in valuable and anaemic pigs. The clinician should encourage haemopoiesis, for example with multivitamin injections. Drugs such as ramitidin are not licensed for use in pigs.

Control

Avoid stress.

Gastric ulceration (fundic)

Gastric ulcers (Fig. 5.30) may be a relatively uncommon incidental finding in diseases including classical swine fever and *Salmonella cholerae-suis* infection. High infestations with *H. rubidus* may also cause ulcerative damage in this part of the stomach. The role of *Helicobacter*, found in pigs and also in people with gastric ulcers, is not known.

Gastric torsion in sows

This condition is uncommon but may be an occasional cause of sudden death. It may be more common in sows fed only once daily and in animals becoming excited after feeding. Cases are possibly particularly common in sows kept individually in sties and that stand on their hind legs looking over their stable door for approaching food.

Intestinal torsion

This occurs in some cases of the intestinal haemorrhagic syndrome but can also occur as an acute abdominal catastrophe. It is usually discovered at post-mortem after a sudden or unexpected death in a pig. The condition is rarely diagnosed in life – surgical intervention might be possible but economic considerations may preclude.

Intussusception

This is reported rarely, usually as a cause of sudden or unexpected death. It may be caused by heavy burden of adult ascarid worms in the small intestine. In the living pig, diagnosis by palpation is difficult but an ultrasonographic scan of the abdomen would aid diagnosis. Surgical correction by resection of the bowel would be possible in diagnosed cases.

Stone swallowing

This is uncommon but is usually seen in animals living outside. Individual animals form the habit of picking up stones in their mouth, playing with them, and then swallowing. In some animals, very large quantities are eaten and accumulate, often in the large intestine. Appetite may fall, and animals become lethargic. The clinician can occasionally palpate or ballot stones through the abdominal wall, when they can be felt grating together. Treatment with oral liquid paraffin can be attempted. Intestinal or gastric rupture (Fig. 12.14) may occur with fatal results. The prognosis is not good, and it may be advisable to send pigs for slaughter if in reasonable condition and with the agreement of the official veterinary surgeon. If in poor condition, euthanasia may be necessary. Sand swallowing leading to intestinal impaction in outdoor pigs has also been reported. Treatment with liquid paraffin may be effective in mild cases.

Abdominal hernias

Inguinal and umbilical hernias are common in pigs. Please see Chapter 15 (*Analgesia, anaesthesia, and surgical procedures in the pig*).

Aflatoxicosis

This is uncommon but can occur if the ration contains groundnuts or maize that has been badly stored.

Aetiology

Aspergillus flavus or *Aspergillus parasiticus*, which produce aflatoxin B1, are the cause.

Clinical signs of poisoning occur if aflatoxin B1 is fed at levels of >0.4 mg/kg of body weight.

Epidemiology

The toxin is hepatotoxic, causing fatty degeneration followed by necrosis of the liver. Exposure to the toxin for >6 weeks may be necessary before signs appear.

Clinical signs

Affected pigs are dull and depressed. They become ataxic and jaundiced. Terminally, convulsions and death may occur. Bleeding disorders may occur in animals in which liver damage is present.

Diagnosis

Diagnosis is based on clinical signs and post-mortem. The carcass is jaundiced. Blood fails to clot. The liver is friable and pale or bright orange. Levels of toxin can be determined by an ELISA test.

Treatment

There is none. Remove any contaminated diet.

Hepatosis dietetica

Please see Chapter 8 (*Diseases of the cardiovascular, haemopoietic, and lymphatic systems*) for discussion of other conditions associated with vitamin E and selenium deficiency.

This condition is one of a number of diseases caused by a deficiency of selenium and vitamin E. Hepatosis dietetica affects growing pigs aged 3–4 months. Pigs may show signs of vomiting, depression, ataxia, and jaundice. Some sudden deaths may occur. Post-mortem reveals an enlarged, pale, haemorrhagic liver. Treatment is by injection of vitamin E and selenium and checking dietary supplementation.

Helminth parasites of pigs

Although seldom diagnosed as a major cause of clinical disease, intestinal worms can cause depression of growth and food conversion efficiency. They should not be forgotten in planning disease control programmes for pig farms. The increase in outdoor pig keeping has increased the risk of helminthiasis.

Hyostrongylosis

Incidence

Hyostrongylosis is quite common. Surveys have shown that up to 30% of sows may be infested in some herds. Younger pigs are less commonly involved but can be at risk.

Aetiology

Hyostrongylus rubidus, the red stomach worm of pigs, is responsible.

Epidemiology

Adult worms are red, 1 cm in length. The adult worms live on the gastric mucosa, where they inflict damage resulting in a protein-losing enteropathy. Heavy infestation may cause necrosis and ulceration of the gastric mucosa. The life cycle is direct. Some larvae enter a resting

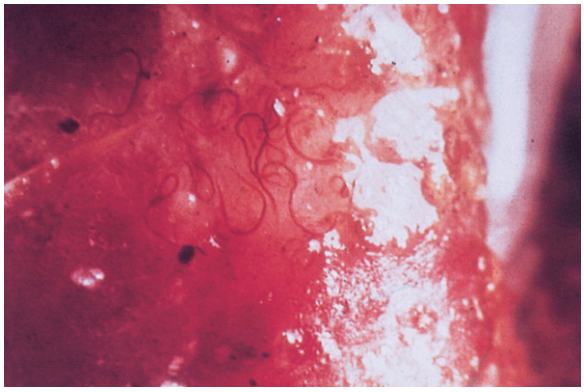

Fig 5.31 – *Hyostrongylus rubidus*: worms on gastric mucosa.

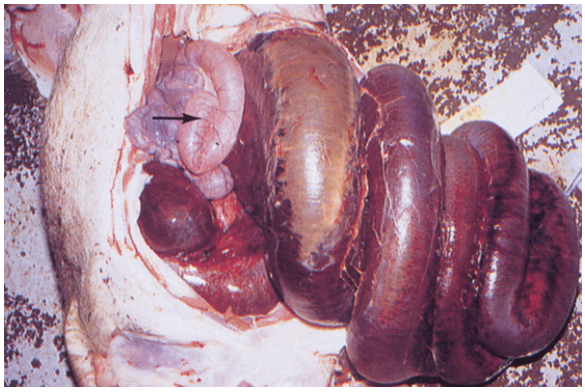

Fig 5.32 – Intestinal torsion caused by *Oesophagostomum*.

stage in the gastric mucosa of sows and are activated by the hormone changes of pregnancy and lactation. Worm egg counts rise in infested sows, increasing the risk to their piglets.

Clinical signs

These are loss in condition, anorexia, and poor breeding performance. The clinician may see melaena and occasionally vomiting. *Hyostrongylus* is sometimes involved in the thin sow syndrome (see above).

Diagnosis

Diagnosis is based on clinical signs, worm egg count, and elevated plasma pepsinogen. Post-mortem may reveal severe damage to the gastric mucosa (Fig. 5.31).

Differential diagnosis

Other causes of weight loss, including poor feeding, are possible.

Treatment and control

An anthelmintic programme is used (see below).

Oesophagostomiasis

Incidence

Oesophagostomiasis (Fig. 5.32) is quite common and easily overlooked. Surveys have shown that 80% of UK sows and 30% of younger pigs may be infested. The incidence is likely to increase with the greater use of outdoor pig units.

Aetiology

Oesophagostomum dentatum and *O. quadrispinulatum*, the pig nodular worms, are the cause.

Epidemiology

Adult worms are 1.5 cm long and live in the colon and caecum. The life cycle is direct. Third-stage larvae are ingested and invade the caecal and colonic mucosa, causing damage. Eggs are passed in the faeces, and infective larvae develop in 1–2 weeks in indoor units but more slowly at pasture. Larvae are thought not to survive long in indoor units. Despite this, infestations in indoor units can reach high enough levels to cause clinical disease.

Clinical signs

Diarrhoea occurs in heavy infestations. Weight loss, poor food conversion, and low milk yield are present in sows. Oesophagostomiasis may be involved in the thin sow syndrome (see above).

Diagnosis

Base the diagnosis on clinical signs, farm worming policy, worm egg count, and nodular lesions seen in the walls of colon and caecum at post-mortem.

Treatment and control

See notes on anthelmintics (below).

Ascariasis

Incidence

Ascariasis is widespread in the pig population. Clinical signs of infestation are uncommon, but the consequences of infestation – especially milk spot liver – can result in condemnation of the liver.

Aetiology

Ascaris suum, the common pig roundworm, is responsible.

Epidemiology

Adult worms are white and very large – up to 40 cm. The life cycle is direct, but larvae migrate through the liver and lungs. Ascarid eggs are extremely resistant; they can survive away from pigs for up to 7 years, and hence the dangers of infestation from fields where no pigs have lived for some years. Cleaning of accommodation is difficult to ensure that all eggs are removed.

Clinical signs

Reduced growth rate and poor food conversion are seen. Occasional sudden deaths are caused by obstruction of the small intestine, intussusception, and blockage of the bile duct (causing jaundice). Larval migration is thought to cause milk spots in the liver, which may lead to liver condemnation. The clinician may see coughing caused by migrating larvae 1 week after infestation; it is difficult to differentiate from other causes.

Diagnosis

Base on clinical signs, worm egg count, and liver abattoir reports. An ELISA test for serum antibody is now available.

Post-mortem – In cases of sudden death, the small intestine may be obstructed by large numbers of ascarids (Figs 5.33 and 16.17).

Treatment and control

See the notes on anthelmintics below.

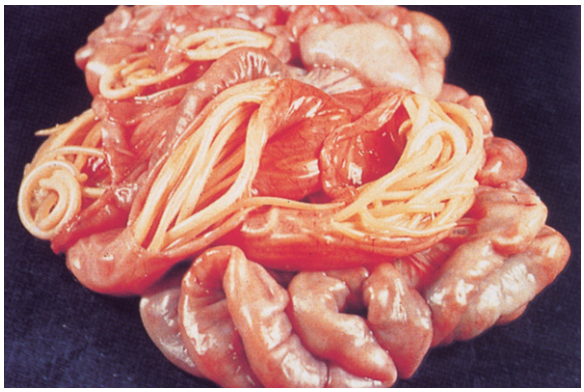

Fig 5.33 – Ascariasis: intestinal blockage by adult worms.

Trichuris infestation

This is found frequently (in 25% of UK pigs) as the small whipworm *Trichuris suis* in the caecum. It rarely causes clinical disease in the UK, but cases of ulcerative typhlitis have been reported overseas. Clinical signs resembling those of swine dysentery have been reported in some cases of *Trichuris* infestation. The worms are controlled by most anthelmintic programmes (see below).

Lungworm disease

For full discussion, see Chapter 4 (*Diseases of the respiratory system*).

Infestation with *Metastrongylus apri* or *M. edentatus*, the pig lungworm, occurs in the UK. The incidence is likely to increase with the development of more outdoor units with access to the intermediate host, the earthworm. For treatment, see the notes on anthelmintics below.

Hepatosis cysticercosis

This is mostly seen in sheep and cattle, less commonly in pigs.

The cause is an intermediate stage of *Taenia hydatigena*. The problem may be recognized only at slaughter.

Liver fluke disease

Infestation with *Fasciola hepatica* is seldom diagnosed in the UK. Increasing numbers of outdoor units may increase the risk of exposure to the intermediate host, the snail *Lymnaea truncatula*, which lives in muddy damp environments. No flukicide drugs are currently licensed for use in pigs in the UK.

Worm control programmes

Helminth parasite control in some countries is neglected. Planning a programme should be logical and use the following steps.

- Is there a worm problem on the farm – is a survey necessary?
- Is worm control necessary? Yes, if there is any evidence of parasitism.
- In outdoor units, pasture rotation is very important but may not be possible.
- Which worms are present, and which mode of therapy would be effective and possible with farm labour?
- In-feed wormers are easily given every 6 months. With injectable wormers, can you be sure that each pig is treated, and do they have a wide range of activity, including against mange? A number of pig wormers are commercially available. The species of worms they are active against and the method and frequency of treatment should be checked before use.
- Antihelmintics include doramectin, fenbendazole, flubendazole and ivermectin.

6

Diseases of the skin

Introduction

The skin is the largest organ of the pig's body. The skin over the dorsal part of the body is thicker and less mobile than that covering the ventral parts. The skin over the shoulders and chest is especially thick and is sometimes called the shield. Hair is relatively sparse but is more plentiful dorsally than ventrally. About 60% of the pig's hair is in the form of bristles, other hair being of a downy nature. The sparse coat of the pig facilitates observation of the skin and of any lesions present.

The skin can be affected by a large number of diseases. Some of the pathological changes seen are directly related to the presence of skin disease. Other changes in the skin, for example in its colour, may provide an external indication of generalized diseases such as septicaemia, circulatory failure, or jaundice. Colour changes are seen only in pigs with unpigmented skin. Porcine skin is easily damaged by fighting and injury.

Aetiology of diseases of the skin

Skin disease can be caused by inherited abnormalities, parasites, viruses, bacteria, fungi, vitamin deficiencies, physical damage, and toxic causes. Inherited diseases include epitheliogenesis imperfecta. Sarcoptic mange is probably the most common cause of porcine skin disease and is an example of parasitic diseases. Viral diseases such as pig pox have lesions confined to the skin. In other viral diseases such as classical swine fever, many body systems including the skin are affected. Bacterial diseases affecting the skin are quite common. Some, such as greasy pig disease, have lesions chiefly confined to the skin. Others, such as swine erysipelas, have generalized signs including fever and depression in addition to characteristic skin lesions. Fungal diseases such as ringworm are relatively uncommon. A number of vitamin and mineral deficiencies can cause skin disease. Excessive selenium in the diet may give rise to alopecia and separation of the hooves at the coronary band. Traumatic injuries caused by fighting or accidental damage are examples of physical causes of skin disease.

Epidemiology of diseases of the skin

This is discussed in detail under each disease condition described in this chapter. In general, predisposing factors include direct contact between pigs. Injuries to the skin caused by fighting are likely to occur when pigs of any age are mixed. Accommodation that is not cleaned, disinfected, rested between batches of pigs, or kept in a good state of repair may predispose to an increased incidence of skin disease.

Clinical signs of diseases of the skin

These vary with the disease. Specific signs of skin disease may include discomfort and pruritus. In cases of sarcoptic mange, pruritus is intense; the affected animal may be unwilling to rest and spends much time rubbing its skin against any solid object. In pale-skinned pigs, sunburn causes reddening of the skin and sloughing of the surface layers. In black pigs, reddening of the skin is not evident but sloughing may occur, and in both categories of pig the lesions are very painful to the touch. Primary lesions are those caused by the causal agent, such as the characteristic diamond-shaped lesions of swine erysipelas. In pruritic diseases, primary signs may be superseded by secondary signs such as those caused by repeated rubbing of the skin.

Diagnosis of diseases of the skin

Skin lesions may be associated with skin disease or are the local manifestation of a generalized disease. Observation of the pig may help to assess the extent of any obvious skin lesions. Observation will also indicate whether there are behavioural signs, such as rubbing or head shaking, that might be caused by skin disease. Close visual examination of the skin should follow to confirm the presence and extent of any lesions. Palpation of the skin and lesions provides further information enabling a diagnosis to be confirmed. In a pig with subacute erysipelas, the animal is often dull and pyrexic. Raised diamond-shaped skin lesions are present, especially over the shoulders, and the skin of the pyrexic animal may be quite hot to the touch. The lesions may be palpable before they are clearly visible.

The clinician should identify the type of lesions that are present. In pig pox, for example, the sequence of skin lesions seen in pox diseases – papule, vesicle, and pustule – may be identified, helping to confirm the diagnosis. Laboratory tests may be required to confirm a

diagnosis. Sarcoptic mange may be suspected in a pruritic pig. The diagnosis of this condition can be confirmed by the isolation of the *Sarcoptes scabiei* mite from skin scrapings or earwax. In cases where the disease is suspected but mites cannot be found, an enzyme-linked immunosorbent assay (ELISA) blood test for mite antigens may confirm that recent infestation has occurred.

Treatment of diseases of the skin

This is aimed at treating the cause of the skin disease and restoring the skin to a healthy condition. The treatment for individual skin diseases is described under each condition below. In general terms, injuries are treated according to surgical principles, and the pigs should be housed where further injuries are less likely to occur. Washing the skin with a mild antiseptic such as chlorhexidine can be used in combination with parenteral antibiotics in the treatment of greasy pig disease. Ivermectin products are effective in the treatment of sarcoptic mange. Resting and cleaning the accommodation aid the efficiency of elimination and prevention of this disease. Skin health can be improved by the local application of liquid paraffin in the form of 'pig oils'.

Control of diseases of the skin

This depends on the disease concerned. Specific methods of disease control are discussed under each of the diseases described in this chapter. In general terms, maintaining a closed biosecure and 'disease-free' herd should help to keep out the infectious causes of skin disease such as sarcoptic mange. Where sarcoptic mange is endemic, a control or an eradication programme should be instituted. The effectiveness of such programmes should be monitored by regular observation and sampling of the pigs.

Careful management to avoid fighting among pigs and keeping them in a clean, well-maintained environment should help reduce the risk of physical injury. Commercial rations should contain adequate levels of vitamins and trace elements, but care must be taken with home-mixed rations to ensure that these are present in correct quantities. Cases of skin disease should be presented for veterinary examination at an early stage to enable treatment to be prescribed before serious skin damage, including secondary problems, becomes established.

Veterinary Investigation Diagnosis Analysis sample submissions: diseases of the skin, 1996–2003

See Table 6.1.

Table 6.1 Veterinary Investigation Diagnosis Analysis sample submissions: diseases of the skin, 1996–2003

Cause	Incidence (%)
Exudative dermatitis	39.26
No diagnosis	30.37
Ectoparasitic diseases (other)	12.22
Sarcoptic mange	5.19
Other diseases	4.07
Ringworm	3.70
Pig pox	2.59
Pityriasis rosea	1.85

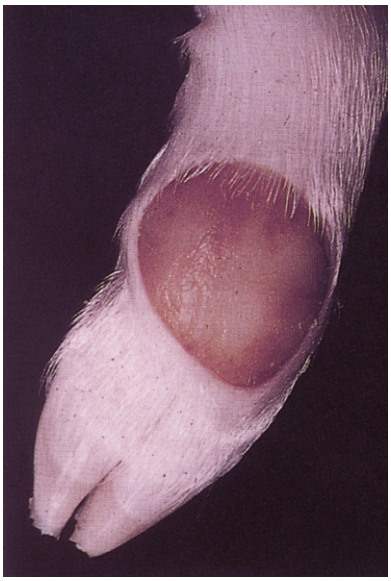

Fig 6.1 – Epitheliogenesis imperfecta lesions on the lower limb.

Inherited skin diseases

Epitheliogenesis imperfecta

Incidence

This is uncommon. It is most frequently seen in Large White and Saddleback breeds. A single piglet or a small number in a litter may be affected.

Aetiology

An autosomal recessive gene is responsible.

Clinical signs

A piglet is born with an area of skin on the flank or limb missing (Fig. 6.1). Death may occur in piglets with extensive lesions. In others, growth may be normal and occasionally some filling of the defect will occur.

Differential diagnosis

Neonatal injury (e.g. treading by sow) is possible.

Treatment

Self-resolution occurs occasionally. Surgical repair may be possible but uneconomic. In cases of severe skin deficit, euthanasia may be necessary

Hypotrichosis (hairless piglets)

Incidence
This is rare.

Aetiology
The cause is an autosomal dominant gene with incomplete penetrance. The condition is occasionally also caused by iodine deficiency.

Clinical signs
One or more piglets in a litter are born with no hair or very little hair. Piglets may also be very small and weak. They may not survive.

Diagnosis

This is based on clinical signs.

Differential diagnosis

Iodine deficiency is a possible cause.

Treatment

None is effective. Euthanize very weak piglets.

Control

Avoid breeding from affected animals or carrier stock.

Dermatosis vegetans

Incidence
Dermatosis vegetans is uncommon. It is confined to Landrace pigs in the UK.

Aetiology
The cause is a semilethal factor with recessive autosomal inheritance.

Clinical signs
Skin lesions are present at birth or develop in the first 3 weeks of life. Affected piglets show skin lesions on the lower abdomen and inner surface of the thigh. Initially, affected areas are erythematous; they then become wart-like and covered with brown crusts. The feet may also be affected; the coronets are inflamed and thickened. Irregular hoof growth occurs. Lung lesions also occur as a giant cell pneumonitis develops.

Treatment

None is effective. Some piglets die but others recover completely.

Control

Avoid breeding from affected strains.

Congenital porphyria

Aetiology
This is caused by a defect in porphyrin metabolism. Excessive porphyrin is produced and cannot be excreted in urine and faeces.

Clinical signs
Some porphyrin is deposited in tissues including bones and teeth, which have a reddish discoloration.

Treatment

No effective treatment is available.

Prevention
Avoid breeding from affected animals.

Parasitic skin diseases

Louse infestation

Incidence
Louse infestation is widespread.

Aetiology
The causal organism is *Haematopinus suis*, a large (5 mm), brown, dorsoventrally flattened sucking louse easily seen on the pig's skin (Fig. 6.2).

Epidemiology
Eggs are laid by lice on to hairs of the coat. They hatch into nymphs within 20 days. The life cycle is complete in 30 days. The pig is the only host. Lice can survive away

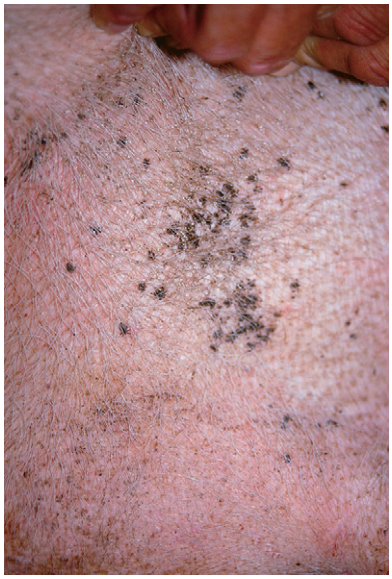

Fig 6.2 – Louse infestation. Note the dorsoventrally flattened large louse. (Courtesy of R.W. Blowey.)

from the pig for up to 3 days. They occasionally crawl on to animal attendants and veterinary surgeons but do not survive. Lice can transmit pig pox and *Eperythrozoon suis*.

Clinical signs

Heavy infestation causes skin irritation, rubbing, hair loss, weight loss, and occasionally anaemia.

Diagnosis

The louse is readily seen on the skin, especially in white pigs.

Treatment

A number of products can be used to treat lice or mange. These include doramectin, ivermectin, and amitraz. Drug use is described in greater detail under *Sarcoptic mange* below.

Control

Lice can be eliminated by regular treatment of the whole herd.

Sarcoptic mange

Incidence

Sarcoptic mange is widespread. Surveys have suggested that 20–90% of UK breeding herds may be affected.

Fig 6.3 – Sarcoptic mange. Note the mild skin inflammation in these pruritic animals.

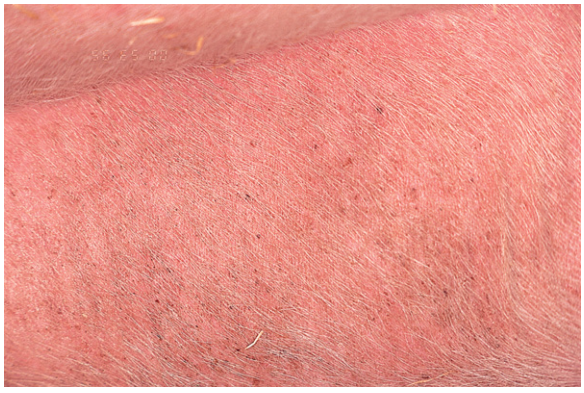

Fig 6.4 – Sarcoptic mange: a closer view of the skin in Fig 6.3.

Aetiology

Sarcoptes scabiei, a small (0.5 mm) burrowing mite, is responsible.

Epidemiology

The mites live on the pig and burrow into the skin, where they may induce an allergic reaction. This may result in increased pruritus, although mite numbers have decreased in the animal. Spread is mainly by direct pig to pig contact. The mites can live off the pig's body for up to 2–3 weeks in a damp environment. The adult female mite lays 50 eggs before her death. The life cycle can be complete in 7–14 days.

Clinical signs

In early infestation, intense pruritus is seen – the skin is red and inflamed (Fig. 6.3). As the infestation becomes established, the skin becomes thickened and there is a proliferation of underlying connective tissue. The head, flank, and legs are all affected. The skin becomes dry and crusty (Figs 6.4 and 6.5). Pruritus continues and may cause reduction in appetite and growth rates. Hair loss may follow. Rubbing and chronic head shaking can cause the development of aural haematomata. There may be an

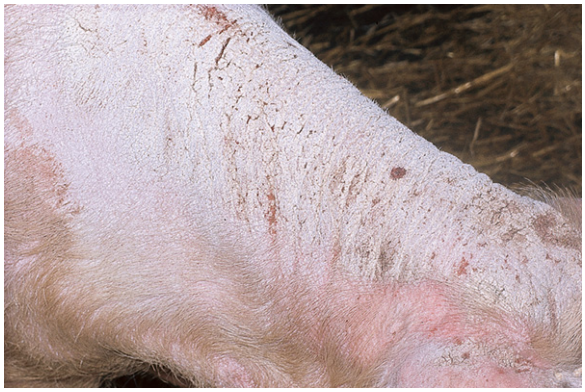

Fig 6.5 – Sarcoptic mange: a chronic case affecting the head and neck.

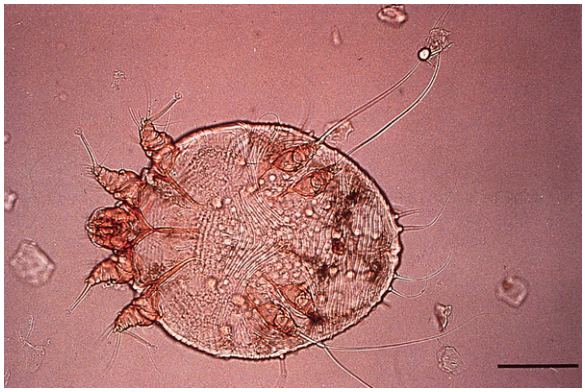

Fig 6.7 – Sarcoptic mange: mite identified by skin scraping.

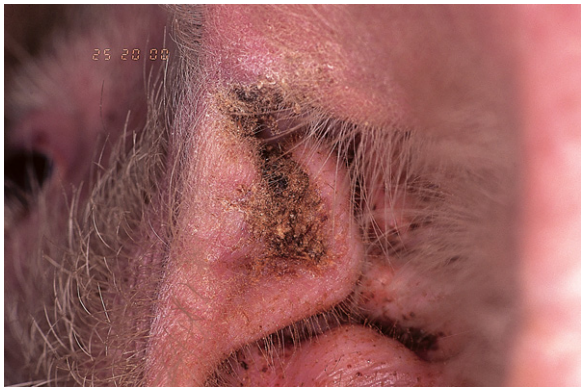

Fig 6.6 – Sarcoptic mange: dark brown earwax.

increased accumulation of dark wax in the ears and brown crusty exudate in the ear canal (Fig. 6.6). Several animals are usually affected within a group. Chronic infestations are commonly seen in breeding stock. Skin damage may reduce slaughter value.

Diagnosis

The condition is diagnosed by finding mites in earwax or skin scrapings (Fig. 6.7). Mite numbers may be low in chronic infestation, and they may be difficult to find. Response to specific treatment may aid diagnosis. Individual mites may be just visible in the dark earwax. A serum ELISA test is available to demonstrate the presence of antigens to the sarcoptic mite.

Differential diagnosis

Parakeratosis and exudative dermatitis are possible. Both are less common than sarcoptic mange, are non-pruritic, and mites are not found. Hyperkeratosis of areas of skin may be found in Vietnamese pot-bellied pigs. These are usually non-pruritic, and no mites are found in skin scrapings.

Treatment

A range of products for treatment are available. In many cases, two doses are given to enable developing larvae from recently hatched eggs to be destroyed with adult mites. Data sheets must be carefully consulted in each case, checking meat withdrawal times. Many of the products are potentially dangerous to people and must be used with care. Some examples are given here.

Doramectin is given by intramuscular injection at 300 mg/kg of body weight, with 49 days' meat withdrawal. Ivermectin is given by subcutaneous or intramuscular injection at 300 mg/kg of body weight, with 28-day meat withdrawal. An ivermectin premix for pigs can be given in the food over a period of 7 days, with 7-day meat withdrawal. Abamectin has been used successfully to treat porcine sarcoptic mange. Amitraz is effective against porcine lice and mange. It can be used as a spray.

Sows can be routinely treated shortly before farrowing, but herd eradication is advised. Meat withdrawal – check with drug data sheets.

Control

A number of regimens have been suggested. Whole-herd treatment at 6-month intervals may be used or individual sows can be treated 7–14 days before farrowing.

Eradication

This is advisable but requires determination and effort. Treat the whole herd with one of the drugs mentioned above at the same time. The buildings should be thoroughly cleaned and then rested for 3 weeks to ensure that no mites survive off the pigs. Repeat the treatment as

required. Keep a closed herd. Quarantine or treat new pigs coming to the farm.

Demodectic mange

Demodectic mange is rare. Small (3 mm) nodules and pustules may be seen on the skin. Abscesses may develop later. Mites (*Demodex phylloides*) are found in purulent material (Fig. 6.8).

Treatment

Amitraz is effective.

Fly worry

Blow fly strike can occur but is less common than in sheep. Very sick pigs with wounds and open abscesses may be at risk from strike.

Treatment

Clean up and treat open lesions. Local application of a fly spray or deltamethrin may help prevent reinfestation. General fly worry can be a nuisance in a very dirty environment, and attention to this should help prevent serious disturbance by flies.

Viral skin diseases

Pig pox

Incidence

Pig pox is potentially worldwide but not very common.

Aetiology

The cause is *Suipoxvirus* from the Poxviridae family.

Epidemiology

There is direct pig to pig spread, but it is also transmitted by lice and flies.

Clinical signs

Skin lesions are seen on the face, flanks, and belly of affected pigs. Small (1 cm) papules develop with vesicles that rupture to produce dark red scabs (Figs 6.9 and 6.10). A *congenital form* of pig pox has been reported; piglets are born with pox lesions from a clinically unaffected sow. A high mortality is seen in congenital cases. In young pigs, conjunctivitis and keratitis may be seen.

Diagnosis

Diagnose by virus isolation and identification from vesicles, using electron microscopy.

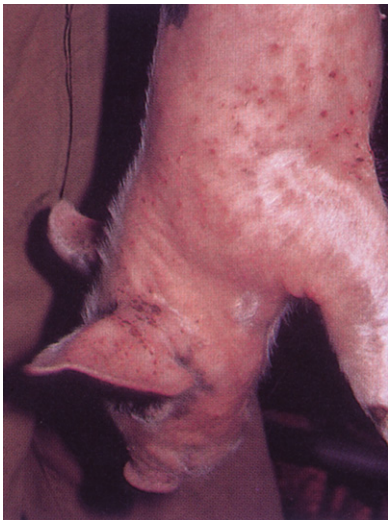

Fig 6.9 – Pig pox: affected pig showing mild inflammation of the skin.

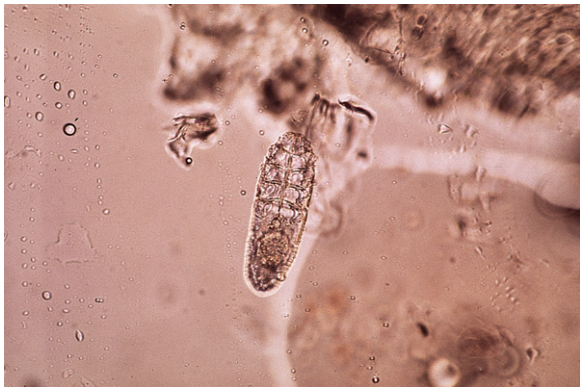

Fig 6.8 – *Demodex* mite.

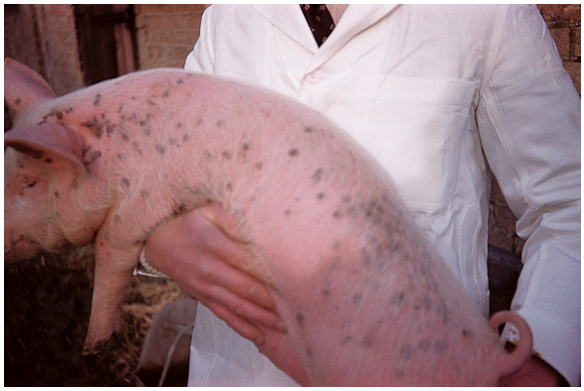

Fig 6.10 – Pig pox. Note the brown-black scabs of older lesions.

Treatment

There is no specific treatment. Antibiotics for conjunctivitis may help avoid secondary infection. Topical liquid paraffin may improve the condition of the skin.

Control

Deal with the louse problem in the herd and improve hygiene. Maintain a closed herd.

*The following virus diseases are often accompanied by skin lesions. The skin lesions are summarized below. For a full account of the diseases, see Chapter 11 (*Polysystemic diseases*).*

Classical swine fever

There is patchy purple discoloration of the abdominal skin. Necrosis of the ear tips with ulcerated areas is also seen.

Swine vesicular disease

Vesicles are seen on the skin of the coronary band. Vesicles and ulcerated areas are also seen on the snout, lips, and tongue.

Foot and mouth disease

Vesicles are present on the coronary band and snout.

Porcine dermatitis and nephropathy syndrome

Extensive dermatitis may be seen over the chest, abdomen, thighs, and forelegs. Affected areas show numerous purple-red nodules of varying size and shape.

Bacterial skin disease

Swine erysipelas

Skin lesions are an important sign of this disease. In peracute erysipelas, a generalized purple discoloration of the skin may be seen. Other signs include severe depression, pyrexia, and sudden death. In acute erysipelas, red, diamond-shaped elevated skin lesions are seen and are palpable, especially over the shoulders and back. As the lesions age, they become darker, and in rare cases sloughing of large areas of the skin of the back occur. For full discussion of this disease, please see Chapter 11 (*Polysystemic diseases*).

Salmonella cholerae-suis

This is probably the most important salmonella serotype associated with skin lesions. Skin lesions, including purple discoloration of the ears, are seen (possibly caused by diffuse intravascular coagulation); purple discoloration is also seen on the flanks, behind the elbows, and on the caudal aspect of the thighs. Note that these are signs of septicaemia and may also be seen in cases of severe mastitis caused by *Klebsiella* spp. For full discussion of salmonellosis, please see Chapter 5 (*Diseases of the gastrointestinal system*).

Spirochaetal granuloma

Spirochaetal granulomata are an unusual consequence of ear-biting injuries. There is local infection by spirochaetes of the injured ear tip or lower ear margin. Occasionally, this skin infection spreads to adjacent areas of the neck. Granulation tissue is present, and some bleeding may occur. Biting of the lesions by other pigs may occur.

Treatment

Treat with local cleaning and application of oxytetracycline spray.

Greasy pig disease (exudative epidermitis)

Incidence

Greasy pig disease is common.

Aetiology

Staphylococcus hyicus is responsible. This organism has virulent and non-virulent strains. Some strains of the organism produce exfoliative toxins that allow rapid intradermal spread of the disease. *Dermatophilus* spp. may be involved in some cases.

Epidemiology

The condition is mostly seen in pigs <6 weeks of age and especially <2 weeks. Infection is carried by the sow or gilt on her skin or within her vagina. Access of the infection is possibly aided by bite wounds inflicted by other piglets. One or two piglets or occasionally the whole litter may be affected. Further spread to other pigs may occur when the litter is moved to flat deck accommodation in warm damp conditions. High humidity and reduced oxygen levels predispose to infection. Infection can persist in farrowing quarters between litters.

Clinical signs

There is sudden onset of facial skin lesions, which are painful and may suppress suckling in affected piglets. The skin is initially reddened and then becomes thickened and covered with a greasy brown exudate that spreads over the body (Figs 6.11, 6.12 and 16.11). Ear tip necrosis may be seen in some animals. The lesions are non-pruritic. Infec-

Fig 6.11 – Greasy pig disease: piglet showing lesions on the head and neck.

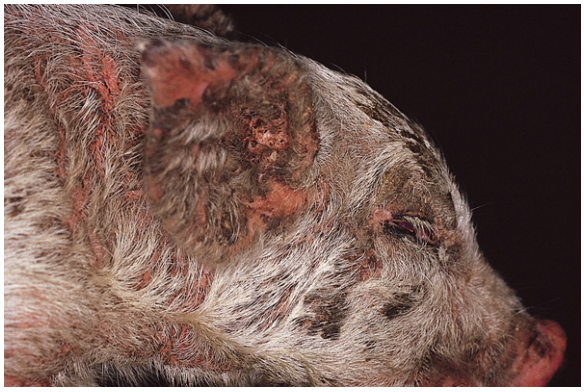

Fig 6.12 – Greasy pig disease: severe facial and neck lesions in a piglet.

tion occasionally spreads to the mouth and tongue. Secondary infection with other bacteria may also affect the skin. Death may occur in untreated piglets. Although chiefly a disease of piglets, localized lesions may occasionally be found in adult pigs.

Diagnosis

Diagnosis is based on clinical signs and culture of the organism. White precipitated material may be found in the kidney of dead affected piglets.

Differential diagnosis

Vitamin B deficiencies may cause somewhat similar lesions but no bacterial cause (see below).

Treatment

Treatment can be effective unless piglets are moribund, in which case euthanasia may be necessary. A sensitivity test may aid the choice of antibiotic. Parenteral penicillin, cloxacillin, neomycin, or novobiocin is often used successfully. Early treatment is essential, and care must be taken to encourage the piglets to feed. Lanolin-based cream with antibiotic and hydrocortisone can be applied locally. Washes with chlorhexidine can also be used. Treatments can be very effective but are labour-intensive.

Control

Control by good hygiene in the farrowing house, with an all in, all out system with pressure washing and disinfection between batches. Washing sows with antiseptic before they enter the farrowing house and clipping piglets' teeth are also helpful. Autogenous vaccination has been used successfully in some herds.

Fungal diseases

Ringworm

Incidence

Ringworm is relatively uncommon. It occurs more frequently in outdoor pig units and in growing pigs. Most pigs in a batch show lesions.

Aetiology

The causal organism is mostly *Trichophyton mentagrophytes*. *T. verrucosum* and *Microsporum canis* are seen occasionally.

Epidemiology

Ringworm is spread by pig to pig contact and also from spores, which can survive in wooden fitments for many months. *T. mentagrophytes* may be associated with rats, and *T. verrucosum* may be contracted from cattle or premises where they have been kept. There is also a risk of zoonotic spread.

Clinical signs

Round or ovoid slightly raised lesions are seen on the shoulders, back, and flanks (Fig. 6.13). Lesions are light brown and the skin may be flaky (Fig. 6.14).

Diagnosis

Diagnose by isolation of fungal hyphae and spores from skin scrapings.

Fig 6.13 – Ringworm: light brown lesions are present on the right shoulder.

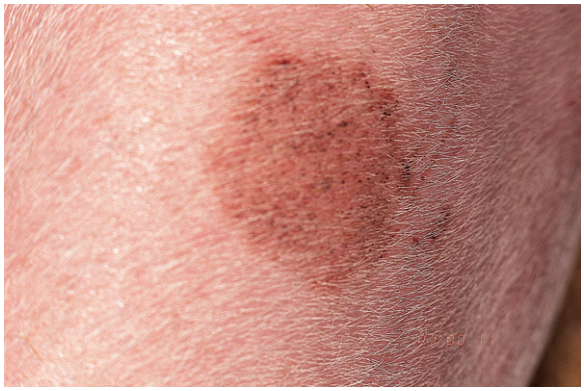

Fig 6.14 – Ringworm: single lesion showing slightly raised light brown lesion of *Trichophyton mentagrophytes*.

Differential diagnosis

- *Sarcoptic mange*. Clinical signs are pruritus and mites.
- *Greasy pig disease*. Lesions are more extensive, non-pruritic, and mostly in baby piglets.
- *Pityriasis rosea* looks like ringworm but only a few pigs in a batch are usually affected. In this disease, fungal hyphae are not present and rapid resolution without treatment occurs.

Treatment

Treatment is difficult, as no licensed treatment for pigs is now available. Enilconazole is licensed for use in cattle and could be used in pigs under the cascade system. A diluted solution is applied to the skin using a brush or spray; the relatively hairless and immobile skin of the pig allows most of the solution to run off the animal. Self-cure occurs in most cases after a few months.

Control

Identify species if possible to aid elimination of the source of infestation. Control vermin. After treatment and removal of affected pigs, thoroughly clean buildings.

Vitamin- and mineral-related abnormalities

Vitamin A deficiencies

Commercial diets should contain sufficient vitamin A, and deficiencies of vitamins are uncommon. Sometimes, vitamins are accidentally omitted from home-mixed diets or are destroyed when food is subjected to a high-temperature pelletting process. Skin lesions are a relatively minor sign of vitamin A deficiency. The coat is dry and has a shaggy appearance, with splits at the ends of the bristles. A non-specific seborrhoeic dermatitis is sometimes seen. More serious signs of vitamin A deficiency include central nervous system lesions, posterior paralysis, night blindness, and congenital defects. Not all are vitamin A-responsive. Diagnosis is by checking vitamin A levels in liver and serum and in the diet. Food should contain 3–9 million IU per tonne.

Treatment

Increase the level of vitamin A in the diet.

Vitamin B deficiency

Most modern diets should have adequate levels of the B vitamins. A number of the B vitamins can be associated with skin and other lesions. In cases of widespread or generalized skin disease, the B vitamin levels should be checked.

Riboflavin deficiency

Skin lesions include a roughened skin and matting of the hairs by a heavy sebaceous exudate. The condition may resemble greasy pig disease (see above). The clinician may also see poor growth, limb and eye abnormalities, scouring, and irregularities of the oestrous cycle.

Nicotinic acid deficiency

This can occur in pigs fed on high levels of maize. The skin may be yellow and show alopecia or irregular hair growth. At post-mortem, lesions similar to necrotic enteritis may be seen.

Pantothenic acid deficiency

There is a risk of low levels if the fat content of food is high or when copper supplementation is used. Skin lesions include dermatitis, patchy alopecia, and a dark exudate around the eyes. Additional signs include goose-stepping gait and diarrhoea. Demyelination, especially of the peroneal nerve, is seen at post-mortem. Treatment is by including 10–12 g of calcium pantothenate per tonne of food.

Biotin deficiency

The diet should contain 180 mg of biotin per tonne of food. Levels may fall in diets with high wheat or barley content. Skin lesions include alopecia, broken hairs, brown crusty lesions, and petechial haemorrhages. One may also see signs of lameness caused by cracks in the solar horn, which readily become infected. The response to biotin therapy is often very slow.

Parakeratosis and zinc deficiency

Incidence

This problem should be uncommon with commercial pig rations.

Aetiology

The cause is zinc deficiency in the diet or malabsorption of zinc from the bowel in animals with diarrhoea. High levels of soya and bran and also high levels of calcium in the diet can predispose to this deficiency.

Epidemiology

Signs are seen especially in piglets aged 7–10 weeks. The incidence is mostly lower in outdoor pigs.

Clinical signs

Poor growth and weight loss occur. One may see poor herd fertility and breeding results. Skin lesions are symmetrical erythema with crusty lesions on the flank, back, belly, and ears (Fig. 6.15). Deep fissures are seen between areas of hyperkeratinization. There is no greasiness and no pruritus. Some secondary abscessation of the skin may be seen.

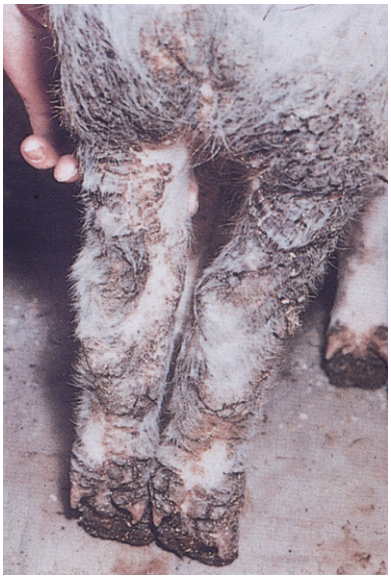

Fig 6.15 – Parakeratosis. Note the dry fissured skin on the hind legs.

Diagnosis

Base the diagnosis on clinical signs and analysis of diet.

Differential diagnosis

- *Greasy pig disease*. This is diagnosed by clinical signs, age affected, and culture of the causal organism.
- *Sarcoptic mange*. Diagnosis is based on clinical signs and detection of mites.

Treatment

Treat with 200 g of zinc carbonate or sulphate per tonne of food. It helps to put affected animals out to grass if practicable.

Control

Ensure adequate zinc levels in the diet.

Physical causes of skin disease

The pig's skin is very susceptible to injury and infection, often as the result of a poor environment or fighting between pigs. Pecking injuries by birds have been reported on some outdoor units (Fig. 12.15).

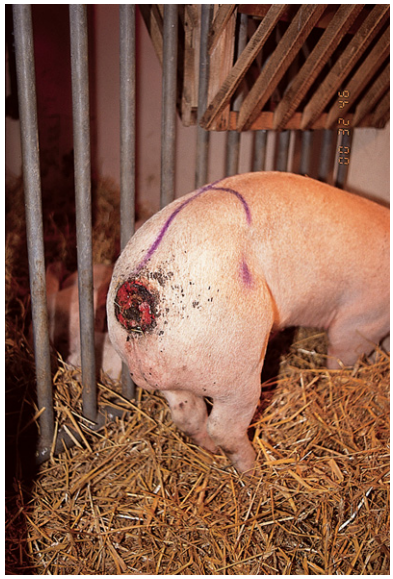

Fig 6.16 – Tail bite injury: almost the whole tail has been bitten off.

Tail biting and ear biting

Vulval and anal biting are also seen.

Incidence

These vices are widespread and the cause of much suffering and loss.

Aetiology

This problem is mainly the result of poor husbandry and inadequate accommodation and feeding. Low temperature, poor ventilation, inadequate feeding and drinking facilities, lack of bedding, and boredom have all been shown to influence the incidence of tail biting. Ear-biting and flank-biting lesions may become infected through invasion of minor injury sites by *Staphylococcus hyicus*.

Epidemiology

The vice usually starts with one pig nibbling the tail of another animal. The first animal may damage the tail tips of every pig in the group within hours. Once the damaged tails start to bleed, other pigs are attracted to them and start biting. Ear biting may be more prevalent in pigs that have been docked to prevent tail biting.

Clinical signs

There are varying degrees of tail or ear damage. Severe damage can occur rapidly – the whole tail can be removed, and deeper biting damage into the perineal tissues can occur (Figs 6.16 and 6.17). Infection can enter the damaged tail and spread upwards through local invasion or by haematogenous means. Vertebral body abscesses are a common sequel and can result in pathological fracture followed by posterior ataxia or paresis (Fig. 6.18). Ear biting can affect all parts of the pinna, and the caudal border is a particularly common area to be damaged (Fig. 6.19).

Fig 6.17 – Extensive tail bite injury with the lesions extending into the muscles of the perineum.

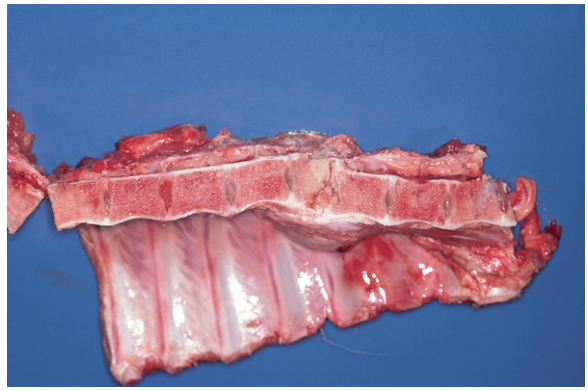

Fig 6.18 – Pathological vertebral body abscess and fracture from infection carried haematogenously from a tail bite injury.

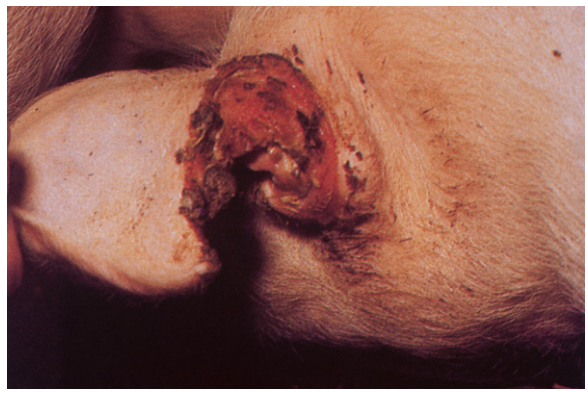

Fig 6.19 – Ear bite lesion: a large portion of the caudal border of the pinna has been lost.

Treatment

Minor injuries are left untreated. Any sign of infection should be treated with parenteral and local antibiotics. Pigs that have been severely damaged may have to be euthanized on humane grounds. Isolate affected pigs.

Prevention

Improve deficient management. Chains and tyres have been put into pens to 'keep the pigs occupied' but are of doubtful value. Identify and if possible remove individuals who start the biting cycle. Tail docking has some benefits in reducing the incidence of the problem.

Tail necrosis in piglets

This may be caused by damage sustained by trauma to the dorsal or ventral surfaces by contact with hard or rough floor or walls; the latter can occur when pigs are suckling.

Teat necrosis

Teat necrosis can occur as a result of damage being sustained to the teats by roughened flooring or mesh flooring in the farrowing house. The teats are vulnerable to injury as the piglets struggle to reach their teat to suckle. Local antibiotic ointment may aid repair, but severe teat damage can be sustained, preventing teat function in adult life. Unsuitable flooring should be avoided.

Flank rubbing and penis sucking

These have been reported as having similar origins to tail and ear biting but are much less common.

Sunburn

(See also *Phytophotodermatitis* below.)

Incidence and aetiology

Sunburn is quite common, especially in outdoor pigs lacking shelter from the sun. White pigs are particularly susceptible. Aggressive members of the herd may prevent other pigs entering shady places to avoid sunburn.

Clinical signs

Painful, reddened areas of skin are seen on the back and flanks (Fig. 12.10). Vesicle formation may occur. Movement is very painful, and affected animals are reluctant to move. They may stand in a 'rocking horse' stance as they try to avoid discomfort in their sore backs.

Diagnosis

The diagnosis is based on recent sunny weather and clinical signs. Ensure that no other causes of back injury are present.

Treatment

Treatment is unnecessary in mild cases. In severe sunburn, local emollient cream may be applied. Flamazine may speed repair to damaged skin but is expensive.

Prevention

Ensure that adequate shade and wallowing facilities are available (Figs 12.11 and 12.12).

Heatstroke

Incidence

Heatstroke is quite common. The pig is very susceptible to overheating; perspiration is minimal, and a thick layer of subcutaneous fat insulates the body. See also notes on porcine stress syndrome.

Aetiology

The condition can occur if pigs are exposed to very hot weather in the absence of effective shade. Hot buildings (e.g. corrugated iron shelters exposed to the sun) can become very hot inside. Sows can become very hot if their farrowing quarters are excessively heated by infrared lamps and other heaters, especially if they become stressed with farrowing difficulties. The situation is worsened by lack of ventilation or a failure of the ventilation system. Black pigs are especially susceptible to heatstroke, as are overweight pet pigs.

Clinical signs

Pigs show progressive distress. Panting and open-mouthed gasping respiration are seen. Temperature is >42°C. Membranes may become cyanotic. Tachycardia occurs. Death follows.

Diagnosis

Base this diagnosis on clinical signs and a history of exposure to heat.

Differential diagnosis

Check for the possible presence of acute disease, usually a history of inappetance with disease but not heatstroke.

Treatment

Cool the pig immediately. Use cold water hosing and increase ventilation. Ensure that drinking water is available and avoid attacks by other pigs.

Accidental burns

Incidence and aetiology

Regrettably, these occur quite frequently, often as a result of overheating or explosion of heat lamps in the piglets' creep area. The straw and wooden construction of older piggeries can rapidly lead to serious fires with extensive loss of life.

Clinical signs

These vary from minor superficial burns to severe and extensive burns. Animals may die from asphyxia through smoke inhalation and shock (Fig. 6.20).

Treatment

Treat with local applications as for sunburn (above). Analgesia and antibiotic cover may be needed if infection of damaged tissue is thought likely. Euthanasia may be necessary for badly injured animals.

Frostbite

Frostbite is relatively uncommon in the UK. In outdoor herds, one may see piglet deaths from exposure to cold and necrosis of ear tips through cold.

Electrocution

This is usually the result of an electrical fault such as poor wiring. The pigs involved are usually found dead. Superficial burn marks may be found on the skin. Signs of a violent death are present. Fractures of bone, especially of the neck of the scapula or the humerus, are common – these are caused by violent uncoordinated muscle contraction. Blood-stained froth may appear at the nose, and premature decomposition may occur. The clinician must treat suspected cases with care in terms of electrical safety and also check that there is no infectious cause of sudden death, such as anthrax.

Fighting injuries

Incidence and aetiology

Pigs naturally fight when mixed with animals from another group. Escape from aggressive fellow pigs is often not possible in the restricted areas of pig housing. Severe injuries and death can be sustained. The risk of fighting must always be remembered if pigs are isolated for treatment or removed from pens for even short periods. Putting pigs back can cause them to be subjected to severe damage and injury. Pigs in the same litter may suddenly start fighting for no apparent reason. Fighting injuries in sows have increased since the banning of sow stalls. Younger, lighter sows and gilts are more likely to be victims of bite wounds when housed.

Clinical signs

These vary from minor and superficial lacerations caused by teeth to deep gashes penetrating the whole skin thickness and caused by the tusks of adult pigs, especially boars. The injuries are typically on the flanks as fighting pigs stand shoulder to shoulder, using their teeth to damage each other (Fig. 6.21). Secondary infection, especially by *Arcanobacter pyogenes* and spirochaete species, occurs in many cases. Facial injuries caused by sharp incisor teeth are seen in piglets (Fig. 6.22) Tetanus can occur in areas where spores are found.

Fig 6.20 – Lesions caused by burning in a sow that died in a farm fire.

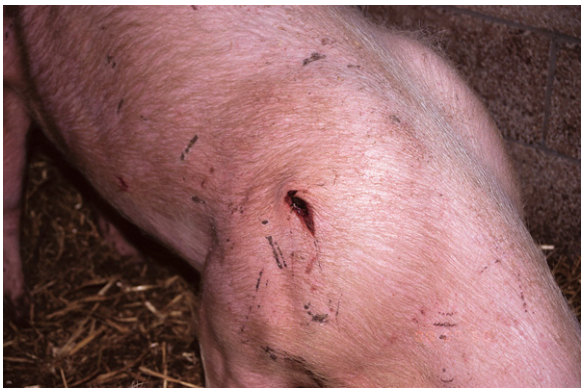

Fig 6.21 – Sow with laceration on her shoulder caused by fighting.

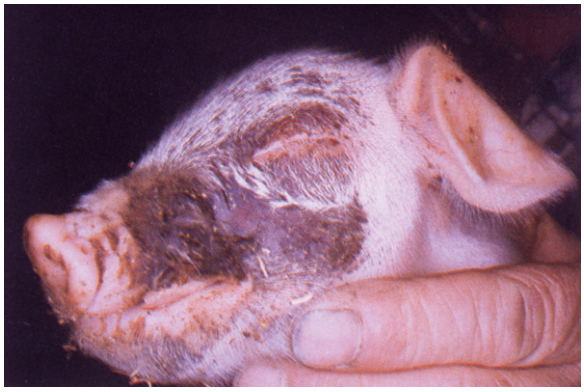

Fig 6.22 – Piglet with facial bite injury.

Fig 6.24 – Injection site nodule following a subcutaneous injection in the neck.

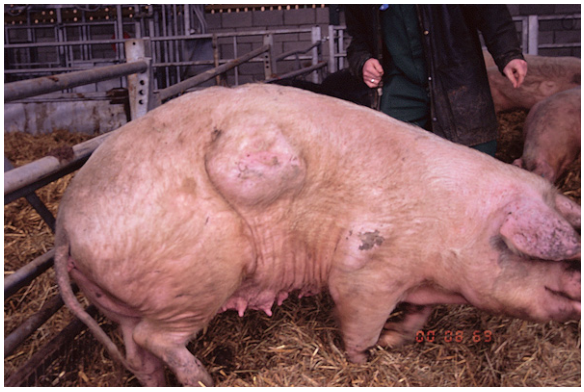

Fig 6.23 – Flank haematomata caused by fighting injuries.

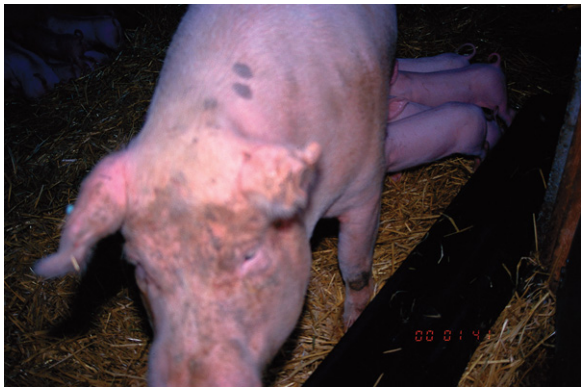

Fig 6.25 – Organizing aural haematoma: 'cauliflower ear'.

Treatment

Minor lacerations usually heal by primary intention. Deep wounds must be sutured under sedation and local anaesthetic. Infected wounds should be cleaned with an antiseptic solution such as chlorhexidine. Abscesses must be opened and checked to make sure that premature closure and reformation of further abscesses do not occur. Antibiotic cover and tetanus antitoxin must be used when necessary. Haematoma formation can occur as a result of fighting injuries (Fig. 6.23). Small abscesses at the site of subcutaneous injections in the neck but not caused by fighting are also seen (Fig. 6.24).

Prevention

Avoid the causes of fighting. If groups have to be mixed, sedation of all the pigs involved with azaperone before mixing may help, but results can be disappointing. Anti-fight aerosols, which usually contain oil of citronella, are of little value.

Aural haematoma

This is usually the result of head shaking or a bite from another animal. Sarcoptic mange (see above) commonly involves the outer ear, causing head shaking and rubbing of the ears. In many cases, the haematoma becomes infected. The weight of blood or pus in the pinna of the affected ear may cause the pig to have a head tilt in the direction of the abnormal ear. If untreated, the haematoma will organize and the ear shrinks and is known as a cauliflower ear (Fig. 6.25). If infected, the ear may burst and discharge pus. It often refills unless the abscess is dealt with effectively.

Treatment

See Chapter 15 (*Analgesia, anaesthesia, and surgical procedures in the pig*).

Phytophotodermatitis

This can be a problem with outdoor pigs and may be regarded as a form of contact photosensitization. Cases

have been reported following grazing in a field of parsley during the summer. Pigs developed quite extensive skin lesions on the snout. These were mainly skin fissures and erythema, and later some necrosis. Lesions disappeared when pigs were moved away from parsley. Parsley and other Umbelliferae contain psoralens. These substances are photoreactive when exposed to the ultraviolet part of sunlight and combine with cell DNA, causing death of superficial cells. Similar problems are seen in human parsley cutters. Contact with these plants should be avoided.

Pressure injuries

Pressure point injury and necrosis are commonly seen in growing and finishing pigs with insufficient bedding. Lesions were frequently seen when sows were kept in stalls or tethered. The shoulders, elbows, knees and hocks, and surrounding tissues are most commonly affected (Fig. 6.26). Initially, the skin in affected areas is inflamed. Callus formation may follow when subcutaneous connective tissue becomes fibrosed. In other cases, pressure causes the skin to ulcerate and some local bleeding may occur. Pressure necrosis occurs in other cases, leading to the local death of skin and adjacent tissues. Treatment is seldom necessary.

Pressure injuries are usually the result of poor bedding or flooring. Fluid-filled bursae may also occur, especially over the hock and carpus. If they become infected, they should be opened and drained as abscesses. In piglets, teat necrosis can result in permanent damage being done to the teats often in the first week of life. Twenty per cent of teats from individual piglets can be lost, with affected animals being unable to raise a litter when mature. Some varieties of mesh flooring in the farrowing quarters lead to piglet teat injury, pressure, and subsequent necrosis. Roughened concrete in the farrowing area can also lead to teat injury and to necrosis of the skin of the knees. These injuries can allow access of opportunist pathogens such as *A. pyogenes*, which can lead to septicaemia and joint ill.

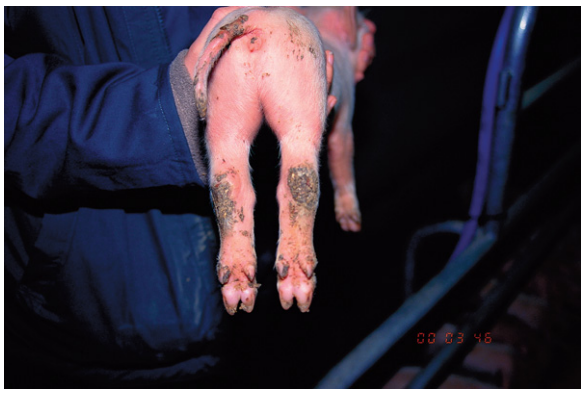

Fig 6.26 – Piglet with superficial pressure injuries on hocks.

Toxic causes of skin disease

Selenium toxicity

Incidence

Selenium toxicity is uncommon, but outbreaks can cause severe losses.

Aetiology

Levels of selenium >5 mg/kg are found in the diet. This can occur through excessive dosing to prevent selenium deficiency diseases such as mulberry heart or white muscle disease in pigs.

Clinical signs

In acute toxicity, there is respiratory distress, prostration, and death. Chronic toxicity causes polioencephalomalacia with ataxia. Poor conception rates in sows are seen. Skin lesions include alopecia. Hoof abnormalities, including separation at the coronary band, are present.

Diagnosis

This is based on clinical signs, selenium supplementation, and analysis of diet. High levels of selenium in the blood and hepatic necrosis are found at post-mortem examination.

Differential diagnosis

A differential diagnosis is vitamin A deficiency.

Treatment

There is no effective treatment.

Ergot poisoning

Ergot poisoning is an uncommon condition. Pigs may occasionally gain access to ergot from contaminated grain or at pasture. A wide spectrum of abnormalities is seen. Skin lesions and gangrene of the extremities can occur. One may also see high levels of stillbirth, agalactia, and abnormalities of the oestrous cycle. Treatment of gangrenous extremities may require surgical aid, local antibiotic therapy, or euthanasia.

Identification of the source of the ergot is necessary to prevent further cases.

Diseases of uncertain aetiology

Pityriasis rosea

Incidence

Pityriasis rosea is quite common on many farms. Usually, only one or two pigs in a group are affected. Although it is not dangerous and will usually self-cure, its presence may compromise the sale of individual young pigs.

Aetiology

Genetic predisposition and familial tendency have been suggested, but the exact cause is unknown.

Epidemiology

Pityriasis rosea is mostly seen in individual pigs aged 10–16 weeks. It appears to be self-limiting and often disappears from affected animal as quickly as it comes.

Clinical signs

Skin lesions are seen chiefly on the ventral surface of the abdomen and the medial aspect of the thighs. They can be easily missed on routine examination and may not be seen until the pig is turned up. Lesions are initially small red nodules, which develop into larger raised plaques with thin crusty covering. Lesions expand centrifugally, leaving normal skin within the irregularly circular lesions (Fig. 6.27). There are usually no systemic signs.

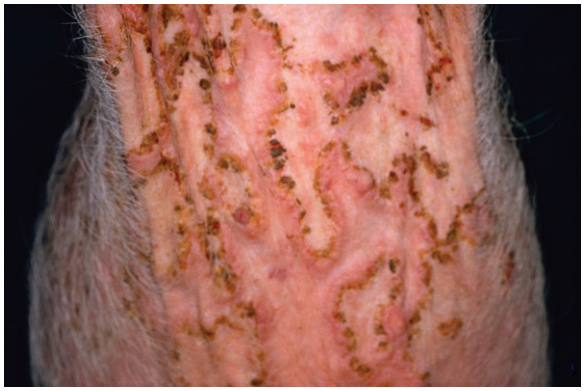

Fig 6.27 – Pityriasis rosea.

Diagnosis

The condition looks very like ringworm but is more common. No fungal hyphae are found. Fewer pigs are affected than in most ringworm outbreaks. The condition has been shown to be histologically a psoriasiform epidermal hyperplasia.

Treatment

There is none. Emollient cream may speed spontaneous resolution. Complete recovery within 6 weeks is usually seen.

7

Diseases of the nervous system

Introduction

Abnormal neurological signs are quite frequently seen in pigs and may be caused by a number of adverse factors, which are discussed below. Detailed clinical assessment of the nervous system of the pig is quite difficult. Some of the clinical neurological tests, such as the hopping and wheelbarrow tests regularly used in small animals, are not well tolerated by the pig. Nonetheless, a detailed clinical examination and assessment of the nervous system will usually allow lesions to be localized and a diagnosis made.

Aetiology of diseases of the nervous system

A wide range of causes are involved and must be considered when investigating neurological signs in pigs. A number of infectious diseases, especially those caused by viruses and bacteria, produce neurological signs. Some virus infections, such as the Teschen's disease enterovirus, cause signs chiefly involving the nervous system. The pestivirus that causes classical swine fever affects many body systems, including the central nervous system (CNS). Various serotypes of *Streptococcus suis* are frequently associated with neurological signs in infected pigs. Such signs are also caused in some cases by *Haemophilus parasuis*. Infection by this, the causal organism of Glasser's disease, produces a polyserositis affecting a number of body systems including the CNS. In some cases, bacteria produce toxins that directly (e.g. the neurotoxin in tetanus) or indirectly (e.g. the verotoxin in bowel oedema) affect the nervous system. Severe hypoglycaemia in neonatal piglets may cause fitting in the terminal stages. Signs of fitting are also caused by sudden water deprivation in older pigs. Liver disease in pigs is relatively uncommon, but in severe cases CNS signs may be caused by hepatic encephalopathy. Access to poisonous substances such as arsenic or selenium will cause neurological signs. Deficiencies of vital dietary ingredients such as pantothenic acid may also cause neurological signs.

Epidemiology of diseases of the nervous system

This depends on the cause of individual diseases and is discussed in greater detail below. Infectious agents are usually brought into a pig unit by infected pigs and possibly by vehicles or people who have contact with an infected herd. In a recent UK outbreak of classical swine fever, the source of pestivirus was believed to be a meat sandwich discarded by an overseas lorry driver. Tetanus is more common in pigs living in outdoor units, where the risk of contact with soil containing the spores of *Clostridium tetani* is much greater than in indoor units. The incidence of tetanus was greater when pigs were routinely castrated, producing open wounds through which infection could enter the body.

Clinical signs of diseases of the nervous system

These vary with the specific cause of disease. The necessity of performing a detailed clinical examination of the pigs is, as with other groups of porcine disease, essential. Observation of the pigs before they are handled is important so that abnormalities of behaviour can be observed. At this time, abnormalities such as a head tilt or circling may be seen. Encouraging the pigs to move slowly may reveal abnormalities of gait or balance. Rapid movement by the pigs, especially if they are in a group, may mask such abnormalities, especially if they are discreet.

Diagnosis of diseases of the nervous system

This is based initially on the history of the unit and the pigs. Observation of the pigs before handling them enables the clinician to observe any gross abnormalities of the nervous system. A clinical examination of affected pigs follows. If necessary, a more detailed examination of the nervous system should be performed. Further tests, for example to identify infectious causes of disease, may be necessary. In many cases, a post-mortem examination with samples taken for histological examination will be helpful in confirming a diagnosis.

Neurological examination

This should indicate what lesions are present and their likely point of origin in the CNS. A detailed clinical examination of affected pigs should always be performed. Individual pigs should be temporarily removed from their pen so that a detailed neurological assessment can be made.

The extent of this examination will depend on the degree of consciousness and mobility of the individual pig. An unconscious piglet suffering from meningitis can be examined without difficulty, but a finisher pig with a mild localized neurological deficit may be very difficult to examine without sedation. Physical restraint is often counter-productive in such cases, as struggling by the pig can mask subtle neurological deficiencies.

Neurological examination in the severely ill patient can be unrewarding. Abnormalities or deficiencies of the nervous system may be the result of weakness rather than specific neurological pathology.

The neurological assessment should be methodical and is aimed at identifying abnormal clinical signs involving the nervous system. Description of every detail of the examination is beyond the scope of this handbook, but the general approach is summarized below.

Observation

Any abnormalities of posture, balance, and gait? Can the pig see and hear? Is the animal dull or hyperaesthetic? Such gross abnormalities may include opisthotonus, which is frequently seen in animals with tetanus or meningitis.

Examination of the head

Are there any injuries or abnormalities such as an aural haematoma, which might cause physical tilting of the head? Purulent material in the outer ear may indicate the presence of a middle ear infection with vestibular damage in a pig with head tilt.

Observation of the eye

Is nystagmus present? This is a frequent finding in cases of meningitis. The cornea may be severely damaged in pigs that have been in lateral recumbency for some time. Does the pig have the following reflexes: fixation, palpebral, menace, photomotor, and pupillary light response reflex? Ophthalmoscopy is seldom used in pigs unless they are unconscious, when it may confirm slight nystagmus or intraocular lesions.

Specific cranial nerve lesions

These are seldom seen or recognized in the pig but include such abnormalities as facial paralysis associated with compromise of function of the facial nerve (the seventh cranial nerve).

Cerebral lesions

Pigs with lesions or inflammation in this part of the brain may show an alteration in their mental state. This may range from depression and stupor to hyperaesthesia and mania. Convulsions may be of cerebral origin but can also arise as a result of inflammation in the brainstem and thalamus. Infectious agents are the main cause of cerebral lesions causing encephalitis and meningitis. Cerebral signs can also be caused by space-occupying lesions such as abscesses and less commonly neoplasia.

Cerebellar lesions

These may interfere with the normal function of this part of the brain, including unconscious proprioception. Abnormalities indicating cerebellar lesions include intention tremor, dysmetria, and a high stepping gait. Normal muscle strength is usually present.

Vestibular lesions

These occur most commonly as a result of infection gaining access to the vestibular region from otitis media.

Affected pigs have an ipsilateral head tilt. They may circle or less commonly roll laterally until they come in contact with a solid object that prevents further rolling. In mild cases, the pig lives and thrives with a head tilt but may occasionally have a staggering gait.

Upper motor neuron lesions

These may cause isolation of peripheral nerve function from central control. The animal may be unaware of the spatial position of an affected limb. Weakness or increased extensor tone may occur, and some reflexes appear exaggerated.

An abnormal crossed extensor reflex may be present, especially when the hind limbs are involved.

Lower motor neuron lesions

These may cause a reduction of local muscle tone and rapid loss of muscle mass through atrophy. Reflexes may be reduced or absent, and paralysis may be present.

Location of spinal cord lesions

These can be identified by evaluating a number of the reflexes above and below a spinal cord lesion. Lesions may be caused by injury and by local infection. Vertebral body abscesses sometimes arise through haematogenous spread from a tail bite injury. These may result in a pathological fracture with compression of the spinal cord.

A number of reflexes are used to attempt to locate the site of a spinal lesion. These include the skin sensitivity test, the panniculus test, the pedal withdrawal reflex, and the patellar reflex.

Skin sensitivity test

This is done by stimulation of the skin and observation of the animal's conscious response. The skin may be stimulated by gentle pricking with a needle. A conscious response from the pig suggests that the spinal cord and the peripheral sensory nerve are intact. An exaggerated response may be produced proximal to the spinal lesion. A reduced or absent conscious response is found distal to the lesion.

Panniculus test

In this test, a needle prick along the dorsal part of the body produces a twitching movement of the panniculus muscle. Sudden loss of this reflex as the clinician tests the patient caudally may indicate a spinal lesion at that point. In older pigs, the thickness of the dorsal skin reduces panniculus movement and the usefulness of the test.

Pedal withdrawal reflex

This is tested by pinching the skin between the second and third digits of the forelimbs and hind limbs. If all nerve pathways are intact, the foot is rapidly withdrawn by the pig that is aware of the discomfort caused. An exaggerated response may indicate an upper motor neuron lesion proximal to the reflex arc. A reduced response may indicate compromise of the lower motor neurons involved in the reflex.

Patellar reflex

Tapping of the straight patellar ligament in the partially flexed hind limb results in rapid extension of the limb in the normal animal. The test can only be applied reliably to the sedated or unconscious pig.

Cervical cord lesions

A spinal cord lesion in the neck (cervical vertebrae C1–6) may result in upper motor neuron defects in the forelimbs and hind limbs.

Thoracolumbar cord lesions

A lesion slightly lower down the spinal cord (thoracic vertebra 3 to lumbar vertebra 2) does not affect the neuron outflow to the forelimbs that are normal. The upper motor neurons of the hind limbs are compromised, but their lower motor neurons may be normal. The animal has paralysis in its hind legs and normal or exaggerated pedal withdrawal reflexes. It may adopt a 'dog sitting' position.

Further diagnostic tests to identify the location of spinal cord lesions include contrast radiography or a magnetic resonance imaging scan. These would be difficult to justify on economic grounds in individual pigs.

Peripheral nerve lesions

Damage to specific peripheral nerves may occur as a result of local pressure on them or less commonly through damage to their spinal roots. Loss of function may follow. Skin sensitivity may be reduced, and muscle atrophy may occur. A number of nerves can be affected, including the radial, sciatic, and peroneal. Peripheral nerve lesions are much less common in the pig than they are in cattle. Deposits of fat and heavy muscling in mostly older pigs offer greater protection to underlying nerves than is found in cattle.

Evaluation of gait and posture

This evaluation may be difficult in the individual pig. Attempts to escape from restraint or from an obstacle may result in frenzied activity, which makes observation of individual limb function difficult. When possible, the pig should be encouraged to move at different paces and in different directions. The ability to negotiate steps, for example between pens, is observed. Abnormal gait, for example the hypermetria often associated with cerebellar lesions, may be visible.

Evaluation of proprioception

This is usually possible only in piglets, where evidence of upper motor neuron damage may be demonstrated.

The *wheelbarrow test* involves lifting the front or hind end of the piglet up and encouraging it to walk on its other two legs. This should present no problem to the normal animal. Evidence of poor proprioception may be seen in animals with upper motor neuron damage. Other tests include the hopping, placement, and hemiwalking tests. Many of these tests will be found impossible to perform on adult pigs.

Collection and analysis of *cerebrospinal fluid* (CSF) and serum may be useful in some cases. CSF is most easily obtained by lumbar puncture at the lumbosacral space.

A *post-mortem examination* with histopathology and the collection of appropriate blood or tissue samples may help confirm a diagnosis. Obtaining diagnostic samples from the brain may require the help of a specialized laboratory.

A number of diseases affecting the nervous system, such as Teschen's disease and classical swine fever, are **notifiable**. The divisional veterinary manager of the Department for Environment, Food and Rural Affairs (Defra) must be informed if a notifiable disease is suspected.

Treatment of diseases of the nervous system

This will depend on their cause. In the cases of systemic diseases such as Glasser's disease, antibiotic therapy against the causal agent may help reduce the risk of spread of infection to the nervous system of individual pigs. Early treatment is essential before irreversible damage is done to tissues including those of the nervous system. Once severe neurological signs such as nystagmus are present, treatment is much less likely to be successful. Euthanasia may be required on welfare grounds if a pig is unlikely to survive a neurological disease. It may also be required if an animal is left with a chronic lesion incompatible with its future comfort and growth.

Control of diseases of the nervous system

This will again depend on the cause. In cases of specific disease such as tetanus, a programme of vaccination can be used on a farm where the incidence of disease is unacceptably high. Strategic antibiotic therapy may be useful on farms where piglets of a certain age are likely to be

affected by endemic *S. suis* infection. Exposure to antibiotic therapy just before the time of peak incidence is likely to occur may help reduce the incidence of clinical cases. In general terms, the maintenance of a closed herd with high standards of cleanliness and welfare may help avoid the introduction of neurological and other diseases.

Veterinary Investigation Diagnosis Analysis sample submissions: diseases of the nervous system, 1996–2003

See Table 7.1.

Viral diseases

Teschen disease and Talfan disease

Incidence

Teschen disease occurs in central Europe, the USA, and Scandinavia. Talfan disease is reported occasionally in the UK and Denmark, where it has a low incidence. Teschen disease causes an *acute* polioencephalomyelitis. Talfan disease causes a *subacute* encephalomyelitis. Teschen disease is **notifiable** in the UK and Europe.

Aetiology

Both diseases are caused by *enteroviruses*, and other members of this group can also be involved in CNS diseases in pigs.

Epidemiology

Viruses enter the body via the gastrointestinal or respiratory tract. Rapid spread occurs in naive herds, with sporadic clinical cases, but infection is mostly subclinical. Morbidity and mortality can be high in young piglets.

Clinical signs

- *Teschen disease*. Pigs are initially dull and pyrexic (temperature 40–41°C), and then CNS signs including tremor, convulsions, and paralysis are seen. Any disturbance of the pig may stimulate convulsions. Some pigs recover over a few days, others are left with a flaccid paralysis.

Table 7.1 Veterinary Investigation Diagnosis Analysis sample submissions: diseases of the nervous system, 1996–2003

Cause	Incidence (%)
Streptococcal meningitis	70.50
Meningitis or encephalitis	17.55
No diagnosis	9.21
Other diseases	1.44
Spinal abscess	0.86
Cerebrospinal angiopathy	0.43

- *Talfan disease*. Clinical disease is mostly seen in piglets <2 weeks of age. Gilts, sows, and older pigs are less commonly affected. Initially, dullness, anorexia, possible vomiting, weight loss, and diarrhoea are seen. Temperature is normal or mildly elevated. CNS signs are seen within a few days: hyperaesthesia, muscle tremors, knuckling of fetlocks, walking backwards, lateral recumbency, convulsions, and death. Sows and gilts may show hind leg weakness and ataxia.

Survival of infected pigs appears to be age-related. Mortality is higher in younger pigs, and older pigs are more likely to survive.

Diagnosis

Clinical signs are suggestive. An enzyme-linked immunosorbent assay (ELISA) and a virus-neutralizing blood test for antibodies are available. Virus isolation is used. Tissue histopathology may reveal a non-suppurative encephalomyelitis.

Differential diagnosis

- *Aujeszky's disease* is generally more severe and not present on the British mainland.
- *Tetanus*. Signs including intermittent convulsions are distinct.
- *Vomiting and wasting disease (haemagglutinating viral encephalomyelitis)*. Coronavirus is detected.
- *Streptococcus suis* type 1. Isolation of the organism is used.

Treatment

None is effective other than nursing care for survivors.

Control

Maintain a closed herd. Teschen disease vaccine is available in Central Europe.

Vomiting and wasting disease

For a full discussion of this condition, see Chapter 5 (*Diseases of the gastrointestinal system*).

Nervous signs are sometimes seen in outbreaks of this disease, which is caused by a haemagglutinating encephalomyelitis virus – a coronavirus. Clinical evidence of the disease is now quite rare, but serological evidence suggests that many UK pigs have been exposed to the virus. Several

litters are affected in outbreaks. The youngest unweaned piglets may show the encephalitic form.

Affected animals show depression and a transient pyrexia. This is followed by hyperaesthesia, muscular tremors, and some vomiting. Terminally, there are convulsions and death. Older pigs show prolonged vomiting, dehydration, and death. Diagnosis is by virus isolation, fluorescent antibody test, and serology. There is no effective treatment.

Classical swine fever

For a full discussion of this condition, see Chapter 11 (*Polysystemic diseases*)

Nervous signs – which include incoordination, circling, muscle tremors, and convulsions – are sometimes seen quite early on, along with other signs, in outbreaks of this disease. Pyrexia with temperature of 40–42°C is usually present, as are a wide spectrum of other signs. The disease is **notifiable** in the UK and Europe, where it is controlled by a slaughter policy.

Aujeszky's disease (pseudorabies)

Incidence

This disease is common in the USA and Europe, where it is endemic in some areas. It also occurs in South America, Asia, and New Zealand. The incidence is increasing in some areas. It has been eradicated from the UK mainland but is present in Northern Ireland.

Aetiology

The cause is *porcine herpes virus*. It can cause disease in pigs, sheep, cattle, and horses.

Epidemiology

The virus is capable of surviving away from pigs for up to 7 weeks. It may enter a herd through a carrier animal, which is infected for life. It is also brought in on vehicles, by visitors, and by rodents. Once in a herd, the disease spreads rapidly between pigs. Not all infected animals show clinical signs. Strains of the virus vary in their virulence. Wild boar may act as a reservoir of infection.

Clinical signs

These vary with the age of animals. In baby piglets <4 weeks old, the clinician may initially find vomiting and diarrhoea and a temperature of 41°C. CNS signs develop: incoordination, convulsions, recumbency, and death. Cold sore-like lesions may be present on the mouth, snout, and face.

In older pigs, anorexia and then respiratory signs may predominate. Temperature is 41°C. Coughing and cold-like signs are present. CNS signs may not be seen in this age group, but some animals show incoordination and hind limb paresis.

Sows and gilts may have reproductive failure, with abortion, early embryonic death, and a vaginal discharge.

Diagnosis

There is a wide range of clinical signs. Use serology for a specific virus-neutralizing antibody. An ELISA test is also available. The virus is found in tissues. With histological examination, inclusion bodies in the CNS and signs of a viral encephalomyelitis are found. ELISA kits are used to detect the virus on fluid from frozen then thawed meat in abattoirs.

Differential diagnosis

- *Teschen disease and Talfan disease* can be quite similar. Serology is used to diagnose.
- *Rabies* is rare in pigs. One may see pruritus at the site of a bite.
- *Streptococcus suis* type 1 infection affects young pigs only. It responds to antibiotic therapy.
- *Classical swine fever*. Use virus isolation and serology. There is a wider spectrum of signs.
- *Bowel oedema* occurs in weaners only. Eyelid and laryngeal oedema are found.
- *Salt poisoning*. Intermittent CNS signs are seen.
- *Reproductive and respiratory problems*. There is a wide range of specific diseases.

Treatment

None is really effective. The prognosis is poor for young piglets. Recovery is possible in adults, but they may become carriers.

Control

A slaughter policy has been used to eradicate the disease in some countries, including the UK mainland and some US states. Commercial live vaccines are available in Northern Ireland. The disease is **notifiable** in the UK.

Rabies

Incidence

Rabies is not found in the UK (currently). It is uncommon in pigs elsewhere.

Aetiology
A virus from the *Lissavirus* genus of the Rhabdoviridae family is responsible. Several genotypes have been recognized.

Epidemiology
The virus is transmitted by the bite of a rabid animal. Pigs have been bitten by rabid cats in Europe and by skunks in the USA. The incubation period can be >50 days. Death always ensues.

Clinical signs
These are quite variable in pigs. Twitching of the nose may be seen, and there may be pruritus at the site of the bite wound. Excitement, aggression, convulsions, and paralysis follow. The pig may salivate excessively and make frequent chewing movements. Death usually occurs within 48 h of signs first appearing.

Diagnosis

This is based on fluorescent antibody test on brain tissues, dot ELISA test, and Negri bodies in the brain.

Differential diagnosis

Teschen, Talfan, Aujeszky's, and Glasser's diseases are the differential diagnoses for rabies.

Treatment

Treatment is not attempted.

Control

Rabies is a **notifiable** disease in the UK. Vaccination is used in other countries. Import controls may help avoid introduction of the disease.

Louping ill

Incidence
Louping ill is rare in pigs and occurs only where ticks (*Ixodes ricinus*) are present. There may be an increased incidence with the increase in outdoor pig units.

Aetiology
A *flavivirus* is responsible.

Epidemiology
The virus is spread by the *I. ricinus* tick.

Clinical signs
These are pyrexia and neurological dysfunction (muscular tremors, incoordination, and a bounding gait). Later, recumbency, coma, and death occur.

Diagnosis

This is based on a history of tick exposure and clinical signs. Virus isolation and serology are used.

Treatment

There is none.

Control

Tick control is used – avoid exposure. Inactivated vaccine is used successfully in ruminants. It might be considered in pigs if there is a high incidence of louping ill.

Congenital diseases

A group of *at least six diseases* affecting neonatal piglets have been associated with congenital tremor. The diseases cause hypomyelinogenesis and muscular tremor. Affected piglets have increased sensitivity of their spinal cord reflexes. This may prevent piglets gaining access to the sow's teat, making them more vulnerable to maternal physical injury and to the increased risk of hypoglycaemia.

The piglets may appear normal at birth, with signs of tremor developing when they are 3 days old. In some cases, the tremor reduces as the piglet gets older. The speed and severity of tremor vary. Tremor may be seen when the animal walks but not when it is recumbent. Muscle strength is normal in some cases.

Some cases of congenital tremor are caused by infectious agents, and some through hereditary abnormalities. One form of the disease (type A1) is caused by the *classical swine fever virus*, and this must be eliminated from the diagnosis of tremor in every case. Swine fever is a **notifiable** disease and must be reported to Defra.

The main features of each type are listed below.

Congenital tremor type A1

Incidence
This was responsible for 12% of cases of congenital tremor when classical swine fever was endemic in the UK in the 1960s.

Aetiology and epidemiology
Classical swine fever virus affects sows at 10–50 days' gestation.

Clinical signs
Male and female piglets are equally affected. Several litters of any breed are affected. There is high mortality in affected piglets.

Diagnosis

Isolate classical swine fever virus from affected piglets. Test seroconversion in sows.

Post-mortem – Cerebellar hypoplasia and demyelination of the spinal cord at the C3 level are found.

Congenital tremor types A2–5 and type B

Congenital tremor type A2

Incidence
Type A2 is reported throughout the pig-keeping world.

Aetiology
The causal organism is thought to be a *retrovirus*.

Clinical signs
Male and female piglets are equally affected. Several litters are affected, often over a 4-month period. A high percentage of piglets are affected in litters, but most survive. Piglets with tremor are not produced by remating the same sow and boar.

Diagnosis

Diagnose on the basis of absence of classical swine fever virus or seroconversion. No bovine viral diarrhoea antibodies are found.

Post-mortem – Cerebellar hypoplasia and hypomyelinogenesis are present.

Note: types A1 and A2 are sometimes both known as *myoclonia congenita*.

Congenital tremor type A3

Incidence
Type A3 is seen in male Landrace piglets only.

Aetiology
A sex-linked recessive gene is responsible.

Congenital tremor type A4

Incidence
Type A4 is seen only in British Saddlebacks.

Aetiology
It is caused by a non-sex-linked recessive gene. Demyelination occurs through a defect in the piglet's lipid metabolism.

Congenital tremor type A5

Incidence
This is uncommon.

Aetiology
It is caused by exposure of the sow to organophosphorus products during pregnancy.

Congenital tremor type B
This is a group of conditions not in the above categories and with inconsistent pathological lesions.

Congenital meningoencephalocele

This is a rare autosomal recessive defect in which the cranium is not fully closed at the time of birth. The meninges and occasionally the cerebral hemispheres protrude between the cranial bones. The condition has been reported in Large White and Landrace pigs. Euthanasia is used in affected piglets. Eliminate the problem by avoiding repeated breeding of affected litter members and by identifying and culling carrier animals. Other congenital defects that are not compatible with life include hydrocephalus (Fig. 7.1) and spina bifida (Fig. 7.2).

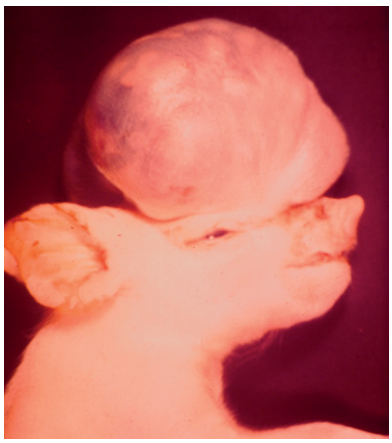

Fig 7.1 – Piglet congenital defect: hydrocephalus.

Fig 7.2 – Piglet congenital defect: spina bifida.

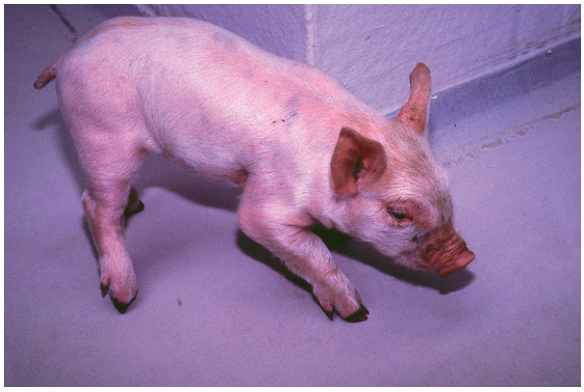

Fig 7.3 – Piglet tetanus.

Bacterial diseases

Tetanus

Incidence

The disease may occur at any time, especially on outdoor units. The incidence has been lower since routine castration of male pigs is no longer practised.

Aetiology

Clostridium tetani producing the exotoxin tetanospasmin causes tetanus. Spores persist in the environment and soil for many years.

Epidemiology

The disease mostly follows a wound accidentally or purposefully inflicted. It may follow tail docking and other procedures such as tooth clipping where hygiene is poor and exposure to tetanus spores possible. Toxin is produced and travels to CNS axons via motor nerve endplates. The exact mode by which pathological effects are produced is unknown. The period of incubation of the disease is very variable, at 3 days to 4 months.

Clinical signs

There is progressive onset of muscle rigidity and spasm. The pig walks with a stiff, tiptoeing gait. The tail is stiff, and ears are held upright and are rigid to the touch (Fig. 7.3). The third eyelid may protrude if the pig is startled. Trismus (lockjaw) prevents feeding, and finally convulsions and almost constant tetanic spasms occur. The pig may become recumbent and unable to stand, as its legs are too stiff to permit flexion before rising is possible (Fig. 7.4). The original entry wound is not always evident.

Diagnosis

Base the diagnosis on history and progressive clinical signs.

Fig 7.4 – Pig tetanus: recumbent animals showing pronounced extensor tone.

Differential diagnosis

- *Meningitis*. Specific signs include nystagmus.
- *Strychnine poisoning* should not occur now, but presentation is more acute than with tetanus and death may occur quite rapidly.

Treatment

If a possible entry wound is found, it should be cleaned and local antibiotic (penicillin) applied. Parenteral antibiotic, tetanus antitoxin, and sedation with acepromazine should also be given. The prognosis is generally poor; treatment is costly and welfare is compromised. Unless there is a rapid response of a mild case to therapy, euthanasia may be required.

Control

Avoid the risk of contamination of wounds. Problem farms may have to use tetanus antitoxin and possibly vaccination (e.g. after tail docking).

Botulism

Incidence

Botulism is rare in pigs, but the risk may increase with more outdoor systems and possible exposure to the toxin.

Aetiology

Clostridium botulinum is the causal organism. Spores persist in the environment for many years. BCD strains of the organism are important in farm animal disease.

Epidemiology

A neurotoxin is produced by the organism, which may be found in warm, decaying vegetable or animal matter. Pigs eating this material may be exposed to infection.

Clinical signs

These are muscle tremors, weakness, and paralysis leading to recumbency and loss of control of the tongue. Animals are unable to eat or drink, and respiratory movement failure may result in death. Several animals in a group may be involved.

Diagnosis

The clinical signs and possible exposure to the organism may raise suspicions of the disease. The toxin may be identified in the blood by an ELISA test.

Differential diagnosis

Other causes of paralysis include Teschen and Talfan diseases.

Control

Avoid exposure to contaminated decaying material.

Streptococcus suis infection

There are at least 20 strains of *S. suis*. Types 1, 2, and 14 (see below) are the most important in terms of porcine clinical disease.

Streptococcus suis type 1 infection

Incidence

Type 1 is a common and important disease. In the 1996–2003 Veterinary Investigation Diagnosis Analysis, 88% of neurological cases were meningitis. 70.5% were cases of streptococcal meningitis.

Aetiology

Streptococcus suis type 1 is the causal organism.

Epidemiology

The organism is carried in the tonsils of carrier sows and enters the piglet's body through the tonsilar crypts. It multiplies and passes into the bodies of susceptible animals, causing a septicaemia. In some cases, the body defences prevent further development. In other animals, the organism multiplies in the tonsils before being carried as a bacteraemia through the bloodstream to various parts of the body, including the meninges, the joints, or the heart. More than one location may be involved in the disease process.

Clinical signs

The infection mostly affects piglets aged 10–14 days. Affected animals are dull and pyrexic (temperature 40–41°C), and up to 66% of the litter may show some signs of infection. One may see sudden death in one or more piglets. Joint involvement occurs: piglets are reluctant to come to feed and are very lame if they try to walk. Joints, especially hock and carpus, are swollen, warm, and painful. Meningitis is shown by piglets that are recumbent with convulsions, opisthotonus, and unconsciousness (Fig. 7.5). Pronounced lateral nystagmus is often present. Traumatic corneal damage may affect the lower eye if the animal is convulsing in a recumbent position. The heart is less commonly affected; the clinician may see poor exercise tolerance, cardiac murmur, and sudden death. Ultrasonographic evaluation of the heart, using a standard B-mode instrument, is possible. One may see clear evidence of vegetative endocarditis.

Diagnosis

Base diagnosis on clinical signs, age and number of piglets involved, possible spectrum of signs, and pyrexia. Post-mortem examination and culture of the organism are useful.

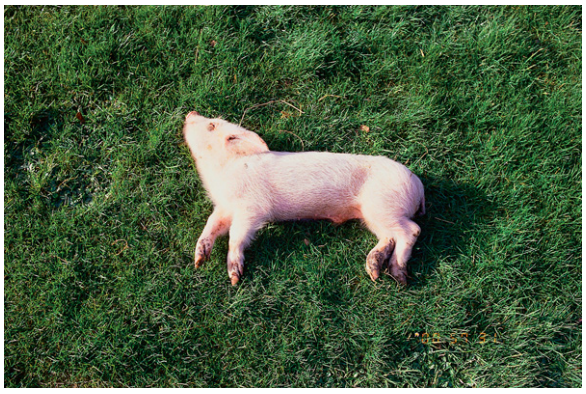

Fig 7.5 – Piglet infected with *Streptococcus suis* type 1.

Differential diagnosis

- *Glasser's disease* (*H. parasuis*) usually occurs in slightly older piglets, and one may see peritoneal and pleural infection.
- *Mycoplasma hyorhinis* may cause serositis and arthritis in young piglets. There is possibly less fever than with *S. suis* infection.

One may need a culture from post-mortem to confirm which organism is involved.

Treatment

Immediate treatment with parenteral antibiotic is needed: penicillin, ampicillin, or trimethoprim-sulpha. A 3- to 5-day course of treatment is essential. Steroids or non-steroidal anti-inflammatory drugs (NSAIDs) may also be used to reduce inflammatory changes. Joint drainage and lavage are not usually undertaken in small piglets but might help recovery, with minimal permanent damage. The prognosis for joint resolution is quite good if the pig is seen at an early stage. The prognosis for meningitis cases is very guarded, and euthanasia may be required. Unconscious piglets rapidly become dehydrated, and fluids may be given with care orally, intraperitoneally, or per rectum.

Control

Improve general hygiene on farm and farrowing accommodation. Check floors of farrowing pens – sharp or roughened areas may cause abrasions, leading to entry of causal organisms. Routine antibiotic injection (e.g. long-acting penicillin) given to sows just before farrowing or to piglets at birth may help reduce the incidence of disease. No vaccines are currently available in the UK.

Streptococcus suis type 2 and type 14 infection

Incidence

This is a common and important disease. Human infection may occur by zoonotic spread and enters the human body via abrasions or via the oropharynx, causing septicaemia and meningitis.

Aetiology

Streptococcus suis type 2 and type 14 are responsible. The organisms can survive in the environment for several days and can live in faeces for 10 days at a temperature of 9°C. The organism is readily destroyed by all common approved farm disinfectants.

Epidemiology

The condition often occurs 3–7 days following stresses such as moving and mixing groups of pigs. The organism may be brought in by carrier animals and settles in tonsilar crypts of in-contact pigs. The original infection may have been passed from the sow to her piglets. *S. suis* type 2 has a polysaccharide capsule that protects it from phagocytosis and allows it to spread rapidly through the body. Infection by the same strain may persist on a farm for years.

Clinical signs

This infection mostly affects the weaners to the finisher age group (usually older pigs than are infected with *S. suis* type 1). The first sign of the disease in a group may be a sudden death. Other pigs become anorexic and pyrexic (temperature 40–41°C). Some are said to have a 'glassy stare' when viewed from in front. Some may show signs of acute arthritis, with warm, swollen, and painful joints. Others show signs of meningitis, rapidly becoming recumbent, convulsing, and showing nystagmus.

Diagnosis

Diagnosis is based on the history of the farm and pigs, clinical signs, and culture of the organism from post-mortem material.

Differential diagnosis

- *Glasser's disease* (*H. parasuis*). Generalized serositis may be seen.
- *Mycoplasma hyorhinis* and (less commonly) *M. hyosynoviae* may cause polyserositis and arthritis in young pigs; there is less pyrexia than with *S. suis*.

Culture may be needed to identify the organism present. An ELISA on the serum of recovered pigs may help to identify and differentiate the strain of *S. suis* involved.

- *Erysipelothrix rhusiopathiae* can cause arthritis, but there is less joint swelling and it is a more chronic condition.

Treatment

Antibiotic therapy including penicillin, ampicillin, or trimethoprim-sulpha is effective. NSAIDs may also help reduce inflammation and relieve pain. A 5-day course of antibiotic therapy should always be given to avoid chronic arthritis. The prognosis for arthritic cases is good if treatment is started early. Prognosis is guarded for meningitis cases. Nursing care comprises food and encouragement to exercise.

Control

- *Strategic medication*. Administer antibiotic at the time infection is anticipated. One can give, for example, long-acting penicillin injection just before weaning or mixing. Soluble oxytetracycline may be given in the drinking water for 7 days over an identified danger period.
- *Husbandry improvement*: an all in, all out system with good disinfection and cleaning between batches. Rest buildings. Multiple site systems can be used to remove weaners from the breeding herd.
- *Eradication* of *S. suis* has been achieved by depopulation followed by stocking with hysterectomy-derived stock. A closed herd and high hygiene standards must then be kept.
- *Vaccination*. No commercial vaccine is currently available.

Glasser's disease

The disease is discussed in greater detail in Chapter 3 (*Diseases of the musculoskeletal system*) and Chapter 4 (*Diseases of the respiratory system*). Its effects on the nervous system only are discussed here.

Incidence

Glasser's disease is quite common and occurs worldwide. It is seen chiefly in piglets from weaning to 4 months of age.

Aetiology

Haemophilus parasuis is the causal organism.

Epidemiology

The causal organism is found in many pigs. Outbreaks of disease may follow stresses of various kinds, including other diseases.

Clinical signs

The disease is a polyserositis causing inflammation in various serosal surfaces. Several pigs in a group are often affected at the same time. CNS signs include meningitis, coma, and death.

Diagnosis

This is based on the history, clinical signs, and isolation of the causal organism.

Post-mortem – Signs include a polyserositis and a fibrinous meningitis.

Differential diagnosis

Other possibilities are infectious diseases affecting the nervous system, including *S. suis* and mycoplasmal diseases.

Treatment

Antibiotic injections for 5 days, based on sensitivity tests, should be given to sick pigs. In-contact and at-risk animals should be given medication in the drinking water. Euthanasia may be required in animals with severe and non-responsive CNS signs.

Control

An inactivated vaccine is available.

Middle ear disease

Incidence

This disease is common and is frequently seen in groups of growing or finishing pigs, where one or (less commonly) several pigs may be affected.

Aetiology

It is caused by an ascending infection from the pharyngeal region or tonsil. *Arcanobacter pyogenes*, streptococci, and other opportunist pathogens are usually involved.

Epidemiology

The infection probably originates in the pharynx or tonsil and gains access to the middle ear by ascending the Eustachian tube. Haematogenous spread may occur. In some cases, infection arises from the external ear. Infection may spread from the middle ear to the inner ear.

Clinical signs

In many cases, the pig is just seen with a unilateral head tilt and with no other signs (Fig. 7.6). Occasionally, middle

Fig 7.6 – Pig middle ear disease.

ear presents as an acute infection. The affected pig is pyrexic and anorexic, with some degree of imbalance. If vestibular damage is severe, the pig may be unable to stand and shows compulsive rolling towards the affected side.

Diagnosis

This is based on clinical signs.

Differential diagnosis

Aural haematomata are common in pigs and develop as a result of injury, often inflicted by head shaking in pigs infested with sarcoptic mange. A large haematoma will cause a mechanical head tilt that may resemble middle ear disease. Careful clinical examination should differentiate the two conditions.

Treatment

Antibiotic treatment is given parenterally. Penicillin-streptomycin combinations or other broad-spectrum antibiotics are effective. A 3- to 5-day course should be given. In acute infections with severe vestibular compromise, NSAIDs may also be used. Treatment rarely corrects the head tilt but allows the pig to grow and finish without any worsening of the signs.

Metabolic disease

Hypoglycaemia in neonatal piglets

Incidence

This is very common and a major cause of piglet mortality.

Aetiology

The cause is low blood glucose, usually the result of inadequate milk intake (see below).

Epidemiology

Piglets are born with little body fat, no brown fat, and minimal glycogen reserves in the liver. If they do not drink milk they soon become hypoglycaemic, and the early signs of this disease can make them less competitive in finding a teat and taking colostrum. Hypothermia exacerbates the problem by reducing the ability of piglets to suckle their mothers. Hypoglycaemia may predispose to hypothermia. The risk is greatest in the first 3 days of life. Mortality if the condition is untreated can be 100%. The weakened hypoglycaemic piglet has a greater chance of being crushed by the sow. Enteritis, which is very common at this stage of life, reduces the efficiency of glucose absorption from the gut and thus may predispose the animal to hypoglycaemia.

Clinical signs

These are general weakness, incoordination, loss of balance, falling over, and walking with a wide-stance hind limb gait. Low temperature, shivering, pallor, and hairs of the coat standing on end are present. Affected piglets have a weak reedy squeal. The clinician may also see galloping movements, champing of the jaws, fitting, and opisthotonus. Terminally, recumbency, convulsions, and death occur.

Diagnosis

Diagnosis is based on clinical signs and a blood glucose level of <2.8 mmol/L.

Post-mortem – Sometimes there are no specific gross lesions but crushing injuries may incidentally be present.

Differential diagnosis

- *Congenital tremor.* The piglets are usually strong.
- *Hypothermia*. The piglets are cold and depressed but may also be developing hypoglycaemia.

Treatment

Treatment is with 15 mL of 20% glucose solution given intraperitoneally and repeated every 4–6 h until the piglet can drink by itself. Warmth and good nursing care are essential, watching for development of enteritis etc.

Control

Prevent hypoglycaemia by very careful management in the neonatal period to ensure that each piglet sucks and obtains colostrum. Monitor the sow and litter for any signs of milk shortage. Provide heat in the creep area to avoid hypothermia.

Neurological signs caused by injury or degenerative disease

These are quite common. A detailed clinical examination and neurological examination (see above) are required to diagnose the cause. Posterior paralysis is quite common

and may result from a pathological fracture in the body of a spinal vertebra. Similar clinical signs also arise as a result of tail-biting injuries. Infection from the tail wound is carried haematogenously to a vertebral body, mostly thoracic or lumbar, leading to collapse of the vertebra and compression of the spinal cord. Antibiotic therapy and NSAIDs may alleviate clinical signs if active inflammation is present, but in many cases euthanasia may be required. Other limb neurological injuries can result from fighting or other trauma. Sciatic nerve damage can result from careless placement of injections into the gluteal region. Thiamine and steroid injections may help recovery, and a slow improvement may be anticipated unless permanent nerve damage has been sustained.

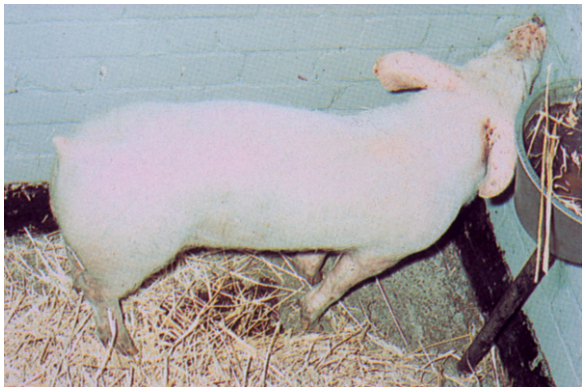

Fig 7.7 – Pig water deprivation ('salt poisoning').

Water deprivation: 'salt poisoning'

Incidence

The condition is quite common.

Aetiology

It is caused by either excessive salt in the diet or rapid rehydration following water deprivation.

Epidemiology

Excessive dietary salt or salt in the water is rare in the UK. In some countries, artesian water supplies may have excessive salt levels, which can put pigs and other livestock at risk. In most cases, the condition is caused by a lack of water for 12 h or more – usually the result of a plumbing fault. Water is restored and the pigs drink excessively to quench their thirst. The farmer may be unaware that they have been short of water, and by giving water ad lib exacerbates the problem. Younger pigs seem more susceptible than older animals. When water is available it enters the brain, causing cerebral oedema and a raised intracranial pressure.

Clinical signs

Very large doses of salt may cause severe gastroenteritis, but CNS signs predominate in cases of water deprivation. Affected pigs appear to be blind, deaf, and non-responsive to other stimuli. They may appear to search for water and possibly drink drainage water contaminated with urine. A spectrum of clinical signs may be seen in an affected group. There may be pruritus and head pressing. Periodic convulsions occur at regular intervals of approximately 3–7 min. The pig adopts a dog sitting position or lies in sternal recumbency. Nodding and champing movements of the head occur, and the pig raises its front end and may fall backwards or to one side as the convulsion ceases. Head pressing may also be seen (Fig. 7.7). This type of convulsion is not seen in any other CNS disease of pigs.

Diagnosis

Diagnosis is based on clinical signs, history of water deprivation, and high NaCl levels in the diet. Blood sodium may be elevated to 170–210 mmol/L (normal blood sodium 135–145 mmol/L). Haematological examination may show an eosinopenia.

Post-mortem – Eosinophilic meningitis and possibly gastroenteritis are seen.

Treatment

Treatment is by slow rehydration, with small aliquots of water being made available or given orally by syringe. Access to ad lib water must not be given initially. In many cases, pigs have already been given ad lib water and may have cerebral oedema. Mannitol at 2 g/kg and 20 min later dexamethasone at 1 mg/kg, both intravenously, have been used in people with high intracranial pressure. CSF withdrawal by lumbar puncture may also help. In practice, the costs of treatment may be prohibitive. The worst-affected animals do not recover, and the prognosis for any pigs showing severe CNS signs must be guarded.

Control

Adequate water supplies and warning systems of water failure must be available. If water deprivation has occurred, reintroduce supplies slowly.

Vitamin deficiency

Pantothenic acid deficiency

Incidence
This is unusual but may occur if pigs are fed on high quantities of bakers' waste.

Aetiology
A deficiency of pantothenic acid may lead to demyelination of neurons.

Clinical signs
Nervous signs include abnormal hind limb action – the animal shows signs of hypermetria known as 'goose stepping'. As the animal walks, the hind legs are lifted (snatched) up and forward in an extended position. They are lowered hock first. Skin lesions also occur; see Chapter 6 (*Diseases of the skin*).

Diagnosis

Diagnose based on the characteristic gait and history of abnormal feeding.

Treatment

Give 10–12 g of calcium pantothenate per tonne of food.

Control
Give a calcium pantothenate supplement as under treatment if the diet is likely to be deficient.

Vitamin A deficiency

Incidence
Vitamin A deficiency is rare.

Aetiology
This condition occurs with a ration containing <6 million IU of vitamin A per tonne, or if the diet is very rich in unsaturated fatty acid. The heating of the food during the pelleting process can also destroy vitamin A.

Epidemiology
The diet must be deficient for a long period, 4 months or more, before signs of deficiency appear. Prolonged deficiency of vitamin A can lead to bony proliferation in the spinal column and cranium. This in turn causes a rise in CSF pressure and compression of nerves passing through foramina. Blindness occurs in some cases.

Clinical signs
Neurological signs include incoordination, head tilt, and hind limb paralysis. In a breeding herd, other anomalies may be seen, including microphthalmia, high stillbirth rate, and weak neonatal piglets.

Diagnosis

Diagnosis is based on history, food analysis including vitamin A content, clinical signs, and histology. CSF pressure may be high, and lumbar puncture may demonstrate this.

Differential diagnosis

Eliminate other causes of neurological disease.

Treatment

Supplement vitamin A by injection and in the diet. Resolution of the severe CNS signs may not occur.

Control
Ensure adequate vitamin A supplementation. Ensure an adequate amount of vitamin A in the diet.

Toxic causes

Selenium toxicity

Aetiology
Vitamin E and selenium injections are given for the treatment of mulberry heart, hepatosis dietetica, and myopathy. Toxicity can occur with overdosing. The toxic dose of selenium for pigs by injection is >1–2 mg/kg or >5 mg/kg of food.

Incidence
The condition is uncommon.

Epidemiology
There is a potential danger if excessive selenium supplementation is given, either by injection or in the diet. Some pastures have very high selenium content, which can be dangerous to pigs.

Clinical signs
A single injection overdose (e.g. when treating mulberry heart) can lead to sudden death. Chronic toxicity leads to incoordination, mild ataxia, paraplegia, and

quadriplegia. Histopathology shows evidence of severe spinal cord damage with poliomyelomalacia. Alopocia and hoof separation have also been reported.

Diagnosis

This is based on clinical signs, histology, and high levels of selenium in tissues.

Treatment

No specific treatment is effective. Withdraw the source of selenium and slow recovery may occur. Recumbent pigs should be euthanized.

Aflatoxicosis

This is an unusual condition caused by toxins of *Aspergillus* spp. They cause fatty degeneration and necrosis of liver. Hepatic encephalopathy causes ataxia, jaundice, convulsions, coma, and death.

Diseases of the cardiovascular, haemopoietic, and lymphatic systems

Introduction

Specific diagnoses of cardiovascular diseases, haemopoietic diseases, and diseases of the lymphatic system are relatively uncommon in pigs. All three systems are vital to the effective function of most other body systems.

The pig is not an athletic animal, and its heart is relatively small. Heart failure can occur at times of stress such as moving, mixing, weighing, and fighting. The myometrium is also very sensitive to oxygen deficiency. This may lead to sudden death in cases of severe lung damage caused by respiratory disease.

Aetiology of cardiovascular diseases

Conditions within this group are caused by a wide range of agents and pathological processes. Hereditary cardiac anomalies are relatively uncommon in pigs. Ventricular and atrial septal defects have been described and are mostly incompatible with life. A number of infectious agents may cause cardiac disease. Examples include *Actinobacillus* and *Haemophilus parasuis* (Glasser's disease), both of which may cause pericarditis. Excessive pericardial fluid may accumulate, and later pericardial thickening may restrict cardiac movement and function. Vegetative endocarditis, which chiefly involves the tricuspid valve of the heart, may be present in chronic cases of swine erysipelas. Other agents, including streptococci, less commonly cause similar lesions with resultant valvular incompetence and cardiac failure.

A number of vitamin deficiencies are responsible for cardiac disease (e.g. mulberry heart disease, in which selenium deficiency may also be involved) or failure of the blood-clotting mechanism (warfarin poisoning, vitamin K deficiency). Blood-clotting failure can also occur in cases of autoimmune thrombocytopenia purpura in the newborn and also in Von Willebrand's disease.

Iron deficiency is associated with severe anaemia in piglets. Red cells can be destroyed by autoimmune-mediated haemolysis and also by the rickettsial parasite *Eperythrozoon suis*.

The oxygen-carrying capability of the blood can be compromised by a number of agents that damage the haemoglobin molecule. These include monensin poisoning converting haemoglobin into myoglobin. Methaemoglobin is formed in nitrite or nitrate poisoning and carboxyhaemoglobin in CO poisoning.

The lymphatic system may be compromised in a number of ways. Infectious agents such as *Bacillus anthracis* may infect the lymph nodes, resulting in reduced circulation of lymph and tissue swelling. Lymph node enlargement occurs in a number of conditions, including postweaning multisystemic wasting syndrome (PMWS). Injury producing tissue damage and swelling can also compromise the flow of lymph. Hereditary lymphosarcoma is relatively uncommon but causes swelling and dysfunction of some or all of the lymphatic system.

Epidemiology of cardiovascular diseases

In the case of infectious diseases, infection enters the body and may affect only the cardiovascular system or a number of body systems. Severe cardiovascular disease will result in the rapid or slow compromise of a number of vital body systems. It may eventually result in the death of the affected animal.

Cardiovascular disease may be primary or secondary. Primary disease directly affects the heart, while secondary disease occurs as a result of this (e.g. in renal or hepatic failure).

The failure to include vital dietary ingredients such as selenium may occur through carelessness or an attempt to save money. Exposure of pigs to toxic doses of monensin or warfarin can occur in a number of ways, all of which may have devastating results.

Clinical signs of cardiovascular diseases

These will again depend on the individual condition. In the case of acute conditions such as mulberry heart, cardiac failure is rapid and death may ensue within hours. In right-sided heart failure, venous return to the heart is compromised. In left-sided failure, the pulmonary circulation is compromised. In severe cases of cardiac disease, one form of heart failure rapidly leads to failure of the whole circulatory system.

In more chronic conditions such as endocarditis, the onset of cardiac failure is insidious. Exercise intolerance can be an early sign. Cyanotic discoloration of the skin and visible mucosae are seen in some cases. It may be possible to detect a cardiac murmur over the tricuspid valve in advanced cases. The difficulty of detailed cardiac

auscultation in the pig may make this impossible in early cases. In some cases, pronounced tachycardia may be found. Ultrasonography or radiology can be used to investigate cardiac function, but the expense may not be justified in many cases. It may be possible to detect the presence of pericardial fluid by auscultation. Evidence of consequential problems may include poor urine output (which may be difficult to detect under farm conditions) and in terminal cases evidence of diffuse intravascular coagulation.

Diseases of the haemopoietic system may result in failure of blood clotting, with profuse haemorrhage occurring after simple procedures such as intramuscular injections. In thrombocytopenia, numerous petechial haemorrhages are seen in the mucous membranes and skin. Pallor of the mucous membranes and of the skin is an early sign of anaemia. In more advanced cases, poor exercise tolerance and pica may be seen.

The carcass lymph nodes of the pig are not normally palpable. In cases of multicentric lymphosarcoma, the nodes are readily palpable and visible throughout the body. Multicentric enlargement of the lymph nodes is also seen in PMWS and in porcine dermatitis and nephropathy syndrome.

Diagnosis of cardiovascular diseases

This is based on careful clinical examination and a knowledge of the physiology and pathology of the system.

Special diagnostic tests include the use of ultrasonography to evaluate the heart. Haematological studies will enable the red and white cell counts to be determined. Clotting and bleeding times will help identify the presence of clotting defects. The presence of haemolysis and abnormal haemoglobin may be detected in the laboratory. Post-mortem examination including histopathology is an important aid to diagnosis. For example, the gross appearance of the heart and its histopathology are very distinct in cases of mulberry heart disease.

Treatment of cardiovascular diseases

The heart has considerable powers of compensation, and the gross signs of cardiac disease may not be apparent until the pig is in severe heart failure. In such cases, treatment is ineffective and euthanasia should be considered if the pig is terminally ill. In some diseases of the blood, treatment is effective, especially if diagnosis and therapy can be commenced early in the course of the disease. For example, *Eperythrozoon suis* is responsive to oxytetracycline therapy, and some cases of thrombocytopenia purpura can be successfully treated with prednisolone.

Control of cardiovascular diseases

This varies with the cause. Some examples are as follow.

- Vaccination against erysipelas is advisable in all herds, and vaccination is also available to reduce the incidence of Glasser's disease.
- Careful feeding and the safe disposal of toxic material should avoid pigs gaining access to excessive amounts of such materials.
- The risk of CO poisoning can be reduced by avoiding the use of gas heaters and providing good ventilation.

Veterinary Investigation Diagnosis Analysis sample submissions: diseases of the cardiovascular system, 1996–2003

See Table 8.1.

Diseases of the cardiovascular system

Actinobacillosis

This excludes *Actinobacillus pleuropneumoniae*, which causes pleuropneumonia of pigs and is discussed in Chapter 4 (*Diseases of the respiratory system*).

Incidence

Actinobacillosis occurs in the UK but is uncommon. It mostly occurs in pigs <3 months of age.

Aetiology

Actinobacillus suis or less commonly *A. equuli* is the cause.

Epidemiology

The organisms may be opportunist pathogens that gain access to the pig via the respiratory tract. Their mode of sudden entry into otherwise minimal disease herds is not known. *A. equuli* is normally an equine pathogen and may reach pigs through contact with horses.

Clinical signs

A spectrum of signs are seen. Some sudden deaths in piglets with signs of septicaemia occur (Fig. 8.1). Other pigs show a more chronic course of disease. They are

Table 8.1 Veterinary Investigation Diagnosis Analysis sample submissions: diseases of the cardiovascular system, 1996–2003

Condition	Incidence (%)
Mulberry heart disease	53.55
Others	18.03
Thrombocytopenia purpura	9.56
Lymphosarcoma	8.47
No diagnosis	8.20

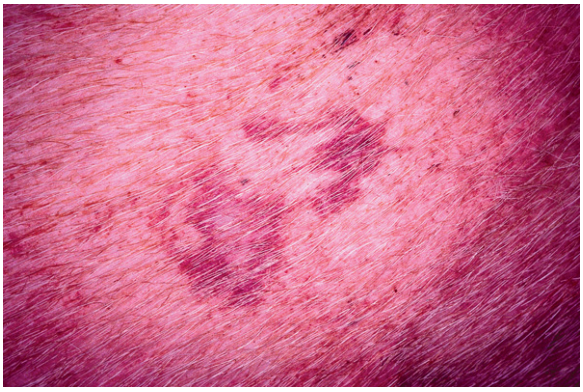

Fig 8.1 – *Actinobacillus equuli* infection: petechial skin haemorrhages that may resemble those caused by thrombocytopenic purpura (see Figs 8.13 and 8.14). (Courtesy of W.D. Strachan.)

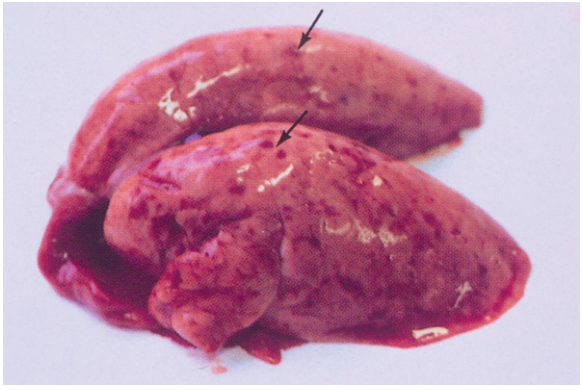

Fig 8.2 – Lungs of piglet with septicaemia caused by *Actinobacillus suis*. Note the small areas of consolidation in the lungs, which may resemble those caused by *A. pleuropneumoniae*.

listless and have elevated temperature and petechial haemorrhages on the skin of the ears and abdomen. Older pigs may have skin abscesses caused by actinobacilli. Some pigs show signs of polyarthritis and show pain and difficulty when attempting to walk. In other animals, signs of heart failure (including sudden death) may be seen, with lesions including endocarditis and pericardial effusions.

Diagnosis

To confirm, culture of the organism is essential.

Post-mortem – Post-mortem signs vary from those of septicaemia to those of polyarthritis, endocarditis, and pericardial effusion (Fig. 8.2).

Fig 8.3 – Sow with endocarditis: the animal was dull and had poor exercise tolerance.

Differential diagnosis

Other causes of heart disease and septicaemia, including Glasser's disease, could be responsible. Culture of the causal organism is helpful in confirming the diagnosis.

Treatment

The whole litter should be treated parenterally with one of the following antibiotics: penicillin, ampicillin, oxytetracycline, or enrofloxacin.

Glasser's disease

This is caused by *Haemophilus parasuis*. Polyserositis causes a variety of signs, including sudden death (Fig. 4.9), cyanosis, and heart failure – often as a result of pericarditis. For fuller discussion, see Chapter 4 (*Diseases of the respiratory system*).

Swine erysipelas

Endocarditis (Figs 8.3–8.5) is present in some cases of chronic *Erysipelothrix rhusiopathiae* infection. There is often no direct history of the pig having earlier signs of the acute disease. Pigs with endocarditis may have exercise intolerance, cyanosis (white pigs), cardiac murmur, and signs of heart failure including ascites. For full discussion, see the section on swine erysipelas in Chapter 11 (*Polysystemic diseases*).

Mulberry heart disease

Mulberry heart disease and hepatosis dietetica are both forms of vitamin E and selenium deficiency. Although

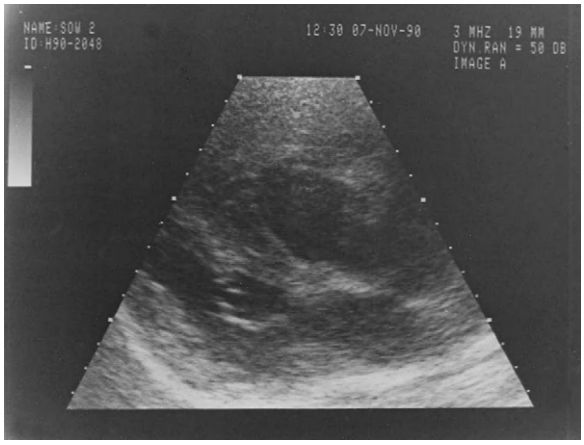

Fig 8.4 – Ultrasonographic scan of the heart from the sow in Fig 8.3. Irregular lesions are visible on an atrioventricular valve.

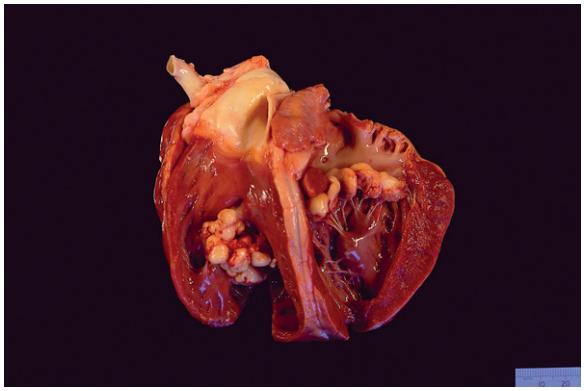

Fig 8.5 – Sow endocarditis: post-mortem appearance of the sow in Fig 8.3. Large vegetative lesions are present within the heart.

they seldom occur together, both conditions are described here. A further condition is muscular dystrophy, which is mentioned here but described in greater depth in Chapter 3 (*Diseases of the musculoskeletal system*).

Incidence

This disease is less common than it was some years ago, but the condition was responsible for 53.55% of the Veterinary Investigation Diagnosis Analysis (VIDA) cardiovascular cases diagnosed in 1996–2003. The condition occurs most commonly in fast-growing finisher pigs but can occur in weaners or adult pigs. Rations should now contain sufficient vitamin E and selenium, but the economic difficulties of the industry or an oversight may lead to the omission of these substances from a home mix. Cases can be sporadic or involve up to 25% of animals in a group. Mortality can be as high as 90% in affected animals.

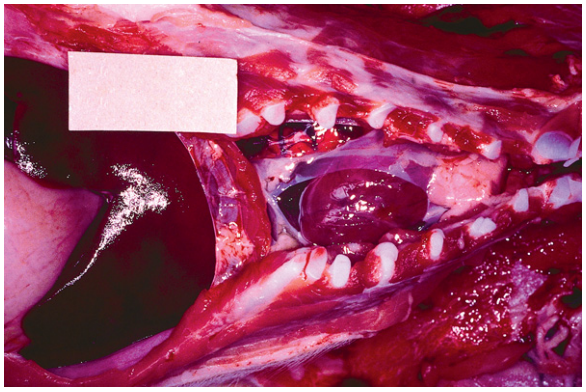

Fig 8.6 – Mulberry heart disease: gross appearance. (Courtesy of W.D. Strachan.)

Aetiology

The cause is a deficiency of vitamin E and/or selenium, which causes a serious lack of antioxidants in the body.

Epidemiology

Mulberry heart disease is mostly seen in fast-growing animals in good condition, and can occur if animals are fed barley treated with propionic acid. Diets high in barley, maize, and soya are especially likely to be low in vitamin E or selenium.

Clinical signs

An affected pig may be found dead and is often the best in its group. Living pigs may be dull, reluctant to move, and slightly dyspnoeic. The skin over the withers may be slightly cyanotic and cold to the touch. Temperature is normal or slightly depressed. Pulse is elevated and slow to recover after exercise. Any stress, such as weighing, handling, or movements, may lead to the sudden death of affected animals.

Diagnosis

Diagnosis is based on typical post-mortem findings and confirmation of low vitamin E and/or selenium in the diet.

Post-mortem – The pig is often in very good condition. Excessive amounts of fluid and fibrin tags are found in all body cavities including the pericardium. Pericardial fluid may be slightly gelatinous. Multiple haemorrhages are visible under the epicardium and the endocardium (Figs 8.6, 8.7 and 16.14). Lesions may be worse in the right side of the heart. Other signs of congestive heart failure may be seen, including enlargement of the liver, which has a nutmeg appearance. The lungs may be congested and oedematous.

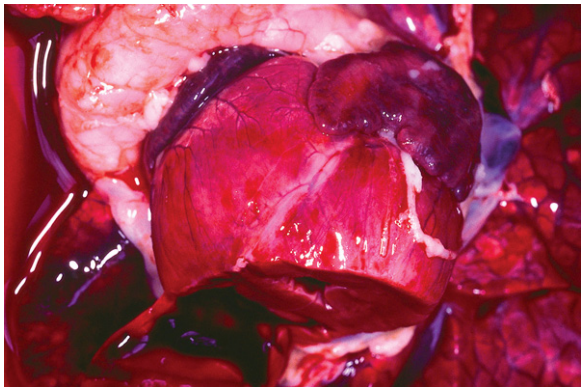

Fig 8.7 – Mulberry heart disease: external appearance of myometrium.

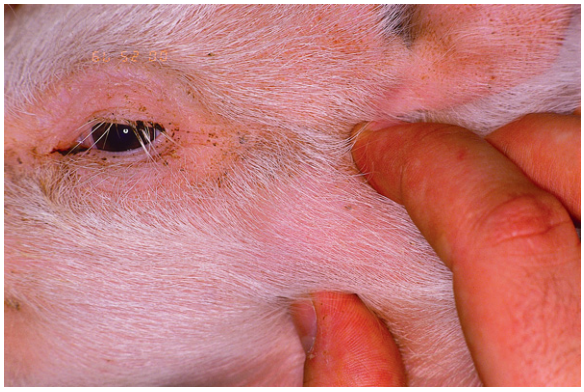

Fig 8.8 – Piglet lymphosarcoma: enlarged parotid lymph node.

Differential diagnosis

A post-mortem is necessary to confirm the absence of other conditions that may increase the amount of pericardial fluid, for example pericarditis, bowel oedema (clinical signs are distinct from those of mulberry heart), septicaemia (culture of organism), and torsion of the bowel (mesenteric torsion and gut necrosis obvious at post-mortem). Porcine stress syndrome and heat exhaustion are possible. For the final diagnosis, low levels of vitamin E or selenium are found in the liver and plasma.

Treatment

The whole group should be dosed orally with vitamin E and selenium. Animals at particular risk may be given parenteral treatment by injection, and others treated through a supplemented diet.

Control

Ensure adequate dietary levels of vitamin E and selenium and/or supplement individual pigs on problem farms every 3 months.

Hepatosis dietetica

This is seen chiefly in young growing pigs at 3–4 months of age. Affected animals are often found dead with liver lesions: pale or haemorrhagic lobes of the liver. Vomiting, depression, ataxia, and some jaundice may be seen in living animals. Confirmation of diagnosis is by post-mortem signs and histology of liver. Treatment and prevention are as for mulberry heart (above).

Muscular dystrophy

Muscular dystrophy may be present with mulberry heart and hepatosis dietetica but possibly not recognized in some cases other than by histological changes. Clinical signs are seen in growing breeding stock: swelling of upper limb muscles and of longissimus dorsi muscles, with walking difficult and painful. Diagnosis is by clinical signs and elevated muscle enzymes. Treatment is with vitamin E and selenium. Analgesia is given as required. (See also Chapter 3 – *Diseases of the musculoskeletal system.*)

Disease affecting the lymphatic system

Lymphosarcoma

Incidence

Cases are mainly sporadic, but the condition can occur as a herd problem in which heredity plays a part. In the VIDA survey, 8.47% cases were reported to be lymphosarcoma (see above). Lymphosarcoma can affect pigs from a few days to several months old. It is rare in adults.

Aetiology

The cause is unknown, but the condition may have a genetic predisposition. In one report, an autosomal recessive gene was thought to be responsible and affected animals could be bred for experimental purposes.

Clinical signs

These are mostly ill thrift and enlargement of some or most of the carcass lymph nodes (Figs 8.8 and 8.9). Mediastinal lymph node involvement is seen occasionally in

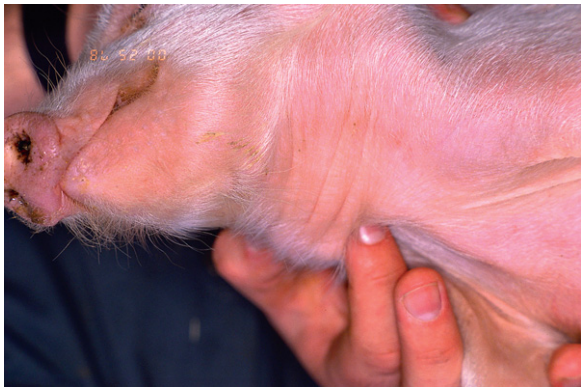

Fig 8.9 – Piglet lymphosarcoma: enlarged submandibular lymph node.

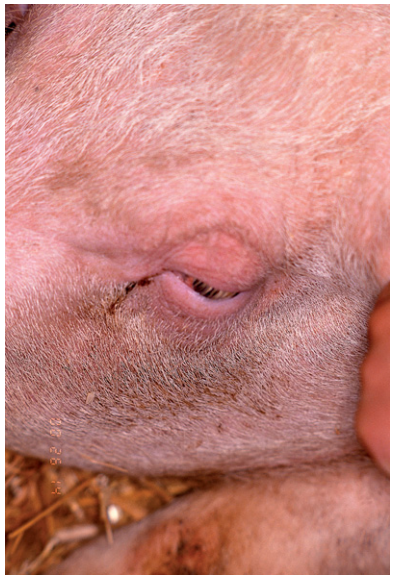

Fig 8.10 – Pig with lymphosarcoma showing signs of exophthalmus, which may be caused by enlargement of the mediastinal lymph nodes.

growing or even finishing pigs. Dyspnoea and poor exercise tolerance may be seen. An exophthalmic appearance is seen in some animals (Fig. 8.10). This may be due to poor lymphatic drainage from the head or to the presence of retrobulbar lymphoid tissue.

Diagnosis

This is based on clinical signs and histology of lymph nodes. Some cases show peripheral lymphocytosis.

Post-mortem – Examination may reveal extensive involvement of lymph nodes throughout the body (Fig. 8.11).

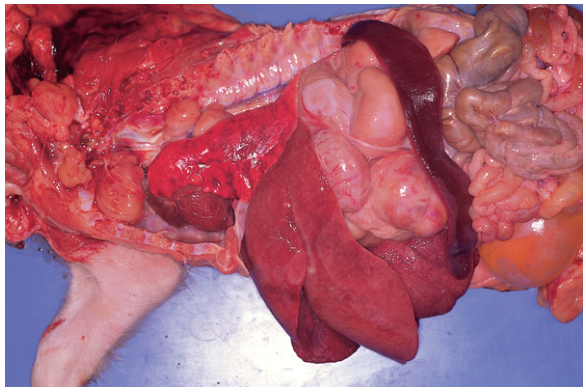

Fig 8.11 – Enlarged mediastinal lymph nodes in a piglet at post-mortem.

Treatment

None is effective. Steroid therapy may cause temporary resolution of signs. Euthanasia is required in most cases.

Control

Avoid breeding from affected or carrier animals.

Diseases affecting the haemopoietic system

Iron deficiency anaemia

Incidence

This should be a rare occurrence but cases are still seen, especially if iron injections are not given to litters kept indoors. One piglet or the whole litter may be missed.

Aetiology

Piglets are born with approximately 12 g of haemoglobin/100 mL of blood, which declines to 8 g/100 mL by 8 days of age. Piglets grow rapidly as active haemopoiesis occurs. Piglets require 15 mg of iron/day, but the sow can provide only 1 mg/day in her milk. Unless supplementary iron is injected or is available orally, anaemia will occur. Oral availability of iron, unless actually dosed into piglets, may be less reliable than iron injections, and the occasional piglet may not take its dose.

Epidemiology

Outdoor piglets should theoretically obtain iron from the soil, but most farms use supplements. For pigs kept

indoors, no source of iron is available and anaemia inevitably occurs if iron supplementation is not given. By 3–4 weeks of age, piglets should obtain an adequate supply of iron from their creep feed.

Clinical signs

These are mostly seen at about 3 weeks of age and over. Piglets are apparently growing well but are pale and may appear slightly yellow. The mucosae are very pale (Fig. 8.12). If the pen is entered, the piglets immediately try to eat any small portions of soil on the attendant's (or veterinarian's) boots. Severely affected pigs show exercise intolerance and dyspnoea, and the apex beat of the enlarged flabby heart may be visible through the thoracic wall. Pale diarrhoea may be seen. If stressed (e.g. handled to inject with iron), sudden death may occur. Pericarditis may be present in such cases.

Diagnosis

Base the diagnosis on a history of no iron injection and blood haemoglobin <7 g/100 mL. A hypochromic microcytic anaemia is present.

Post-mortem – Plump pale piglets are seen. Gross cardiac enlargement with thinning of the heart walls is present. The liver is also enlarged.

Differential diagnosis

Other causes of blood loss are possible: navel bleeding, isoimmunity causing haemolysis, thrombocytopenic purpura, and blood loss from tail removal or castration wounds (see below).

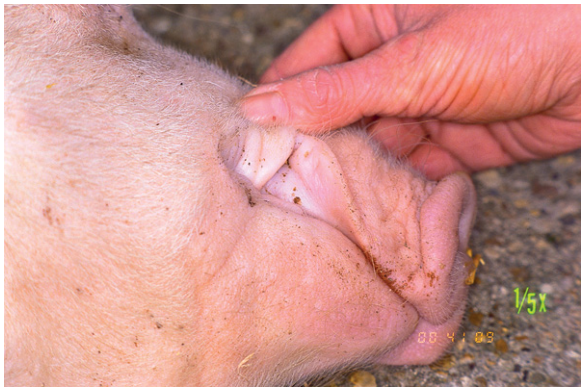

Fig 8.12 – Iron deficiency anaemia. Note the pallor of oral mucosa.

Treatment

Iron injection is used. The standard dose is 200 mg/piglet by intramuscular injection. The dose of iron should not be exceeded, as iron toxicity may occur. Oral iron – ferrous sulphate or iron dextran – can be used. Clumps of grass with earth attached are eaten voraciously if offered to iron-deficient anaemic piglets.

Control

Control is by routine use of iron supplementation. Poor-quality iron injections may cause sudden death, especially if vitamin E levels are low.

Haemolytic disease of the newborn

Note: this condition and thrombocytopenia purpura produce quite similar signs (see below). Neither is very common, but both are seen from time to time. They have a distinct aetiopathogenesis. Laboratory help may be needed in confirming the diagnosis.

Incidence

The condition is sporadic.

Aetiology

The cause is isoerythrolysis, an immune-mediated destruction of the piglets' red blood cells by antibodies acquired through the dam's colostrum. It occurs only if the sow is exposed to red blood cell antigens that come from the sire and that she does not possess herself. The sow produces antibodies to these 'foreign' antigens, but these do not pass through the placenta during pregnancy, and hence the piglets are healthy until they drink colostrum. Note that pigs have 15 blood groups; haemolytic diseases occur as a result of the dam's exposure to antigens of the E blood group.

Epidemiology

The condition occurs mostly in multiparous sows that have had previous exposure to the foreign antigen – very rare in primipara. It was seen more commonly when crystal violet swine fever vaccine was used against swine fever infection.

The pathology involves the piglets' red blood cells being destroyed by colostral antibodies, causing haemolytic anaemia and its consequences.

Clinical signs

Piglets are normal at birth but show signs of jaundice usually at 24 h following ingestion of colostrum. By 48 h, they may be severely anaemic and may die. Some cases in

an affected litter are subclinical. The strongest piglets in the litter may be worst affected, because they often take more colostrum and hence more antibodies.

Diagnosis

This is based on clinical signs and their time of onset. Blood examination reveals anaemia and a low packed cell volume.

Differential diagnosis

Other causes of neonatal haemolysis may be responsible. Crushing injuries and septicaemia may produce a similar picture.

Treatment

Nursing care is important. Intraperitoneal blood transfusion may help severely anaemic cases. Small doses of whole blood collected from an unrelated pig are injected carefully into the peritoneum. Vitamins may aid haemopoiesis.

Control

Avoid mating the sow to the same boar next time. If many sows are apparently affected by one boar, he should not be used for breeding. If the clinician suspects that sow–boar mating has already occurred, pigs can be saved by avoiding the mother's colostrum. Take them from the sow at birth, put on to bovine colostrum – possibly including plasma from an unrelated sow, and put back on to their own sow at 36–48 h, when colostral closure has occurred. A practical difficulty may be to get the sow to take her own litter again and also to strip colostrum from the sow; one could use older piglets to do this if the sow will accept them. An injection of oxytocin before trying to strip the udder may encourage milk let-down.

Thrombocytopenic purpura

Incidence

This is uncommon, but there were three cases in the VIDA survey for 2000 and one case in 2002.

Aetiology

The condition is caused by isoimmunity by foreign antibodies via the sow's colostrum, causing destruction of piglet platelets with a resultant tendency to bleeding.

Epidemiology

Thrombocytopenic purpura occurs in a multiparous sow's litters in most cases. Most cases are seen when Landrace sows are served by a Large White boar.

Clinical signs

The litter is normal at birth but may look uneven in size at a few days of age. The best piglets are often the worst affected, as they take more colostrum and adverse antibodies. Affected piglets are pale, weak, and listless. Temperature is normal. The faeces may contain blood. Haemorrhages in the skin may be visible (Fig. 8.13). Excessive bleeding from, for example, docked tail or castration wounds may occur.

Diagnosis

Haematology shows low platelet count but no haemolysis. Very few megakaryocytes are present in bone marrow. Check for crushing or septicaemia.

Post-mortem – Numerous internal petechial haemorrhages are seen (Fig. 8.14).

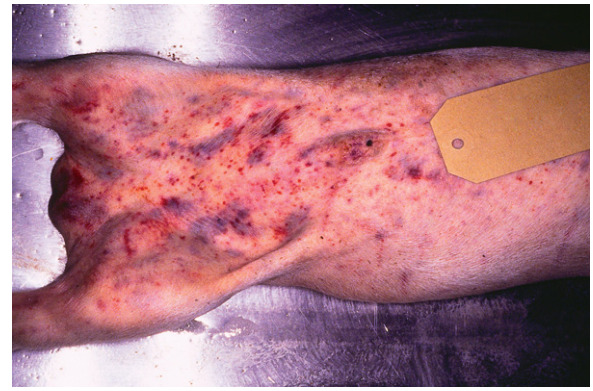

Fig 8.13 – Piglet thrombocytopenia purpura: petechial haemorrhages on the skin. (Courtesy of W.D. Strachan.)

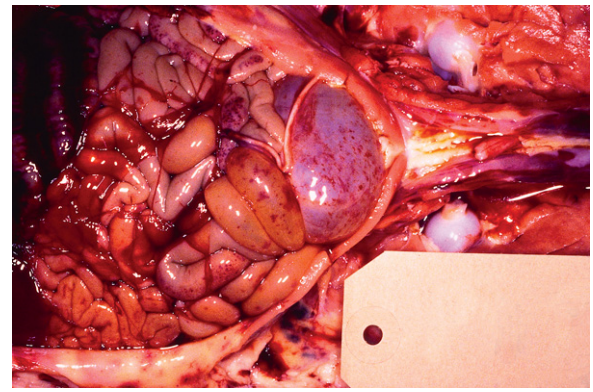

Fig 8.14 – Piglet thrombocytopenia: post-mortem appearance showing petechial and gross haemorrhages. (Courtesy of W.D. Strachan.)

Differential diagnosis

Purpura haemorrhagica is possible, in which case the platelet count is normal. Other causes of blood-clotting defects are other possible diagnoses.

Treatment

Keep piglets warm. Prednisolone injections may help. Intraperitoneal blood transfusion is given in severely anaemic cases.

Control

Avoid repeat breeding, and change the boar if several cases are seen in the herd that were sired by him. Avoid colostrum if there is a possibility of the disease (as under *Haemolytic disease of the newborn* above).

Purpura haemorrhagica

This has been reported in pigs after cases of septicaemia. Immune complexes are deposited in capillary walls. Subsequent vasculitis and loss of blood and plasma into surrounding tissues occur. One may see subcutaneous haemorrhages and local swelling. Death occurs in severe cases. Platelet count is normal (cf. thrombocytopenic purpura).

Navel bleeding in piglets

Incidence

This is relatively uncommon, but sudden outbreaks can occur on some units.

Aetiology

The cause is abnormal haemorrhage through the umbilical blood vessels as a result of a failure to close normally or through recurrent haemorrhage after previous closure. Vitamin C deficiency or vitamin K deficiency have been suspected in some cases, but in some herd outbreaks vitamin C therapy appears to have had little effect. The incidence of the condition may be increased by the use of sawdust or wood shavings as bedding. There is no evidence that the use of prostaglandin F2α to induce farrowing has any effect on the incidence of navel bleeding.

Clinical signs

The umbilical cord bleeds either at birth or suddenly starting a few hours after birth. The umbilical cord and navel appear thick and fleshy (Fig. 8.15). Serious blood loss can occur if unchecked; the piglet is pale, weak, and showing all the signs of anaemia – including death in severe cases.

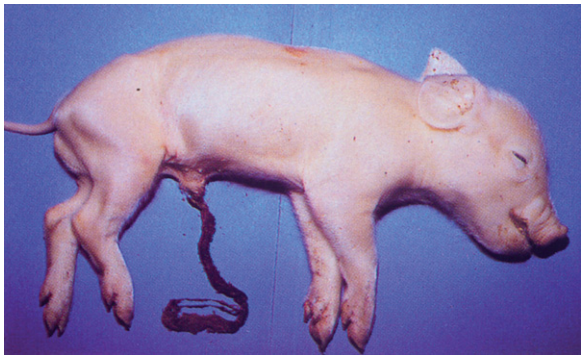

Fig 8.15 – Piglet navel bleeding. (Courtesy of S.H. Done.)

Treatment

Ligation or clamping of the umbilical cord is used.

Control

Vigilance at and after farrowing or routine ligation of cords at birth is necessary.

Ascorbic acid fed at a rate of 1.5 g/sow per day for 6 days prior to farrowing may help. Vitamin K therapy has also been used, but its effectiveness is unclear.

Avoid the use of sawdust and wood shavings as bedding for piglets.

von Willebrand's disease

Incidence

This is a rare disease.

Aetiology

The cause is an autosomal recessive gene affecting the blood-clotting mechanism. Factor VIII levels are reduced.

Clinical signs

There is excessive bleeding after minor injury, including injections. Some similarities to haemophilia are seen, but both male and female piglets are affected. Clotting time is prolonged to >15 min.

Diagnosis

This is based on the genetic background of the breeding stock, low levels of factor VIII in the blood, and the elimination of the presence of other clotting defects.

Treatment

First aid could be by local surgical treatment of accessible haemorrhages and intraperitoneal blood transfusion. Response to treatment is poor, and affected piglets are unlikely to survive in the long term.

Control

Avoid bloodlines carrying the disease.

Eperythrozoonosis

Incidence

Cases have been seen in the UK and Europe, but the condition is much more common in North America. Three cases were reported in the VIDA 2000 survey, one in 2002.

Aetiology

Eperythrozoon suis, a rickettsial parasite living in the pig's red blood cells, is responsible.

Epidemiology

Eperythrozoonosis is spread mostly by transmission of infected blood between animals, including by needle injecting pigs serially with a contaminated needle. It can also spread vertically and horizontally, with possible transmission by external parasites including the louse *Haematopinus suis*. Subclinical infection may pass unnoticed and can be activated by stress. The organisms cause haemolysis of the red cells.

Clinical signs

The organism is responsible for quite a wide spectrum of diseases, from sudden and severe to subclinical, and should always be kept in mind.

- Weakness and anaemia in neonatal piglets.
- Anaemia, jaundice, and poor growth in weaner pigs.
- Reproductive inefficiency in breeding herds, including delayed oestrus and embryonic death.
- Acute anaemia and jaundice in finishing pigs: high temperatures (40°C), poor exercise tolerance, and some deaths within 5 days of clinical signs appearing.

With subclinical infection, there is overall poor herd performance.

Diagnosis

An indirect haemagglutination test is helpful in diagnosing individual infected pigs. For herd diagnosis, enzyme-linked immunosorbent assay and also polymerase chain reaction tests are available.

The organism is also visible in Giemsa-stained blood smears.

Differential diagnosis

Other causes of anaemia and ill thrift are differential diagnoses.

Treatment

Parenteral administration of oxytetracycline is given; a single injection is usually effective, but a 5-day course is preferable. Larger groups can be treated with oral oxytetracycline for 14 days.

Control

In infected herds, chlortetracycline can be included in the food at 300 g/tonne. Oxytetracycline can be included in the drinking water. In herds where piglet infection is a problem, the sow can be given intramuscular oxytetracycline 7–14 days before farrowing. Control of lice is also important.

Vitamin B_1 deficiency

Cardiac aspects only are dealt with here.

Incidence

This is an uncommon condition.

Aetiology

A deficiency of vitamin B_1 is the cause. This may arise through:

- consumption of heat-treated carbohydrate waste, for example, bread; or
- long term (>10 weeks) consumption of bracken fronds and rhizomes containing thiaminase in outdoor herds.

Clinical signs

The deficiency can cause sudden death from heart failure and myocardial degeneration. One may also see anorexia and poor growth.

Diagnosis

This is based on clinical signs, exposure to bracken, and response to treatment.

Post-mortem – Heart muscle fibrosis, pleural effusion, and fibrosis are seen.

Treatment

Treat pigs with thiamine injections.

Control

Avoid exposure to bracken.

Warfarin poisoning

Incidence

This is sporadic after exposure to and eating of rat poison.

Aetiology

The cause is consumption of warfarin or coumatetryl and other coumarol derivatives. Accidental inclusion of warfarin poison in the diet can occur. Vitamin K deficiency is induced.

Epidemiology

Careless use of rat poison is responsible. Coumatetryl is particularly potent.

The blood-clotting mechanism is compromised, and fatal haemorrhages follow normal physiological movement and activity. Exposure over a period is mostly necessary to cause signs: 0.2–0.5 mg of warfarin/kg body weight is needed for >6 days to produce symptoms of poisoning. Dogs, cats, and pigs are the most susceptible species.

Clinical signs

Signs and consequences of haemorrhage are seen: weakness, pallor, recumbency, anorexia, blood in faeces, and epistaxis. There are swellings (haematomata) in leg muscles, with associated lameness.

Diagnosis

This is based on history, possible exposure, blood loss and its consequences, and prolonged clotting time.

Post-mortem – Numerous haemorrhages throughout the carcass are seen (Fig. 8.16).

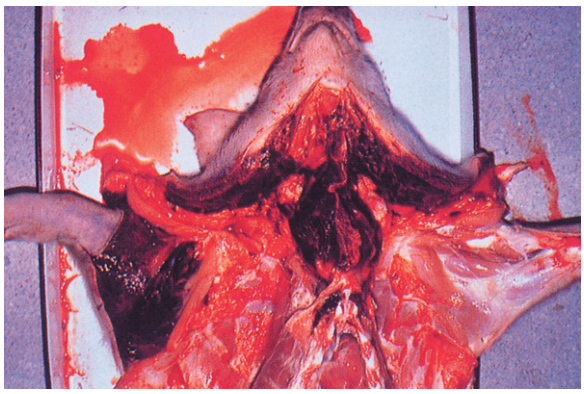

Fig 8.16 – Pig warfarin poisoning.

Differential diagnosis

Other causes of failure of the blood to clot.

Treatment

Vitamin K 50 mg/kg body weight is given by intramuscular injection.

Control

Avoid exposing pigs to rat poison.

Monensin poisoning (myoglobinuria)

Incidence

Monensin poisoning is rare, but there is a risk if pigs accidentally gain access to monensin and the effect of other additives (see below). Monensin is not licensed for use in pigs in the UK.

Aetiology

The condition is caused by consumption of monensin or its accidental inclusion with other ionophores (e.g. salinomycin and also tiamulin). These drugs are all coccidiostats and may have been accidentally included together in the diet. There is also accidental exposure to monensin concentrate. Levels of monensin >20 mg/kg of body weight can cause toxicity.

Clinical signs

These are anorexia, weight loss, and myoglobinuria. Sudden deaths, from acute heart failure, also occur.

Diagnosis

Base the diagnosis on clinical signs, ionophores in the diet, and elevated creatine kinase in blood. Estimation of the amount of monensin and other ionophores in food is carried out.

Post-mortem – Myonecrosis of muscles of tongue, limbs, and diaphragm is seen.

Differential diagnosis

Other causes of sudden death, anorexia, and weight loss are possible. The history of exposure to monensin and the characteristic post-mortem appearance will usually confirm the diagnosis.

Treatment

There is no specific antidote. Remove monensin from the diet. Provide nursing care.

Nitrite and nitrate poisoning (methaemoglobinuria)

Incidence

This is uncommon, but there is always a risk in intensive units.

Aetiology

Nitrite, nitrate, and nitrous oxide are all potentially toxic to pigs.

Epidemiology

Nitrates are in slurry and straw, nitrites in drainage water and in whey. Nitrous oxide can come from a farm silage pit. Nitrate is converted in the gut to nitrite, which causes conversion of haemoglobin to methaemoglobin. With over 75% of methaemoglobin in the blood, death occurs as a result of anoxia.

Clinical signs

These are dyspnoea, lethargy, cyanosis, and a rapid weak pulse. Terminally, opisthotonus, coma, and death occur.

Diagnosis

The clinician can assay methaemoglobin in blood by spectophotometry.

Post-mortem – Blood looks brown at post-mortem.

Treatment

Methylene blue is given intravenously into an ear vein at a dose of 2 mg/kg of body weight.

Carbon monoxide poisoning: anoxia due to carboxyhaemoglobin in piglets

This occurs mainly through exposure to faulty gas heaters in the farrowing or creep area.

High levels of CO have an adverse effect on pregnant sows and their unborn piglets. Beware of the problem on farms where gas heating is used – seek expert advice as required.

- CO >120 ppm in the air (n=50 ppm): abortion and high levels of stillbirth occur.
- CO >150 ppm: fetal death occurs. The fetus becomes anoxic; post-mortem shows cherry-red tissues and blood-stained pleural fluids. Extramedullary haemopoiesis occurs.
- CO >200 ppm: newborn piglets show lethargy and reduced growth; the sow is unaffected. In weaners, one may see growth reduction if CO is >200 ppm.

Carbon monoxide is highly toxic to people, and investigations should be carried out with care. Pigs should be removed from the source of CO, and oxygen therapy may be helpful in some cases.

Diseases of the urogenital system and the mammary gland

Please note: obstetrical conditions such as dystocia and uterine prolapse are covered in Chapter 10 (*Obstetrics and reproduction in pigs*). The infectious causes of infertility, abortion, etc. are also covered in Chapter 10.

THE UROGENITAL SYSTEM

Introduction

Infectious diseases of the porcine urogenital system are not very frequently encountered. When they occur, they may quickly compromise the health of the animal, and death from septicaemia or renal failure may ensue.

Aetiology of diseases of the urogenital system

A wide range of causes can result in disease and dysfunction of the urogenital system. Hereditary conditions such as renal agenesis are rare and are usually incompatible with life. Pyelonephritis with cystitis can be caused by a number of infectious agents, including *Actinobaculum suis*. A number of mycotoxins produced by fungi and contaminating food can rapidly damage the renal system. One of the most severe of these is ochratoxin A, produced by fungi of the *Aspergillus* and *Penicillium* genera. Physical obstruction of urinary flow can occur as the result of urolithiasis.

Epidemiology of diseases of the urogenital system

Although *Actinobaculum suis* is the causal agent most commonly associated with pyelonephritis in pigs, its presence in the urinary tract is not always associated with disease. It can be carried by the boar, but the trigger to clinical disease is not always identified.

Clinical signs of diseases of the urogenital system

The acute onset of some diseases in this section may not always be accompanied by obvious signs of clinical disease. Pyelonephritis may run such an acute course that death occurs before visible clinical signs such as abnormal urine are observed. An early sign of metritis may be a vaginal discharge.

In cases where metritis is caused by fetal retention and death, severe and sometimes fatal toxaemia may occur before any external signs, including a vaginal discharge, are seen.

Diagnosis of diseases of the urogenital system

This is based on the history of the patient, its herd, the clinical signs, and the use of special diagnostic tests. In outbreaks of pyelonephritis, for example, there may be a history of a new boar serving a batch of gilts. Sudden death may be seen in one gilt, haemorrhagic and purulent urine in another. Post-mortem examination of the dead gilt may reveal purulent material in the pylorus of each kidney, with infection possibly involving the bladder. Urine samples can be collected from any other gilts looking unwell, and blood samples may reveal elevated urea and creatinine levels.

Treatment of diseases of the urogenital system

Early treatment is necessary for the infectious diseases in this group. This is essential to prevent severe tissue damage and dysfunction of the organs involved. In cases of pyelonephritis, antibiotic therapy supported by non-steroidal anti-inflammatory drugs (NSAIDs) should be commenced as soon as the disease is diagnosed. In-contact animals should also be treated with antibiotics if they are thought to be at risk. The rapid progress of this disease is such that uraemia and toxaemia develop rapidly. Euthanasia may be required for severely ill animals.

Control of diseases of the urogenital system

Dirty conditions in sow or gilt accommodation may predispose to pyelonephritis and also mastitis. Clipping piglets' teeth may be indicated on farms with a high incidence of mammary disease. A poor water supply may predispose to urolithiasis and to other conditions, such as 'salt poisoning'. As in other diseases, good standards of management are essential to reduce the incidence of disease.

Veterinary Investigation Diagnosis Analysis sample submissions: diseases of the urinary system, 1996–2003

See Table 9.1.

Hereditary abnormalities of the urogenital system

Bilateral renal hypoplasia

This is uncommon and incompatible with life. The cause is an autosomal recessive gene. The condition is seen chiefly in Large White pigs. Piglets may live for up to 8 weeks before dying.

Renal agenesis

Occasionally, complete absence of kidneys occurs in renal agenesis. Death as a result of uraemia occurs within a few hours of birth.

Cystic kidneys

These are uncommon but occasionally seen in Landrace and Large White pigs. They are caused by an autosomal dominant gene. Multiple cysts are found in tubular parts of the kidney. The lesions are often subclinical, unless they lead to renal failure. They may be found at slaughter.

Cryptorchidism

Cryptorchidism is quite common. It is caused by a sex-linked autosomal recessive gene. Several male piglets may be affected in a litter. The undescended testis may be within the peritoneal cavity or the inguinal ring. The left testis is most commonly involved. Affected animals should not be used for breeding. Cryptorchidism is not a problem in finishing pigs, which are usually slaughtered before puberty.

Table 9.1 Veterinary Investigation Diagnosis Analysis sample submissions: diseases of the urinary system, 1996–2003

Cause	Incidence (%)
Porcine dermatitis and nephropathy syndrome	60.58
Pyelonephritis	21.88
No diagnosis	5.77
Nephrosis not specified	4.81
Nephritis not specified	2.88

Pseudohermaphroditism

This condition is quite commonly seen. Affected pigs are male pseudohermaphrodites that have a partially male phenotype but are genetically female. They have a uterus and testes, which may be either abdominal or scrotal. They may have either a penis or a vulva and clitoris. Affected animals are infertile, and the condition has a hereditary predisposition.

Other conditions of the urogenital system

Cystitis and pyelonephritis

Incidence

This is an important condition that can reach near epidemic proportions on some units. It occurs wherever pigs are kept throughout the world, especially in intensive units. The condition can be a major cause of sow deaths in some units.

Aetiology

Actinobaculum suis (formerly *Corynebacterium suis* and *Eubacterium suis*) is responsible. A vaginal injury at service or parturition may be needed to allow the organism access to the renal system. Very similar signs may be caused by infections with streptococci, staphylococci, and *Escherichia coli*, either alone or in combination. *Actinobaculum suis* is usually responsible for multiple case outbreaks. Poor hygiene and lack of good drinking water also predispose, as does the short urethra of the sow.

Epidemiology

The organism is transmitted by the boar at service. The organism is found in the prepuce and prepucial diverticulum of up to 90% of boars, who often pick up the infection at a very early age. In other cases, the boar can become infected at the time of service to an infected sow or gilt.

The condition was more common in tethered sows or sows in stalls (both now illegal in the UK), especially if faeces were allowed to accumulate behind the sow. The reduced movement and infrequency of urination in these animals was thought to predispose to urinary infection.

Outbreaks of the disease may be attributable to one boar in the herd. It is more common in older animals and if water intake is low.

Clinical signs

Dullness, discomfort, and anorexia are apparent 1–3 weeks after service. Urination is painful and the urine bloody and purulent. The animal may stand with her back arched. Temperature is initially raised to 40–41°C, but the sow quickly goes into renal failure and temperature

may be low when first presented. Clinicians should always check for any evidence of cystitis and pyelonephritis in a sow that is dull and has a low temperature after service. If untreated, the animal often deteriorates rapidly as renal failure develops. Signs of ill health and abnormal urine may be overlooked if sows are not closely observed at least once daily.

Diagnosis

There is a history of recent service in most cases, and the clinical signs are suggestive of the problem. A urine sample should be collected. It may encourage the sow to urinate if her vulva is tickled. Alternatively, a plastic catheter can be passed manually into the urethra. Blood, pus, and shreds of mucosa are visible in the urine of affected animals. The bladder can be examined endoscopically, which allows the clinician to check that blood-stained fluid is not coming through the cervix from the uterus. Examination by vaginal speculum will also help identify the source of haemorrhagic or purulent material. The bladder is found to be thickened on rectal examination. Blood urea and creatinine levels are elevated.

Post-mortem – A thickened, inflamed bladder wall is observed. Obstructed ureters are found in some cases, and pyelonephritis is often present (Fig. 9.1). The causal organism can be difficult to culture. The blood urea level in the aqueous humour may be elevated in pigs that have died of renal failure.

Prognosis

Prognosis is guarded unless treatment is commenced very early.

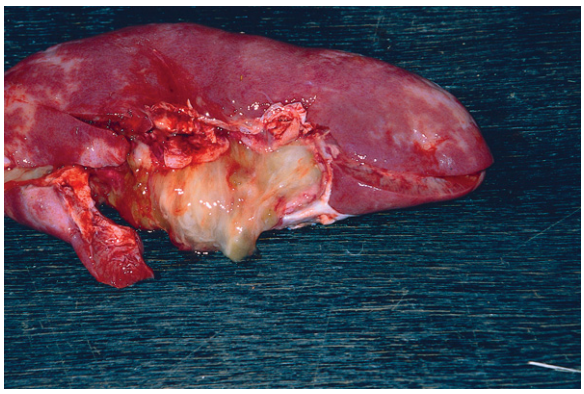

Fig 9.1 – Pyelonephritis: diseased kidney. (Courtesy of R.W. Blowey.)

Treatment

Antibiotic therapy is used: penicillin–streptomycin, oxytetracycline, or enrofloxacin should be given for at least 7 days. NSAIDs may provide pain relief and reduce the severity of endotoxic shock. Fluid therapy via an ear vein may be indicated, but the cost and management of this should be discussed with the farmer. Euthanasia is advised in terminal cases.

Control

There should be a general improvement in hygiene at mating and in the postservice accommodation. Water quality and availability should be checked. If one boar is suspected of being the source of infection, treatment by prepucial irrigation and parenteral antibiotic therapy for 7 days should be given. Antibiotics may be given prophylactically for any sow showing bleeding after service. In herd outbreaks, regular treatment of all sows with oxytetracycline in the food or water may reduce the incidence of clinical cases.

Urolithiasis (urethral urolithiasis)

This condition is uncommon, but occasional outbreaks have been described. Uroliths are occasionally found incidentally at post-mortem in normal adult and neonatal pigs. A range of chemical compositions of uroliths have been identified. These have included calcium carbonate, calcium phosphate, magnesium ammonium phosphate, and also uric acid.

Predisposing factors include reduced water availability and a pre-existing urinary infection. In some outbreaks, no predisposing factors have been identified.

Clinical signs may include dullness and anorexia. Anuria or oliguria may be present but are not always identified. Chalky fragments may be found in voided urine. Rupture of the bladder may occur; abdominal distension, pain, and death may follow.

Cases are seldom identified until the pigs are terminally ill. Surgical treatment is seldom considered economic, and euthanasia may be required.

Porcine dermatitis and nephropathy syndrome

This important condition of weaned pigs is believed to be caused by porcine circovirus 2, with other additional causal factors being involved. It can result in terminal renal failure in affected pigs. For full discussion, see Chapter 11 (*Polysystemic diseases*).

Metritis

This may be part of the mastitis–metritis–agalactia (MMA) syndrome, in which case a white or yellowish

vaginal discharge may be seen. This mild condition is further discussed below.

Acute metritis after farrowing is a serious and potentially life-threatening disease. The problem may develop as the result of an unhygienic farrowing environment or careless obstetrical assistance. It may also be associated with the retention of piglets in the uterus at the end of farrowing. When piglets are retained, they may become infected and oedematous and very difficult for the sow to deliver or the obstetrician to remove. Such sows become seriously ill, with pyrexia, anorexia, and a foul-smelling vaginal discharge. In other cases, if infection is not present the retained piglets may become mummified and may then cause no adverse signs. For further information on treatment of this condition, see Chapter 10 (*Obstetrics and reproduction in pigs*).

Vaginal discharge during pregnancy

Incidence

Vaginal discharge during pregnancy (Fig. 9.2) is very common. It can be a herd problem, and up to 25% of herds may be affected. The condition may be associated with poor breeding results, but in some cases these are normal and the discharge appears to be incidental.

Aetiology

A number of bacteria may be associated with the condition, either alone or in combination. *Arcanobacter pyogenes*, *Escherichia coli*, *Staphylococcus hyicus*, streptococci, and *Proteus* have been cultured from cases.

Epidemiology

The condition is thought to be associated with poor hygiene either in the sows' accommodation or at service. Some infections may be transmitted by the boar at service. Infection may lead to endometritis and infertility.

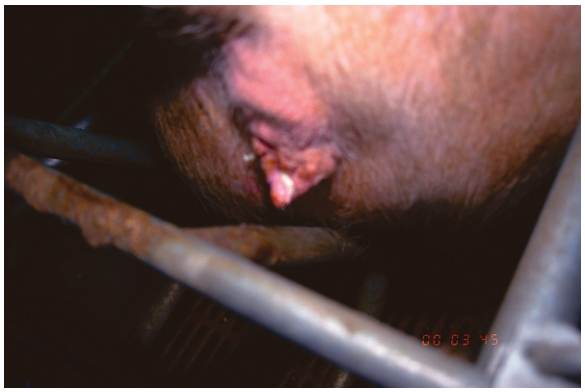

Fig 9.2 – Sow vaginal discharge.

Clinical signs

Vaginal discharge is present 1–3 weeks after service. Purulent material may be observed around the vulva or adherent to the ventral surface of the tail. The sow is usually quite bright and shows few systemic signs, and her temperature is normal.

Diagnosis

Vaginal discharge is seen. A speculum is used to view the vagina – the clinician may observe a vaginitis and, if the uterus is involved, purulent material coming through the cervix. The presence of blood in the discharge suggests that cystitis may be present. A urine sample should be evaluated to check if this might be the case. Pregnancy diagnosis should be performed as early as possible.

Treatment

Antibiotics are given parenterally. Penicillin–streptomycin, ampicillin, cephalexin, and trimethoprim can be used, depending on sensitivity tests, if a herd problem is present. A 5- to 7-day course of antibiotic is required. Intrauterine treatment seldom is prescribed or needed. In non-pregnant animals, the uterus may be irrigated with very dilute chlorhexidine solution, but the length of uterine horns makes the value and efficacy of this treatment questionable.

Control

Improve hygiene. Check fertility records. Administration of prophylactic antibiotic therapy after service in herd outbreaks may help.

Vulval damage

Bite wounds to the vulva may occur when sows are mixed after weaning and in late pregnancy. They can also occur if sows are backing out of an electronic feeder while others are waiting to feed. Wounds may bleed profusely or become infected (Fig. 9.3). Treatment is by using general surgical principles. Local antibiotic spray is useful to prevent local infection becoming established. Tetanus antitoxin should be administered in areas where the disease is endemic. Damage should be prevented by careful mixing of pigs – this can be difficult to achieve under farm conditions.

Prepucial diverticulitis

Prepucial diverticulitis is uncommon. Blood coming from the prepuce or the sow's vagina may be seen after service. Penile bleeding is not observed. The diverticulum may be

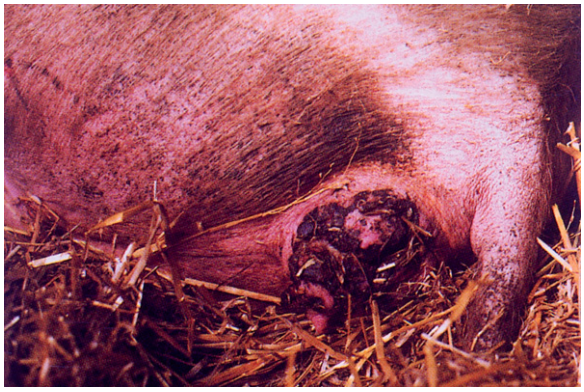

Fig 9.3 – Sow vulval injury.

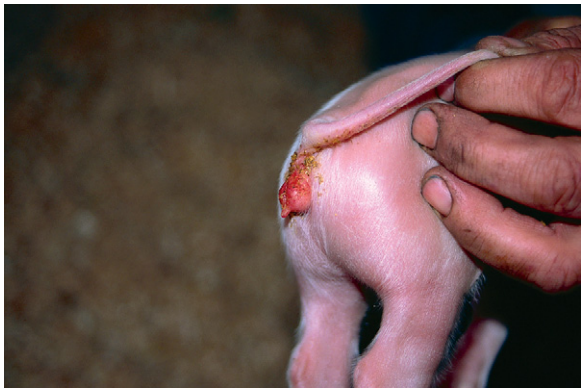

Fig 9.4 – Piglet with vulvovaginitis. (Courtesy of R.W. Blowey.)

swollen and firm. Ulceration may be seen on the internal surface if this can be examined. The exact cause of the condition is unknown; *Chlamydia* and *Actinobaculum suis* infection have been suspected, but their significance has not been confirmed. Distension of the normal prepucial diverticulum may occur; the contents can be expressed with a gloved hand – they are foul-smelling but without purulent material.

Treatment is with oxytetracycline solution, which can be administered by instillation into the prepuce. The prognosis for recovery is poor. See also discussion of surgical removal in Chapter 15 (*Analgesia, anaesthesia, and surgical procedures in the pig*).

Mycotoxins affecting the urinary system

Vulvovaginitis and zearalenone poisoning

Incidence

Vulvovaginitis (Fig. 9.4) is uncommon but if confirmed it can have severe consequences for an affected herd.

Aetiology

The cause is an *oestrogenic mycotoxin* produced by a species of the fungus *Fusarium*, which can grow in mouldy barley or maize. The toxin is thought to act by suppressing serum gonadotrophin (luteinizing hormone) levels.

Epidemiology

It can affect all farm species, but the problem is most frequently seen in pigs. Signs appear within a few days of pigs being exposed to the mycotoxin and disappear within a few days of the toxin being absent from the food.

Clinical signs

Vulvovaginitis is seen in all ages of pig; signs may be most pronounced in gilts aged 6–7 months. Female piglets on the sow may also be affected; toxins pass into the milk and hence to unweaned piglets. Vaginal and rectal prolapses may be seen in some affected animals. Other signs include anoestrus or constant oestrus, infertility, early farrowing, stillbirth, and pseudopregnancy in breeding animals. Newborn piglets may show splay legs and paresis. Male piglets may develop seminiferous tubule malformations.

Diagnosis

This is based on clinical signs and exposure to mouldy food. The clinician may be able to demonstrate the presence of the toxin in food or plasma by an enzyme-linked immunosorbent assay (ELISA). The toxin can be found in the bile of slaughtered sows.

Treatment

Change the diet urgently. Affected gilts should return to normal, and breeding can be resumed after dietary change has been instituted.

Control

Avoid mouldy food if possible. Food should contain less than 10 ppb of zearalenone toxin. Improve food storage to reduce the possibility of mycotoxins being present.

Note that *Fusarium* spp. fungi may also produce vomitoxin, another mycotoxin that can be present with zearalenone. Vomitoxin produces vomiting and anorexia.

Mycotoxic nephropathy: ochratoxicosis

Incidence

This is uncommon but has serious consequences. It has worldwide incidence. The first case was observed in Denmark, but the condition has also been seen in the UK.

Aetiology

The cause is ochratoxin A, a powerful renal toxin produced by a number of fungi, chiefly *Aspergillus ochraceus*. It may also be produced by a number of other *Aspergillus* and *Penicillium* species. The fungi can be present in damp and mouldy barley and other cereals – a risk when storage is poor.

Epidemiology

Contamination by >200 ppb of mycotoxin in the diet can cause signs of toxicity. Cases of ochratoxicosis have been reported in which up to 7000 ppb were found in the diet. Signs may appear 3 weeks after exposure. When pigs are put on to uncontaminated food, the mycotoxin may take 4 weeks to disappear from the body. The mycotoxin causes degenerative changes in the renal tubules and loss of effective renal function.

Clinical signs

In finishing pigs, anorexia, diarrhoea, polydipsia, polyuria, and reduced weight gain may be seen. Weaners may have subcutaneous oedema, arched backs, and swelling of the upper parts of the lateral flanks, which may indicate pronounced renal enlargement and oedema.

Diagnosis

An ELISA is available to detect and measure ochratoxin. Usually, no attempt is made to identify the fungus or mould involved. Histological changes of tissue degeneration are found in the kidneys.

Differential diagnosis

Other causes of renal failure, including porcine dermatitis and nephropathy syndrome, could be responsible. Bowel oedema in weaner pigs should also be considered – for details see Chapter 5 (*Diseases of the gastrointestinal system*).

Treatment

Exposing food to ammonia and also to sodium hydroxide has been claimed to help reduce the mycotoxin content of contaminated food. Immediate replacement of the diet with uncontaminated food is essential.

Control

Monitor mycotoxin levels in the diet. One can dilute affected food to reduce ppb levels and thus possible toxicity. The diet should contain <10 ppb of the mycotoxin.

Ergot poisoning

Incidence

Ergot poisoning is uncommon.

Epidemiology

Contaminated cereals with >2 g of ergot/kg of food can lead to toxicity.

Clinical signs

Ergot poisoning may increase the stillbirth rate in a herd. Small and non-thriving piglets may be born. Agalactia and loss of piglets through starvation may also be seen. In some cases, the udder fails to develop and no milk is produced. See also Chapter 6 (*Diseases of the skin*), where local gangrene of the skin caused by ergot poisoning is described.

Treatment

Remove ergot from the diet. Milk let-down improves within a few hours of removal of ergot from the diet. There is no milk let-down response to oxytocin in animals suffering from ergot poisoning.

THE MAMMARY GLAND

Introduction

Diseases of the udder are quite common. They may pose a threat to the life of the sow and also affect the well-being of the litter. Acute mastitis can be severe and life-threatening, but all diseases of the udder cause some compromise of the sow's milk supply. Starvation of the litter may result in severe cases. Piglet health may be further put at risk through insufficient colostrum intake, and growth rate can be reduced.

These are a very important group of diseases that are quite common. Good milk production and effective milk let-down are vital if piglets are to survive. Piglets are extremely vulnerable to starvation, hypothermia, and dehydration in the first few hours and days of life. Failure to take in colostrum can predispose them to disease, and failure to drink can also rapidly lead to hypoglycaemia and death.

The sow and her litter should always be regarded as a unit, and whenever the veterinary surgeon is called to see either of them they should always both be examined.

The following questions should be considered.

- Are the piglets ill because their mother has no milk?
- Are the piglets not feeding, even though the sow has adequate milk, because they are unwell?
- Are there problems with both the sow and the piglets that are making them both unwell?

Aetiology of diseases of the udder

Infectious agents are a major cause of mammary disease in pigs. Acute mastitis can be caused by a number of agents, including *Klebsiella* spp. The exact cause of the mastitis-metritis-agalactia (MMA) syndrome is not known. Infection by *Escherichia coli* is involved, but this can be predisposed by errors of management, including overfeeding before farrowing. Injuries by the unclipped teeth of piglets may cause minor but painful injuries to the udder, causing the sow to be reluctant to suckle her piglets. In some cases, infection gaining access through these bites and other injuries may result in chronic mastitis.

Epidemiology of diseases of the udder

The incidence of mammary diseases is predisposed by dirty conditions in the farrowing house and by defects of sow management. *Klebsiella* spp. are the causal organisms in some cases of acute mastitis. Wood shavings are thought to be an important source of the organism. Mammary disease can be life-threatening but, as mentioned above, also compromises the neonatal milk supply. Piglet health can be rapidly affected, and failure of passive transfer of colostral antibodies may increase their susceptibility to neonatal diseases.

Clinical signs of diseases of the udder

In acute mastitis and in MMA, the first sign of disease may be hunger and sometimes ill health in the piglets. Further examination reveals that the abnormalities observed in the piglets may have been initiated by maternal mammary disease.

In cases of MMA, a mild generalized induration of the udder is seen with few systemic signs in the sow. Acute mastitis causes inflammation of a small number of mammary glands, accompanied by severe toxaemia and septicaemia.

Diagnosis of diseases of the udder

Ill health in either the piglets or the sow in suckling animals should instigate a careful examination of both. Signs of hunger in the piglets may indicate that they are hungry as a result of reduced milk production by the sow, or that they are too unwell to drink what is available. Careful examination, including palpation, of the udder will indicate hardened diseased areas. A small quantity of milk may be collected from affected glands and the secretion cultured to enable the organism to be identified and an antibiotic sensitivity test performed.

Treatment of diseases of the udder

Acute mastitis is a life-threatening condition, and prompt antibiotic therapy is essential if recovery is to occur. MMA is a less severe condition, but rapid treatment is essential to restore milk production as soon as possible. In cases of mastitis and MMA, artificial milk should be offered to the piglets and they should be observed closely for neonatal disease, especially enteritis.

Control of diseases of the udder

Good hygiene in the farrowing quarters is essential to reduce the incidence of infectious agents. Overfeeding before farrowing should be avoided, as it may predispose to mammary disease. Examine sows before farrowing to ensure that there is no chronic mastitis or damaged teats. Careful watch of the neonatal litter to detect any early signs of milk insufficiency and regular palpation of the udder will allow early detection and treatment of disease. Routine clipping of piglets' teeth may be required in some herds. Regular exercise is very beneficial but cannot always be provided.

Veterinary Investigation Diagnosis Analysis sample submissions: mastitis and metritis, 1996–2003

See Table 9.2.

Diseases of the mammary gland always increase the risk of piglet starvation. A shortage of milk can have very serious consequences, and provision must always be made for artificial feeding if the sow's milk supply is insufficient or unavailable for the piglets. Piglets return to the sow's milk when she is lactating again, preferring her milk to milk substitute.

Causes of agalactia in the sow

Mammary aplasia

This is a complete absence of mammary tissue (Fig. 6.5); it is very rare in pigs but may occur with ergot toxicity (see above).

Table 9.2 Veterinary Investigation Diagnosis Analysis sample submissions: mastitis and metritis, 1996–2003

Cause	Incidence (%)
Metritis	54.90
Mastitis (*Escherichia coli*)	17.64
Other	15.68
Mastitis (*Klebsiella*)	5.88
Mastitis (*Staphylococcus*)	4.90
Mastitis (*Arcanobacter pyogenes*)	0.98

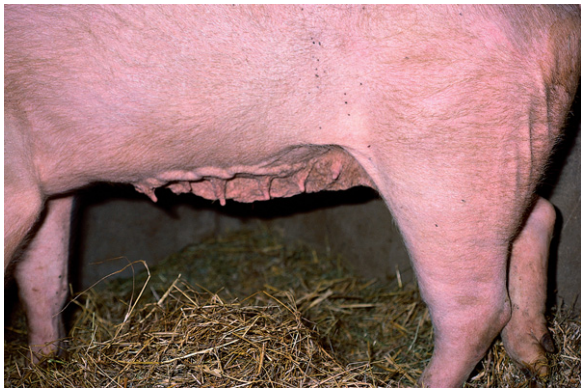

Fig 9.5 – Agalactia in a thin sow.

Inversion of nipples

One or several teats may be inverted. The condition is believed to be hereditary and caused by a recessive gene. It may be caused by excessive fibrous tissue at the base of the affected teat and should have been observed when the gilt was selected for breeding.

Nipple necrosis

Loss of nipples in female piglets is caused by damage from rough or sharp floor surfaces. See also Chapter 6 (*Diseases of the skin*).

Litter savaging by the sow

This results from puerperal psychosis – the sow is too aggressive to settle down and feed her litter. She may have a hereditary predisposition to the problem. The sow may kill her piglets as soon as they are born.

Treatment involves rescuing the piglets and keeping them warm. Check that farrowing is complete and that the sow is otherwise normal. Sedate the sow using azaperone by intramuscular injection or intravenous pentobarbitone sodium. Once the sow is sedated, give 20 IU of oxytocin by intramuscular injection to encourage milk let-down. Clipping piglets' teeth can be justified, and the animals are placed on the sow to take colostrum. The sow will mostly accept pigs when sedation wears off, but it may be necessary to repeat the sedation. Farm staff must ensure that she does not become aggressive again. See also Chapter 10 (*Obstetrics and reproduction in pigs*).

Failure of milk let-down reflex

The sow or gilt has milk but no let-down. The sow is unable to let her milk down at less than 40-min intervals, except when she is farrowing. When farrowing, a jet of milk can be squeezed from the teat under pressure. Such milk ejection is usually possible only later, when the sow is actually feeding her pigs.

Reasons for milk let-down failure are as follow.

- The sow is unwell and unwilling to feed her pigs. The clinician must treat any diseases (e.g. mastitis or MMA; see below). Treatment should include oxytocin to encourage let-down. See below for specific treatment for mastitis and MMA. Ill health often reduces appetite and water intake; milk supply may be reduced in addition to the sow's unwillingness to let her milk down. See further discussion under *Sow unwell* below.
- The sow is aggressive. See under *Litter savaging by the sow* (above). The sow is upset by, for example, late introduction to a farrowing crate, or she is frightened of attendants. Investigate the cause of her upset and treat by sedation and oxytocin.
- The piglets' teeth are hurting the sow's udder when they try to feed – clip the piglets' teeth.
- The piglets are unwell and not trying hard enough to stimulate let-down. Check the piglets, especially for enteritis. Treat them accordingly.

Sow unwell

This could result from general ill health, specific disease, or mammary gland diseases. Any general illness in a lactating sow may cause suppression of milk production and may affect the willingness of the sow to let down her milk. Anorexia, pyrexia, and pain can all result in reduction of milk production and piglet hunger. An elderly, lame sow or a sow with erysipelas or mastitis may have an inadequate milk supply for her litter. Treatment must be specific for the sows' illness and encourage the let-down of what milk they are producing. The litter must receive supplementary milk substitute if they are hungry, and they must be watched in case they become unwell.

Diseases of the mammary gland

Note: there is some confusion in the literature concerning acute mastitis and the MMA syndrome, both of which

produce some induration of the mammary glands. Some authors consider the two diseases to be different forms of the same syndrome. Others, including the present authors, think that they are so different in their clinical presentation that they should be considered separately.

Acute mastitis is potentially life-threatening to the sow, while MMA syndrome is a relatively mild disease. Both result in severe reduction of the milk supply, and if untreated they may rapidly be accompanied by heavy piglet losses.

Acute mastitis usually involves only a few (one to six) of the mammary glands; MMA usually produces induration in all the mammary glands. Body temperature is often elevated in cases of acute mastitis but lowered in cases of MMA.

The conditions are discussed in greater detail below.

Fig 9.6 – Acute mastitis. Note the inflamed udder and piglets in poor condition.

Acute mastitis

This is an acute, potentially life-threatening disease usually involving only a small number of the mammary glands.

Incidence

Acute mastitis occurs worldwide wherever pigs are kept. It may be sporadic or there may be outbreaks on a unit.

Aetiology

Klebsiella spp., *Escherichia coli*, and possibly Gram-positive organisms (e.g. staphylococci) are the cause. Sawdust bedding is particularly likely to harbour *Klebsiella* spp.

Epidemiology

Infection is believed to gain access through one or more teat streak canals in contact with, for example, contaminated bedding. Sows have two glands per teat and two streak canals per teat, and thus many routes for bacteria to gain access. One or both glands supplying each teat may be affected. The disease has been artificially produced by painting cultures of the organism on to the teat ends of recently farrowed sows. Wet and dirty sawdust bedding contaminated by large numbers of pathological organisms is particularly dangerous. Bites by unclipped piglets' teeth may allow organisms to enter the udder. Infection is usually seen 1–3 days after farrowing.

Clinical signs

The sow is dull, depressed, and anorexic. Temperature is initially elevated to 40–42°C, but in toxaemic animals temperature falls rapidly and may soon become subnormal. Small numbers of glands (usually <30%) are usually involved but occasionally more. The affected glands may be all on the same side of the udder or some on either side. Caudal glands are more commonly affected than pectoral glands. The udder is extremely hard ('rock hard'); the overlying skin may be either slightly reddened or pale, and the udder is painful and oedematous (Fig. 9.6). The piglets are in poor condition.

White sows may have purple blotches associated with septicaemia and toxaemia over the skin of the jowl, flanks, and caudal aspects of hind legs. The sow may be reluctant to stand and may have to be helped to get to her feet. She may vocalize with a thin reedy squeal. Her gait may be hesitant and ataxic. The milk in affected glands may be watery, blood-stained, or purulent.

Treatment

This should be intense and requires a combination of drug therapy and good nursing care. For antibiotic therapy, a neomycin–penicillin combination has been found to be effective. The clinician may alternatively use intravenous soluble ampicillin. Clavulanic acid–amoxycillin has also been found effective. NSAIDs or corticosteroids should also be given. Fluid therapy intravenously through an ear vein is beneficial if the expense can be justified. Euthanasia may be necessary in severely ill animals. Intramammary therapy is not normally attempted; in severe cases, antibiotic solutions can be injected directly into the udder, but there is little evidence that this will help.

With nursing care, keeping the very sick sow 'busy' – getting her up at frequent intervals and getting her out for a walk if possible – often has a beneficial effect by encouraging her to drink, eat, and urinate. Tempt her with oral fluids: water, electrolytes, or a little milk. Massage of the affected parts of the udder with udder cream or goose grease may help if the farmer can be persuaded to undertake this task. Piglets will require artificial milk (e.g. Faramate, Volac), which they can drink from a low dish too heavy for them to tip up and

spill. The piglets should be left with the sow, and it is hoped that when her milk returns they will take this in preference to artificial piglet milk. The piglets should be closely observed for evidence of diarrhoea, and they should be treated immediately if they show signs (see Chapter 5).

Prognosis

Prognosis is very guarded in a severely sick sow, especially if the disease is well established. There is a better prognosis if good nursing care is available. Early and intense treatment has the best prognosis.

Control

Improve farrowing house hygiene and avoid sawdust bedding. Clipping piglets' teeth shortly after birth may be justified on farms with mammary problems. Encourage the sow to stand shortly after farrowing and, if possible, to leave the pen at regular intervals; the latter is not possible in many modern buildings. Teat dipping or painting with organic iodine dip before farrowing and once or twice daily may help prevent the condition. In the face of outbreak, try to farrow sows elsewhere and consider prophylactic antibiotic therapy for newly farrowed sows. Thorough cleaning and resting of the farrowing house is beneficial. An outbreak in a herd often disappears as quickly as it arrives.

Mastitis–metritis–agalactia syndrome

Incidence

This is common. Sporadic cases of the condition may occur or there may be a group of cases, sometimes reaching epidemic proportions, followed by a sudden cessation of the problem.

Aetiology

The cause is not clear; it is possibly an endotoxaemia with *Escherichia coli* involvement. Toxin may come from the organism in the gut or (less likely) the uterus. β-Haemolytic streptococci have also been implicated. Management factors predispose – see under *Epidemiology*. Prolactin and oxytocin levels may be reduced.

Epidemiology

The condition is usually seen 12 h to 3 days after farrowing. Affected sows are often thought to have excessive milk at farrowing time. Restlessness and depression of piglets draw attention to the poor milk supply in the sow. Lack of periparturient exercise, excessive feeding around farrowing, very finely ground food, and poor hygiene may all predispose to the problem. Suppression of prolactin production by endotoxins may be one cause of lack of milk. Anorexia and failure to drink may also be involved.

Fig 9.7 – Mastitis–metritis–agalactia: sow with piglets. (Courtesy of W.A. Noble.)

Fig 9.8 – Mastitis–metritis–agalactia. Note vulval discharge. (Courtesy of W.A. Noble.)

Clinical signs

The sow goes off her food and is unwilling to feed her piglets. She lies in sternal recumbency, preventing piglets gaining access to her udder. Piglets appear hungry; they squeak, drink water or urine in the pen, look empty, and may develop other problems such as enteritis.

The udder is generally indurated; mostly the whole gland is involved. A lateral ridge of slightly indurated mammary tissue may be palpable on both lateral edges of the udder just below the normal thicker skin of the flanks (Fig. 9.7). Less milk than normal can be expressed from the teats; any milk that is seen appears to be grossly normal.

Temperature may be slightly elevated but is usually subnormal. Slight white or occasionally yellow vaginal discharge is seen (Fig. 9.8).

Diagnosis

Base diagnosis on a history of recent farrowing and often of apparently excessive milk production at farrowing, as well as clinical signs. Mastitis (see above) is usually much more acute, with only part of the udder involved, and has the risk of death.

Prognosis

This is good. Milk production usually starts again within 3 days, but unless carefully fed and managed, the litter may have died in the meantime.

Treatment

Antibiotic therapy is used. Penicillin–streptomycin is very effective; enrofloxacin and cephalexin are also recommended. Steroid therapy – betamethasone – is also helpful. Oxytocin may be used to encourage milk let-down. Nursing care is also important; exercise, tempting food, and artificial milk (e.g. Faramate, Volac) for the piglets.

Control

Avoid the predisposing factors mentioned above.

Chronic mastitis

Incidence

The condition is common. Twenty per cent of cull sows have chronic mastitis, and in 60% of animals the infection involves one mammary gland only. It is mostly seen after weaning but may occur and probably starts during lactation. When a sow is dried off, the rest of the udder reduces in size, leaving the infected gland more clearly defined.

Aetiology

Chronic mastitis is caused by bacterial infection, especially *Arcanobacter pyogenes*, staphylococci, streptococci, and less commonly bacteroides, *Fusobacterium*, and clostridia.

Epidemiology

Infection may gain access through the teat canal as a result of teat injury or a bite from a piglet or another sow. Perforated metal flooring may predispose to udder injury and infection. An abscess may develop. Fibrous tissue may form around the abscess, which may remain hard and nodular or may burst externally.

Fig 9.9 – Sow chronic mastitis.

Clinical signs

A mass is seen on the lateral side of the udder, mostly after weaning (Fig. 9.9). No systemic signs are usually seen. An abscess is usually present, which may become hard and fibrous or burst, discharging pus over adjacent skin. The teat may show signs of earlier injury. Lesions may be seen, with a soft, warm fluctuant surface before they burst spontaneously. It is not always clear exactly which tissues are involved; it may be the mammary gland and/or the adjacent subcutaneous tissues.

Diagnosis

Base on clinical signs, history, and needle aspirate of the swelling to check if it contains free pus or serum.

Treatment

If an abscess is forming, open and drain under local anaesthesia. Flush and keep open to avoid premature closure. Long-acting antibiotic may help more chronic, established lesions. One may have to cull if response to treatment is poor.

Control

Check the udders of sows carefully at weaning. Treat any lesions seen as above. One can often avoid culling with early treatment, although one or more glands may be lost to future lactation. Try to avoid mixing sows at weaning – fighting may lead to udder lesions.

10

Obstetrics and reproduction in pigs

Introduction

Good fertility levels are essential for pig herd profitability. Any departure from the anticipated reproductive performance can have devastating effects on herd profitability, especially when profit margins are low. Farrowing must be closely monitored if the maximum numbers of healthy piglets are to be delivered by each sow. The stillbirth rate in pigs is closely related to the duration of labour and is particularly high in certain types of dystocia. Early recognition of cases of dystocia and their effective treatment are essential factors in maintaining both good production and welfare parameters.

A good understanding of normal porcine reproduction and breeding procedures is an essential prerequisite to diagnosing reproductive problems and offering sensible advice as to how to deal with them in a cost-effective framework.

Normal reproduction

The sow

Puberty

Puberty occurs at 5–6 months. Gilts are commercially bred at 7 months to avoid the small litters that are produced by younger animals. The onset of puberty is encouraged by presence of a boar nearby. Puberty occurs earlier in loose-housed gilts than in those that are closely confined. The sow is a polyoestrous animal, cycling all year round in moderate climates.

Breeding life

Fertility reaches a maximum at about the fifth litter and falls steadily after that time. Culling of sows after their fifth litter may be cost-effective.

Oestrous cycle

In most sows, oestrus occurs every 21 days, and 95% of sows cycle at intervals of 18–24 days. Sows that come back into oestrus 25–30 days after service may have suffered early and total embryonic death in their litter. Oestrus occurs occasionally in pregnant sows. It is also seen in some lactating sows, especially those in group rearing systems. Serving at such times is not recommended. In many sows, an anovulatory oestrus is seen 2 days after farrowing. Sows normally return to oestrus 3–4 days after weaning and are usually served at this time. It is now thought best to wean pigs at 5–6 weeks to ensure good display of oestrus and subsequent fertility, which may not be seen with 3-week weaning.

Oestrus

The sow stands to be mated and will not move when pressure is put on her back. She often emits a characteristic short repetitive grunt. She is described as being 'hogging' or ' storing' and ready to breed. The sow in oestrus stands still in the presence of a boar in response to his pheromone and if sprayed with synthetic pheromone. Her vulva is enlarged and reddened. The average length of oestrus is 50 h – this long period of oestrus is an important factor in the mating protocol.

Gilts have a shorter oestrus period, at 6–36 h, than older sows. Oestrus is preceded by pro-oestrus; vulval swelling may be seen, and the boar shows some interest and may attempt mounting. Ovulation occurs 36–44 h after the beginning of oestrus.

Synchronization of oestrus

Altrenogest, a synthetic progestogen, is available. Gilts are given 5 mL (20 mg) per day in the food for 18 days and come into oestrus 5–7 days after the last dose.

The porcine corpora lutea are refractory to prostaglandin F2α until 11 days after ovulation, and for this reason the drug is not used for synchronization.

Mating

Practical aspects of mating

The boar may be brought to the gilts and sows or may be allowed to run with them. Care must be taken that bullying, fighting, and injury do not occur. On most units, 'hand mating' is practised so that good service is observed. Better breeding results are obtained using this method. The gilt or sow is usually served again 24 h later if still in oestrus.

A different boar or artificial insemination (AI) may be used for the second service. Slightly improved conception rates are reported. However, the use of two boars can mask infertility of one animal or increase the risk of venereally transmitted disease.

Ninety per cent of the ova released are fertilized in the oviducts. About 30% of fertilized eggs are lost from the uterus, especially at days 12–18 of pregnancy. It has been suggested that a sow needs more than four embryos for pregnancy to be maintained. However, it is not uncommon in gilts to produce a single piglet at farrowing.

Implantation of embryos

The epitheliochorial placenta of piglets attaches to the endometrium at about day 15 of pregnancy.

Fetal age

Fetal age can be estimated from the following formula:

$$\text{Fetal age} = 3 \times \text{crown–rump length (cm)} + 20 \text{ days}.$$

The corpus luteum is the sole source of progesterone in the pig, and none is produced by the placenta.

Progesterone requirements

At least 6 ng/mL of plasma progesterone is required for the maintenance of pregnancy. There is a risk of abortion if plasma progesterone falls below this level.

Pregnancy diagnosis

This is essential for good management.

- *Non-return to oestrus* may be used to indicate pregnancy. Oestrus detection must be very good for this method to be reliable. Sows must be actively exposed to the boar daily to reliably detect oestrus in non-pregnant animals.
- *Ultrasound*. A B-mode scanner used via the flank is probably the best method of pregnancy diagnosis, but there is a risk of damage of the instrument and its leads by pigs. In pregnant animals (30 days +), a honeycomb appearance in the uterus, with piglets in 'cells' formed by the long chorioallantois, is visible. A *Doppler scanner* has 90% accuracy after 18 days of pregnancy. The instrument, often attached to headphones, is widely used to confirm pregnancy by detecting the characteristic sounds of fetal movements, uterine artery enlargement, and fetal heartbeats from 21 days of pregnancy.
- *Rectal examination* enables detection of the enlarged uterus or typical spiral of the cervix in non-pregnant sows at 21 days after service. Middle uterine artery fremitus is palpable from 35 days. The operator requires small hands, and the technique is seldom used.
- *Vaginal biopsy*. Two layers of epithelial cells are found in the vaginal wall of pregnant animals. The sow in or near oestrus has 9 or 10 layers of cells. An accurate service date must be available to allow the biopsy to be taken at exactly 18 days of pregnancy. Histological analysis is required, and the technique has been replaced by ultrasonography.

Normal birth in the sow

Gestation length in sows is in the range of 111–119 days, with an average in most herds of 115 or 116 days. Although some sows are allowed to farrow naturally, induction of birth (for details see below) is practised on many units.

Pregnancy diagnosis is performed routinely on most farms, and sows are moved into their farrowing quarters 2–3 days before their due date. On some units, sows are washed to remove bacteria such as *Staphylococcus hyicus* and ascarid eggs from the skin before being moved into their cleaned and rested accommodation. Mammary development and abdominal enlargement are noticeable in the last third of pregnancy, and fetal movement can be seen beneath the flanks of the resting sow.

Some relaxation and lengthening of the vulva occurs in the last few days of pregnancy. Milk can be expressed from the teats 24 h before farrowing.

Stages of labour

In the *first stage of labour*, the sow often appears restless and experiences mild discomfort, probably associated with uterine contraction. If she has bedding available, she will spend much time chewing it. Her ability to arrange it into a nest is restricted if she is confined in a farrowing crate. This stage lasts 12–24 h.

Fetal delivery occurs in the *second stage of labour*, which is completed in an average of 2.5 h. Abdominal straining does not appear to be very intense, and the sow usually remains in lateral recumbency. Many sows pass their ovoid-shaped piglets with apparent ease, although more intense straining may be seen in gilts farrowing for the first time. The delivery of each piglet may be preceded by tail swishing and the passage of a small amount of allantoic fluid. Approximately half the piglets are born in anterior presentation, and the same percentage are still enclosed within their amnion at the time of delivery. Piglets are born at intervals of approximately 15 min.

Placental delivery occurs during the *third stage of labour*, which is usually completed within 0.5–1 h after the birth of the last piglet. In many cases, the sow passes the placentae of all her piglets after the last fetus is born. Occasionally, the placentae are passed after each piglet or after a batch of piglets have been delivered.

Supervision of birth

Sows should be closely watched without disturbing them to ensure that there are no signs of dystocia and that the piglets are safe. The piglets are encouraged to take colostrum from the sow as soon after birth as possible.

Synchronization and induction of birth in sows

Batch farrowing has become increasingly popular in pigs. It enables farm labour to be concentrated at times when it is needed, such as at farrowing and weaning. It also allows an all in, all out system to be used in each farrowing house. A batch of sows enter the farrowing house, farrow at about the same time, wean their piglets, and move out of the house together. The house is then thoroughly cleaned, disinfected, and rested before the next batch of sows is brought in.

Induction of birth can be used to synchronize the time of farrowing of a group of sows. If the sows farrow at about the same time, their piglets can be weaned at the same time. The sows return to oestrus after weaning and are served at about the same time. They will then be ready to farrow again at about the same time. Not all sows will come into oestrus or conceive to their first service at the same time. Some members of the batch may drop back into a following batch or, in some cases, are culled. When batches of sows are induced, they should have all been served within 2–3 days of each other.

The stillbirth rate of piglets in cases of dystocia may rise to 19% from the rate of 3–6% that is seen in normal farrowings. Supervision of birth, early recognition and treatment of dystocia, and care of the newborn have been shown to reduce total mortality from birth to weaning by 5%.

Induction of birth has been shown to be effective in causing most sows in a unit to farrow during the daytime, when labour is available. Any delays or other problems observed during farrowing can be investigated and dealt with immediately. Newborn piglets can be managed to ensure that they take colostrum as soon as possible after birth and are protected from damage either by the sow or from the environment.

Management must be of a high standard if the benefits of induction of birth are to be achieved. Good records of service must be maintained and sows clearly identified so that the right animals are batched for induction.

Sows should be induced only when they are within 2 days of their calculated farrowing date. It is recommended that sows should not be induced if there is evidence of either porcine reproductive and respiratory syndrome (PRRS) or swine influenza on the farm. These and other conditions may result in the birth of weak piglets, and this may be exacerbated by induction.

The use of prostaglandin F2α

Induction of birth is achieved by the injection of either synthetic prostaglandin F2α or an analogue such as cloprostenol or luprostiol. The drug is given by intramuscular injection 1–2 days before the anticipated farrowing date. The drug has also been given in half-dose aliquots subcutaneously into the medial walls of the vulva. Gestation length in the sow is normally 115 days but may be longer on some farms. Most sows commence farrowing 20–30 h after receiving their prostaglandin injection. The drug should be given during the period early to late morning, thus anticipating that most sows will farrow during the following working day.

The use of oxytocin

It has been suggested that oxytocin might be used in two ways to aid the efficiency of induction and in particular to hasten the time of fetal delivery.

- Administration of oxytocin to any sow that has not commenced farrowing 24 h after the administration of prostaglandin – provided that the cervix is found to be open on vaginal examination.
- Administration of oxytocin once the induced farrowing has commenced.

Oxytocin should always be used with caution and only if the clinician or trained attendant is sure that no obstructive form of dystocia is present. Ideally, a careful vaginal examination should always be performed before its use.

Dystocia in the sow

Porcine dystocia is relatively uncommon, occurring in 0.25–1.0% of all farrowings. When dystocia does occur, however, it may result in a large increase in the incidence of stillbirth in the litter. As might be anticipated in a polytocous animal like the sow, uterine inertia is the most frequently observed cause of dystocia.

Causes of dystocia

In a survey of 200 cases of sows suffering from dystocia attended by a practising veterinary surgeon, the causes identified were as shown in Table 10.1.

Uterine inertia

Three forms of uterine inertia are recognized in pigs.

1. *Primary uterine inertia*. Two forms have been recognized depending on the living state of the litter.

Table 10.1 Causes of dystocia in pigs

Cause of dystocia	Percentage
Uterine inertia	37.0
Fetal maldisposition	33.5
Obstruction of the birth canal	13.0
Deviation of the uterus	9.5
Fetopelvic disproportion	4.0
Maternal excitement	3.0

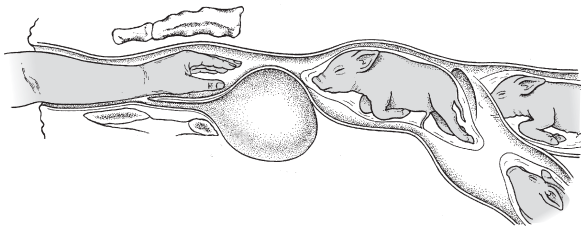

Fig 10.1 – Dystocia: obstruction of birth canal caused by distension of the urinary bladder. (Redrawn from Jackson PGG. Handbook of Veterinary Obstetrics, 2nd edn. Edinburgh: Saunders. © 2004 Elsevier.)

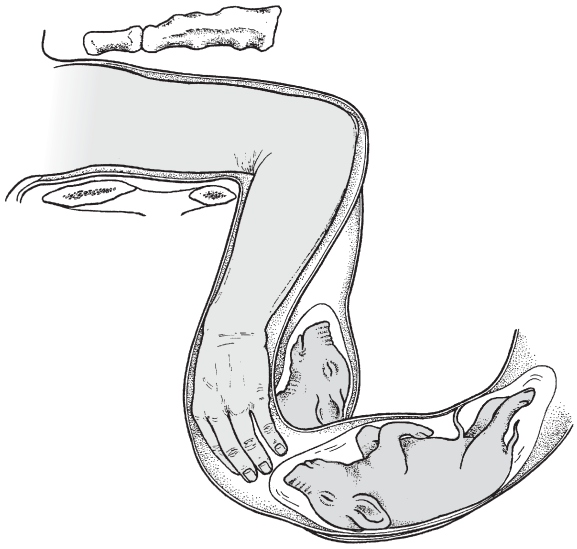

Fig 10.2 – Dystocia: deviation of the uterus. (Redrawn from Jackson PGG. Handbook of Veterinary Obstetrics, 2nd edn. Edinburgh: Saunders. © 2004 Elsevier.)

- When the litter is normal, the sow is ready to farrow but birth does not occur. The cause of the condition is unknown, but there may be lack of oxytocin receptors in the myometrium. The condition responds well to oxytocin.
- When the litter is dead and the sow toxaemic, fetal death may be as a result of an infectious agent such as parvovirus. The prognosis is very grave, and euthanasia may be required.

2. *Idiopathic uterine inertia*. In this condition, which has also been termed *partial primary inertia*, farrowing starts normally but then stops. Uterine contractions have ceased, and further piglets remain within the uterus. The condition is responsive to oxytocin.
3. *Secondary uterine inertia*. Uterine contractions cease as a result of another initial cause of dystocia such as fetal maldisposition. The condition is treated by removing the primary cause and using oxytocin to initiate uterine contractions.

Obstruction of the birth canal

This may be caused by abnormalities of either soft or bony tissues, the former being the more common. The most common cause is distension of the urinary bladder, which presses on the uterine lumen, obstructing it when the sow strains (Fig. 10.1). The condition is treated either by encouraging the sow to stand up and urinate or by emptying the bladder by catheterization. Other causes of obstruction, such as a persistent hymen, may be less readily resolved and caesarean section may be required.

Deviation of the uterus

This condition is seen chiefly in older sows with a deep conformation. In such animals carrying a large litter, the uterus may deviate sharply downwards just anterior to the pelvis, developing tight bends that the fetus is unable to negotiate (Fig. 10.2). Manual delivery of the first few piglets permits the uterine horns to straighten and farrowing to resume.

Maternal excitement

This condition is seen chiefly in gilts and especially those that have been moved into their farrowing crate just before going into labour. The affected animal may be very disturbed and struggle to escape. Farrowing ceases, and sedation is achieved by administration of azaperone by intramuscular injection at a dose of 2 mg/kg of body weight. Following sedation, farrowing normally recommences. An injection of oxytocin can be given if there is any evidence of reduced uterine contractions. In some cases, maternal excitement may lead to litter savaging (please see below).

Fetal maldisposition

The short neck and limbs of the piglet render it less likely to become maldisposed during birth than the lamb or calf. The most common cause in this category is the simultaneous delivery of twins. Individual piglets may become wedged in the uterine body and occasionally within the horns (Fig. 10.3). Manual correction of the problem is usually possible.

Fetopelvic disproportion

This relatively uncommon problem can occur if the litter size is very small with large individual piglets. Delivery by caesarean section may be necessary if manually assisted delivery is not possible.

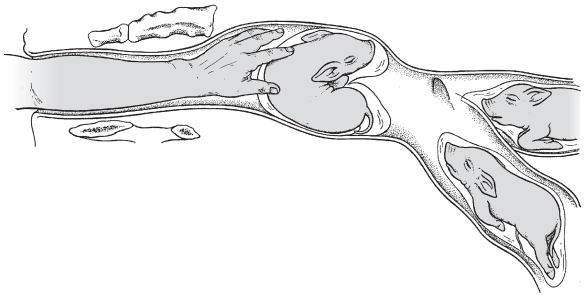

Fig 10.3 – Dystocia: fetal maldisposition in the uterine body. (Redrawn from Jackson PGG. Handbook of Veterinary Obstetrics, 2nd edn. Edinburgh: Saunders. © 2004 Elsevier.)

Fig 10.4 – Dystocia: vaginal examination. (Redrawn from Jackson PGG. Handbook of Veterinary Obstetrics, 2nd edn. Edinburgh: Saunders. © 2004 Elsevier.)

Signs of dystocia in the sow

The following signs may suggest that dystocia is present. They indicate that a full gynaecological examination should be performed:

- straining for 5–10 min without delivering a piglet,
- farrowing fails to commence despite signs of imminent birth,
- farrowing ceases after the delivery of one or two piglets only,
- farrowing is prolonged beyond the average duration of second-stage labour (2–3 h),
- the sow has not passed any placenta, or
- there is a foul-smelling vaginal discharge.

Management of a case of dystocia in the sow

Most porcine obstetrical work is performed by skilled farm staff with veterinary help being called to particularly difficult cases. The aim of any obstetrical intervention is to investigate and if possible resolve an abnormality as soon as it is observed and to assist the sow complete farrowing as soon as possible.

The sow suffering from dystocia should be given a clinical examination to ensure that she has no concurrent disease and in particular that she has no mammary abnormality. Hyperthermia can occur if heat lamps are in use. Body temperature should be normal or slightly depressed during farrowing. The number of piglets already born should be noted and their viability assessed.

A *vaginal examination* must be undertaken with great care. The sow should be encouraged to remain in lateral recumbency. Her vulva is washed with a mild antiseptic solution. The operator's hands are carefully washed before the generous application of obstetrical lubricant. Gloves are not usually worn and no special instruments are required.

It is normally possible to pass the hand gently forwards along the vagina (Fig. 10.4), through the uterine body, and as far as the bifurcation of the uterine horns. As the hand is passed forwards within the birth canal, it passes through the bony pelvis. This is normally achieved with ease, although it may be difficult in some young gilts in which the internal pelvic measurements are small.

Palpation of the inner surface of the uterine horns will allow an assessment of their muscular tone. If normal good tone is present, the uterus feels very hard and rigid to the touch. If abnormally poor tone is present, the uterine walls feel soft and flabby. In cases of uterine inertia, the horns have poor tone and digital manipulation of them does not normally induce an increase in their tone.

If the cervix is closed, it can be palpated just anterior to the bony pelvis, and the uterine horns are not accessible. When the cervix is open, it is possible to explore only the proximal parts of the uterine horns. The distal parts of the uterine horns are inaccessible per vaginam. Sows will normally not permit external abdominal palpation, but if expense permits the viability of the fetuses can be assessed ultrasonographically through the maternal flank.

In some cases of dystocia, one or more piglets may be encountered within the birth canal. Careful digital palpation should determine if their presentation, position, and posture are normal. The operator's fingers should never be placed into the mouths of unborn piglets in case a bite from the sharp incisor teeth is sustained.

Any piglets within the birth canal should be individually delivered by traction, gently easing them through the birth canal towards the vulva (Fig. 10.5). Any amniotic tissue should be removed from each piglet. Fluid from the pharynx should be removed by holding the piglet up by its hind legs to allow drainage. If still attached to the chorioallantois, the umbilical cord of each piglet should be severed by traction 6–10 cm from the piglet. The proximal cord should be knotted or the end dipped in a strong

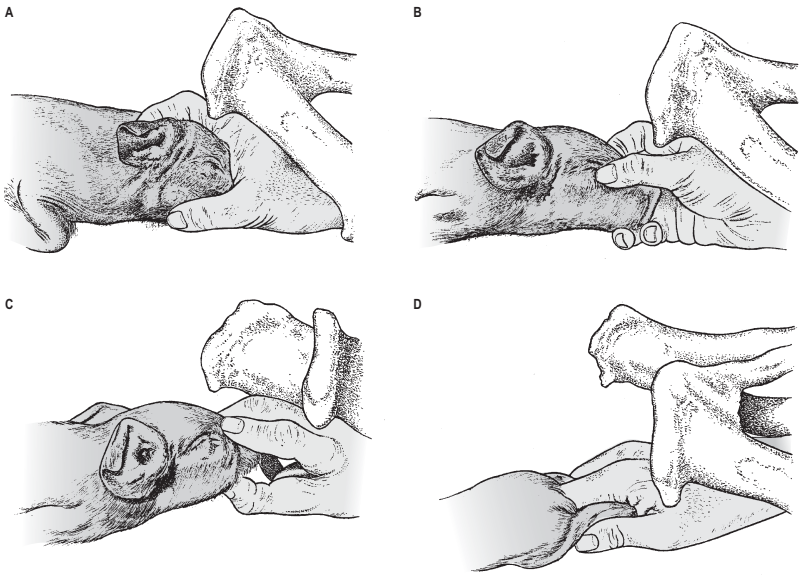

Fig 10.5 – Dystocia: fetal delivery – piglet holds. (Redrawn from Jackson PGG. Handbook of Veterinary Obstetrics, 2nd edn. Edinburgh: Saunders. © 2004 Elsevier.)

iodine solution in an attempt to reduce the risk of infection gaining access to the body via the umbilicus.

Once all palpable piglets have been removed, the uterine tone can be assessed. If this is thought to be deficient, an injection of 20 IU of oxytocin should be given by careful intramuscular injection. The injection is painful and may cause a nervous animal to leap to her feet. This can be avoided by using a fine needle introduced carefully through the skin. The sow's attention can be diverted by gently massaging her udder, which will normally ensure that she remains in lateral recumbency.

A further vaginal examination should be performed 10 min after administration of oxytocin to check if further piglets have been brought towards the pelvis by the uterine horns. These should be manually delivered.

If the palpable uterine tone remains good, no further oxytocin should be given at this stage. The sow's progress should be monitored and a further internal examination should be performed if there is any doubt about the progress of the case. In cases of uterine inertia, a number of further doses of oxytocin may be required.

In cases of obstructive dystocia, such as the simultaneous presentation of two piglets, farrowing often resumes (if uterine tone is satisfactory) after removal of the piglets.

The end of farrowing

The length of the uterine horns prevents the clinician exploring the entire uterus. Cessation of straining, the passage of dark red areas of chorioallantois, and the sow rising to empty her bladder and then feeding her piglets are all signs suggesting that she may have delivered all her piglets. None is entirely reliable. It may be possible to detect further piglets by internal ballottement (Fig. 10.6) and by ultrasonographic scan. Detailed abdominal palpation is not tolerated by the sow. Cervical closure is a firm indication that farrowing is complete, and the closed cervix can be readily palpated per vaginam (Fig. 10.7).

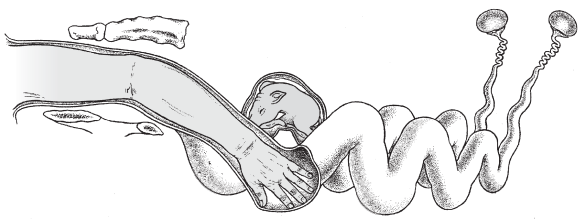

Fig 10.6 – Dystocia: internal ballottement. (Redrawn from Jackson PGG. Handbook of Veterinary Obstetrics, 2nd edn. Edinburgh: Saunders. © 2004 Elsevier.)

Indications for caesarean section

The majority of cases of porcine dystocia can be treated by manual delivery and the use of ecbolics. Caesarean section may be indicated in a few cases, but the cost of surgery may not be economically justified and must be discussed with the owner.

Specific indications for caesarean section are as follow.

- Uterine inertia cases that are not responsive to ecbolic therapy.

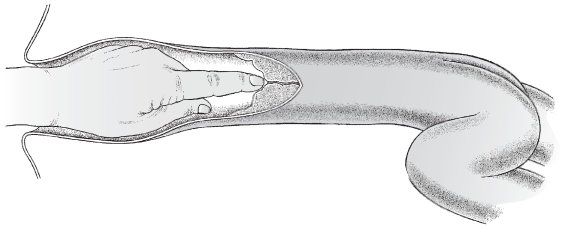

Fig 10.7 – Dystocia: closed cervix indicating completion of farrowing. (Redrawn from Jackson PGG. Handbook of Veterinary Obstetrics, 2nd edn. Edinburgh: Saunders. © 2004 Elsevier.)

- Fetopelvic disproportion: fetal size is very large or the pelvis is naturally narrow or has been damaged by previous fracture.
- Vaginal and rectal prolapse with severe mucosal damage causing delivery per vaginam to be difficult or liable to cause further damage.
- Severe damage to the genital tract following attempted fetal delivery by laypersons.
- As an alternative to hysterectomy in the production of gnotobiotic piglets.

For information on anesthesia and surgical technique for caesarean section, please see Chapter 15 (*Analgesia, anaesthesia, and surgical procedures in the pig*).

Aftercare of the sow

It is advisable to prescribe a 3-day course of antibiotic such as a combination of penicillin and streptomycin in sows in which assisted delivery has been required. The vaginal and uterine mucosa may have sustained superficial injury, and repeated vaginal examination may increase the risk of infection. Ideally, the sow should be allowed some daily exercise after the completion of farrowing. This can be very difficult to arrange in many units. Ad lib water must be available, and a light laxative diet is recommended for the first 3 days after farrowing. Excessive feeding may predispose to mastitis–metritis–agalactia. For further details, see Chapter 9 (*Diseases of the urogenital system and the udder*).

Litter savaging by the sow or gilt

This condition is seen occasionally in sows and more commonly in gilts farrowing for the first time. An affected animal seems frightened by her own offspring and will snap at and kill any piglet that comes within reach of her head. If unattended, the whole litter may be killed in this way.

Treatment involves immediate retrieval of any piglets already born and their placement in a warm box until farrowing is complete. The sow or gilt is then sedated or anaesthetized before her piglets are reintroduced. Sedation is achieved by administration of acepromazine at a dose of 0.5 mL of a 10 mg/mL solution by intramuscular or intravenous injection. The sow is left quietly for 15 min before the piglets are carefully reintroduced. Between 10 and 20 IU of oxytocin may be given by intramuscular injection to encourage milk let-down. Azaperone is a less effective sedative for this condition. In some cases, it may be necessary to administer a general anaesthetic using pentobarbitone given by intravenous injection.

Postparturient problems in the sow

Postparturient recumbency

Sows often stand to urinate after the completion of farrowing. They also stand to feed but often lie for long periods in sternal recumbency.

When feeding their piglets, they lie in lateral recumbency. Older sows with a 'deep' conformation may find it difficult to get to their feet within the confines of a farrowing crate. Such animals should be assisted to rise by opening the crate and raising the hindquarters using the tail. Outside exercise is very beneficial in such animals, which should be accommodated in a pen rather than a farrowing crate.

Retention of the fetal membranes

The fetal membranes are normally passed either individually in small batches or after delivery of the whole litter. Retention of the fetal membranes is uncommon but may be caused by the presence of a retained fetus. In cases in which the fetal membranes have not been passed, a vaginal examination should be performed to check for the presence of one or more retained piglets. These should be removed, and an injection of oxytocin (20 IU) is given by intramuscular injection. The induced myometrial activity should result in the passage of the fetal membranes. Any remaining piglets should move towards the uterine body, from where they can be delivered manually.

Vaginal prolapse

This condition is seen in both preparturient and postparturient sows. For details of clinical signs and treatment, please see Chapter 15 (*Analgesia, anaesthesia, and surgical procedures in the pig*).

Uterine prolapse

This is a relatively uncommon condition associated with a high mortality rate.

One or both uterine horns are involved. The condition usually occurs in the early postparturient period, but occasionally one empty uterine horn is prolapsed before farrowing is complete. In many cases, death as a result of sudden haemorrhage occurs following rupture of an

ovarian artery as the prolapse occurs. For details of clinical signs and treatment, please see Chapter 15.

Acute septic metritis

A mild metritis is seen in sows suffering from mastitis–metritis–agalactia and normally responds well to treatment. Acute severe septic metritis is a life-threatening condition.

Incidence

The condition is relatively uncommon but has a grave prognosis.

Aetiology

It may arise from fetal death in the uterus as a result of parvovirus or another infection (see earlier discussion of primary uterine inertia). It can also occur as a result of infection gaining access to the uterus during obstetrical procedures or in the postparturient period.

Clinical signs

These are seen within 48 h of farrowing. The sow is initially pyrexic (temperature 40–41°C), anorexic, and reluctant or unable to stand. A foul-smelling vaginal discharge is present, and some patches of skin discoloration may be seen in pale-coloured sows. Vaginal examination – which must be carried out with great care using a gloved hand – reveals that the vaginal and any palpable uterine mucosa are thickened, inflamed, and dry. Remnants of fetal membrane may be present, and it is possible that a dead bloated fetus may also be palpated. Further detailed vaginal examination should not be performed in case additional damage is done to the already compromised tissues.

Body temperature may fall, and the sow becomes toxaemic. She is dehydrated and her visible mucous membranes are dry and injected.

Diagnosis

This is based on the history of the animal, the clinical signs, and the findings on vaginal examination.

Treatment

Treatment may not be economically viable in the severely ill animal, in which euthanasia may be required. If treatment is a viable option, a catheter should be placed in an ear vein. Fluid therapy with normal saline is commenced, and intravenous antibiotic (such as soluble ampicillin) and non-steroidal anti-inflammatory drug therapy is given. Intrauterine antibiotics are not normally used, as their duration of action is short and their administration may cause distress to the sow and further tissue damage. Any fetus or fetal membranes within reach should be carefully removed, aided by generous lubrication. Nursing care – the provision of a warm pen, generous bedding, and ad lib water and food – should be provided. The sow should be encouraged to move and, if possible, moderate exercise is very beneficial.

Prognosis

This is poor. Response to treatment should be evident within 24 h if recovery is to be anticipated.

Agalactia

This condition, which can be caused by a number of factors including mastitis, is discussed in Chapter 9 (*Diseases of the urogenital system and the udder*).

The boar

Puberty

This occurs at approximately 5–6 months of age. The first service may be permitted at about 7 months. In commercial herds, the boar may have been performance-tested, including a food conversion assessment. Following such testing, the boar may be too thin to work and may need to be allowed to gain weight before use. The testes should have fully descended by birth, although monorchidism is not uncommon. The condition is unacceptable in a breeding boar.

Accessory glands

The boar has a small prostate, two large saccular seminal vesicles, and two large bulbourethral glands. In large boars, the prostate and other structures can be palpated per rectum.

Prepucial diverticulum

Present in all males, the diverticulum contains a large volume of foul-smelling fluid. It is easily mistaken for an abscess or umbilical hernia. The diverticulum is bilobed and has an entrance in the dorsal wall of the prepuce just within the opening. Ulceration of the mucosa of the prepucial diverticulum has been reported and in valuable animals may necessitate removal of the affected mucosa. Magnesium, ammonium phosphate (struvite) stones have been very occasionally found in the prepucial diverticulum. See also Chapter 15 (*Analgesia, anaesthesia, and surgical procedures in the pig*).

Pseudohermaphrodites

Pseudohermaphrodites are frequently seen with male external genitalia but with developed uterine horns. Testes or ovaries may be present. Five per cent of all piglets born may be intersexes – the condition may have a hereditary predisposition. True hermaphrodites with both an ovary and a testis are very uncommon.

Frequency of use

A mature boar can complete 10 services per week, i.e. five sows each having two services. Young boars should be gradually introduced to work.

Semen parameters

Volume should be 250 mL. There are three fractions: presperm, sperm-rich, and the gelatinous postsperm fraction, whose function may be to plug the cervix. It has been suggested that a boar needs large seminal volume to assist the passage of spermatozoa through long uterine horns. Satisfactory parameters of density are as follow.

- Density: 100 million spermatozoa/mL.
- Progressive motility: >70%.
- Live and normal spermatozoa: >70%.

Semen collection

This is done mostly by manually holding the corkscrew tip of the penis of the stimulated boar tightly in a lubricated gloved hand and directing semen into a warmed container. Boars at AI stations are trained to mount a dummy sow.

Artificial insemination in pigs

This is widely used but restricted by the difficulty in freezing porcine semen. Although freezing of boar semen is possible, the results of AI are inferior to the use of extended semen, and there is great variation in the freezing ability of semen between boars. Most commercial AI units use non-frozen extended semen. Insemination can be either by inseminator service or by extended semen distribution service. The latter is most common in the UK, but inseminator service is widespread in Europe.

Semen collection is as described above, using dummy sows. Semen is collected from donor boars at 5- to 7-day intervals or as required.

Semen handling

The gelatinous postsperm fraction is filtered off. Semen is evaluated and diluted to produce doses for insemination of 1000–2000 million spermatozoa in 70 mL of diluent. Diluted semen is said to maintain reliable fertility for 3 days. Seventy millilitres of extended semen is required to provide sufficient fluid volume entering the uterus to stimulate uterine contractions, which aid semen transport.

Insemination technique

Inseminate when the sow is in standing heat (see above) using a rubber spiral or plug catheter passed into the sow's cervix via the vagina and locked by rotating in an anticlockwise direction. Semen in a plastic bottle is attached to the end of the catheter, and semen is allowed to run in by gravity. A second insemination is performed 24 h later.

Regarding the results of AI using extended semen, one should achieve a 65–75% conception rate and 9–11 piglets per litter following double insemination.

Infertility

Although problems seen in individual animals – especially for example in an expensive new boar – are important, porcine infertility is usually considered as a herd problem. Individual infertile or subfertile sows are mostly culled. Follow-up post-mortem reproductive pathology may be important in identifying a herd problem.

Infertility in the sow

Seasonal fertility

Fertility levels may fall in very hot ('August infertility') and in extremely cold weather, especially if feeding is substandard or of marginal quality. Hot weather may also increase incidence of early embryonic death. Feeding is also depressed and can lead to low sow weights at weaning, with resultant difficulty of getting the sow in pig again. The adverse effects of high temperature in outdoor herds can be reduced by providing shade and wallows for the sows. In anticipation of August infertility, the farmer may serve extra gilts to maintain production in preparation that some may fail to conceive. For further discussion of seasonal infertility in outdoor pigs, please see Chapter 12 (*Outdoor pigs and organic herds*).

Functional infertility

Anoestrus

This can be an important and quite common problem that may cause severe disruption of the breeding programme. The incidence of anoestrus may be higher in summer. The condition is a greater problem in gilts than in sows.

The method(s) of oestrus detection should be monitored to ensure that it is satisfactory. Is there really an anoestrus problem or are heats being missed? Weekly plasma progesterone profiles can be taken to confirm whether ovarian activity is occurring in problem groups. Some workers claim to be able to monitor ovarian changes by ultrasonographic examination through the flank.

Causes of porcine anoestrus are as follow.

- Lack of boar stimulation: sight, smell, and contact – the most important cause?
- Gilts are prepubertal – too young to cycle.
- Poor nutrition, including inadequate trough and drinking space and poor food quality.
- Harsh weather conditions: very cold or very hot.
- Poor accommodation, including inadequate light.
- Poor sow identification.
- Large groups and badly matched groups, allowing bullying.
- Lameness: sows are reluctant to stand and show oestrus.
- High parasite burden or other insidious illness.
- Genetic predisposition.
- Ovarian aplasia: this is rare.
- Cystic ovarian disease: the exact incidence is unknown. It may be more likely after early weaning. Affected sows may show prolonged or irregular oestrus.

Investigation of anoestrus

Full appraisal of the extent of the problem, the groups involved, and elimination of the above causes are needed.

Treatment

Elimination of causes and general improvement in management.

Use drug therapy: a combination of equine chorionic gonadotrophin (eCG) and chorionic gonadotrophin. Use a freeze-dried preparation with 400 IU of eCG and 200 IU of human chorionic gonadotrophin. Given as 5-mL intramuscular injection to anoestrus gilts or sows, heat normally follows in 5 days. It can be used 'prophylactically' at weaning (within 48 h) to 'encourage' postweaning oestrus. If an animal has CL, no response will be seen – advise repeat injection in 10 days.

For poor oestrus detection, investigate and if possible improve the methods used.

Failure of conception

One may have a complete failure of conception (no piglets born) or a partial failure (small litter). Possible causes include the following.

Mistiming of service

It is claimed that a single correctly timed service should ensure conception, but selecting the correct time for service is difficult under farm conditions. Boar spermatozoa can survive up to 40 h in the female genital tract, but ova must be fertilized within 8 h of ovulation. Ovulation occurs 36–44 h after the beginning of oestrus. The best practical regimen is to serve on the first day of standing heat and again 24 h later.

Unsatisfactory service

This could result from an inexperienced boar failing to achieve intromission, or there could be a badly designed service area. There could be failure to observe unsatisfactory services or a boar with poor libido. Lameness or other illness may prevent service.

Poor semen quality

There could be inadequate volume, motility, or count, or a low percentage of live and normal spermatozoa. One or all of these parameters may be deficient. Semen samples should be evaluated from problem boars as necessary.

Investigation of poor conception

A full appraisal of records, service management, and individual boar fertility records is required. The records may suggest that one age group (e.g. gilts) is affected, or one boar, or that one attendant is associated with the problem. Have there been changes in the management of service, staffing levels, feeding, ill health, etc.? Discuss possible causes with farm staff: have they noticed any possible cause, or has there been a change in management? Also investigate the extent of the problem: are sows really not conceiving or is there evidence of early embryonic death (e.g. by irregular return to oestrus)? In particular, check the following.

- Timing and management of service: supervision and repeat service protocol.
- If AI is used, is the semen being handled correctly, and is the service procedure satisfactory?
- Condition of sows and boars: feeding, accommodation, and flooring.
- Is there any suggestion of infectious disease on the farm, especially among breeding stock? Infectious problems are less likely than management problems.
- Oestrus detection: methods and efficiency (see also above).
- Pregnancy diagnosis: methods and efficiency.

Infectious causes of infertility and abortion in pigs

Please note that some of these conditions are also discussed in Chapter 11 (*Polysystemic diseases*). Only the reproductive aspects of these conditions are considered in this chapter.

A wide range of infectious agents can have an adverse effect on porcine fertility, and their severity may be increased by poor management practices on the farm.

The organisms involved may be opportunist commensals such as erysipelas or specifically associated with infertility, for example porcine parvovirus and *Brucella suis* (not in the UK). Fungi and bovine viral diarrhoea virus

Table 10.2 Veterinary Investigation Diagnosis Analysis sample submissions: fetopathies, 1996–2003

Disease	Incidence (%)
No diagnosis	68.48
Leptospirosis	8.84
Parvovirus	7.09
Others	6.74
Erysipelothrix rhusiopathiae	3.24
Streptococci	2.80
Porcine reproductive and respiratory syndrome	1.75
Pasteurella	0.70
Escherichia coli	0.26
Listeria monocytogenes	0.09

are among uncommon organisms occasionally encountered in infertility cases.

As with other species, infection should be suspected in outbreaks of reproductive failure until eliminated from the enquiries. Fresh samples of placenta and piglets should be submitted to the Veterinary Laboratories Agency (VLA) to establish whether infection is involved in an abortion outbreak. In the majority of cases, it is not possible to determine the cause.

The relative incidences of the VLA diagnoses of infectious causes of reproductive failure in pigs are given below.

Veterinary Investigation Diagnosis Analysis sample submissions: fetopathies, 1996–2003

See Table 10.2.

Leptospirosis

Aetiology

Leptospira canicola, *L. icterohaemorrhagiae*, and *L. australis* are most common in the UK.

Incidence

Leptospirosis occurs worldwide. *L. pomona* is not found in the UK. The incidence of *L. bratislava* is increasing in outdoor herds.

Epidemiology

Infection is carried by rodents or carrier pigs. *Leptospira* organisms can survive in damp conditions and in the soil for 14 days. Infection is by ingestion, inhalation, venereal spread, or via cuts on skin. The organism persists in the kidney and is shed in urine.

Clinical signs

The clinician may see signs of acute infection (e.g. pyrexia, anorexia, and jaundice). A pig may be a subclinical carrier. Specific reproductive signs include abortion in late pregnancy, increased stillbirth, and mummified and macerated fetuses.

Diagnosis

Serology in the sow and possible isolation of organism in the fetus are used.

Treatment

A single dose of dihydrostreptomycin at 25 mg/kg (i.m.) repeated in 14 days is claimed to eliminate the carrier state. Penicillin, oxytetracycline, and tiamulin are also effective. Control is by high standards of cleanliness, controlling vermin, and maintaining a closed herd. Vaccines against some serotypes are available and may reduce the incidence of abortion in infected herds. In the UK, killed vaccines are available against *L. canicola* and *L. icterohaemorrhagiae* but are not licensed for use in pigs.

Porcine parvovirus

Aetiology

The cause is a small single-stranded DNA virus.

Incidence

The virus is found worldwide and is endemic in many UK herds.

Epidemiology

Oral, nasal, and venereal spread is possible. The virus may be introduced by an infected sow or boar. Postfarrowing discharges in infected sows are very rich in virus and highly infective. Subclinical viraemia in the sow or gilt follows infection, and the virus crosses the placenta 14 days later. Fetuses <70 days are immunoincompetent and usually die and are aborted or resorbed. Older fetuses are mummified or may be born stunted and weak.

Clinical signs

The clinician occasionally sees early embryonic death and irregular return to oestrus but mostly small litters with mummified and stillborn piglets (Fig. 10.8). Piglets that survive are weak and ill thriven. Note that one may also see suppression of spermatogenesis in boars.

Diagnosis

Base the diagnosis on history and clinical signs, and serology in the sow. Virus isolation from fetuses and fluorescent antibody test in frozen sections of fetal tissues are used.

Treatment

Nothing can be done. The occasional sow may need antibiotic cover.

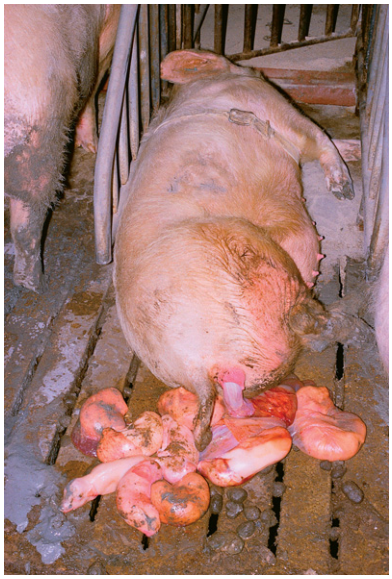

Fig 10.8 – Abortion: litter of dead piglets.

Control

Keep a closed herd. Isolate any aborting sows: do not expose other sows to infected animals or dead piglets if pregnant – they may spread other diseases.

A number of inactivated vaccines are available, providing protection for embryos and fetuses against infection by parvovirus.

SMEDI viruses

SMEDI (the acronym for stillbirth, mummification, embryonic death, and infertility) is a popular term indicating enterovirus infection that caused stillbirth, mummification, and early fetal death in affected sows. Overall signs are very like those of parvovirus infection. SMEDI is thought not to be a common problem now. Note that porcine parvovirus has been sometimes inaccurately categorized as a SMEDI virus.

Porcine reproductive and respiratory syndrome

Aetiology

A virus of the Arterividae family is responsible.

Incidence

The syndrome occurs in the USA and in Europe, including the UK. It is endemic in many herds.

Epidemiology

It is brought in by carrier animals and at service. Airborne spread occurs between farms. Severe signs may occur in naive populations, where a 10% loss in production may occur. Later, the disease becomes endemic.

Clinical signs

Anorexia and fever are found in sows and boars. There is failure to conceive, with regular and irregular return to oestrus. Premature birth occurs, and stillborn and mummified piglets are found. Occasional abortion occurs. There is poor milk production after farrowing.

In the boar, semen quality is reduced for up to 3 months. Piglets are weak, with respiratory signs and high mortality (Figs 11.13 and 11.14). Signs often become less severe when the syndrome is endemic.

Diagnosis

This is based on history, clinical signs, serology, and histopathology.

Treatment and control

Antibiotic therapy may help reduce the risk of secondary infection in postabortion sows. Maintain a closed herd with high health status. A live freeze-dried vaccine is available in some countries, including the UK. Piglets are vaccinated at 6–8 weeks of age.

Infertility in the boar

As in other male animals, the main causes of infertility in the boar can be grouped under three headings.

1. Unwilling to serve – poor libido.
2. Unable to mount and achieve intromission.
3. Unable to fertilize.

Special points relating to the boar follow.

Poor libido

This is seen in young inexperienced boars, especially if they are attacked and frightened by a group of sows or gilts awaiting service. Patience and careful management are required to assist them regain confidence. Most farmers are unwilling to treat a boar if libido is poor. Two injections of chorionic gonadotrophin (500 IU i.m.) twice weekly for 4 weeks with no sexual contact during treatment has been shown to assist restoration of libido in some animals.

Inability to mount

This is often the result of orthopaedic problems, which are quite common in young boars and may be exacerbated by outside management. Infected foot lesions (e.g. 'bumble foot' or bush foot) and hip lesions (such as a slipped epiphysis) are important problems. Diagnosis can be difficult with upper limb lameness unless an x-ray can be arranged and is economically justified.

Inability to extrude the penis

Problems with a lack of erectile tissue in the penis are rare. The prognosis is very poor. In some boars, the penile frenulum may persist after puberty, preventing extrusion of the penis at surgery. The frenulum can be sectioned under anaesthesia. Penile injury can occur as a result of young boars mounting each other or in the case of a boar penned with a number of breeding gilts. Minor injuries often heal with rest, but more severe injuries such as rupture of the penile tunica have a very poor prognosis. In some cases, the injured penis becomes adherent to the prepucial lining. Less commonly, the penis may be trapped as it passes the entrance to the prepucial diverticulum. In both cases, intromission may be compromised. Adhesions may be broken down under anaesthesia but often recur. Removal of the prepucial diverticulum may prevent penile entrapment. For surgical details, please see Chapter 15 (*Analgesia, anaesthesia, and surgical procedures in the pig*).

Inability to fertilize

This is mostly as a result of poor semen quality. Short-term problems may follow parvovirus or PRRS infection. More long-term infertility may occur following PRRS infection – some AI stations check that new boars have negative serology to PRRS. Any acute disease associated with pyrexia may result in temporary suppression of spermatogenesis. Chronic problems may be related to testicular infection (e.g. orchitis), degeneration, and abnormal spermatozoa.

Bleeding at the time of service

This is seen occasionally and is usually related to damage in the glans penis. It is often difficult to find the lesion – sexual rest may be tried as a treatment but is seldom effective. Surgery is possible if the lesion can be identified, but it is often very difficult to locate in the non-erect penis.

Bleeding after service

This can occur as a continuation of preservice bleeding. It can also arise from the sow and in particular from the maiden gilt, in which rupture of the hymen has occurred as a result of service.

Reduction of sperm output

Sperm output is suppressed by hot weather. Poor feeding and management of boars can also result in reduced reproductive performance. There have been reports of interstitial cell testicular tumours in Vietnamese pot-bellied pigs, which may reduce both libido and sperm production.

Other infections found in the male genital tract and that could influence fertility include *Eubacterium suis*, *Actinobaculum* spp., and *Escherichia coli*. The prepucial diverticulum may harbour spirochaetes. It may be advisable to empty the diverticulum manually before collecting semen, but this is difficult to do effectively.

Clinical examination of the infertile boar

History and records

Problems with a boar at service, such as failure to achieve intromission, may be observed at the time of service. In other cases, poor reproductive performance may become evident from service records. On many farms, two boars may be used to serve each sow, and thus records may not directly identify a problem animal.

Observation of service

Service is a slow process and may extend for almost an hour. The early part of service should be observed carefully to ensure that libido is satisfactory. Observation must be carried out in a quiet environment with flooring that allows the boar to obtain good foothold. Young boars may require a little help to achieve intromission. Any signs of difficulty in extruding the penis should be noted, as should the appearance of blood from the penis. Damage to the penis can often be readily observed at the time of service when the penis is fully extruded. Once intromission has been achieved, the boar makes thrusting movements until the penis engages the sow's cervix. Ejaculation commences between periods of apparent somnolence.

Clinical examination

This can be carried out before or after observation of service. The boar is submitted to a full clinical examination to ensure that general health is good and that there are no signs of lameness or orthopaedic damage. The prepuce should be free from any discharge, and the prepucial diverticulum should be fluid-filled but not painful on palpation (Fig. 1.17). Compression of the diverticulum may result in release of foul-smelling fluid.

The penis cannot be extruded in the conscious animal, and it best observed and examined carefully at the time of service. It can be palpated within the prepuce, and evidence of adhesions or rupture of the tunica albuginea can be palpated.

The testes should have the consistency of ripe tomatoes. They should be equal in size and consistency on palpation (Fig. 1.16). They can be further evaluated by ultrasonographic scan.

The accessory glands are seldom involved in disease but if necessary can be palpated per rectum in older quiet or well-restrained animals.

Semen collection and evaluation

This is achieved either in the presence of a sow in oestrus or, if the boar is used for AI collection, by allowing the

boar to mount a dummy sow. As the penis is extruded, the corkscrew-shaped glans penis should be grasped firmly in a warm latex-gloved hand. The penis is pulled gently forwards from the prepuce, and ejaculation usually commences quite quickly as thrusting lessens.

The semen is collected into a warmed insulated vessel, which should be at about 30°C. It is strained through a cotton gauze, and the liquid sperm-rich fraction is collected. This fraction is followed by a large quantity of gel, which is discarded.

Semen evaluation is performed as soon after collection as possible. Semen volume is approximately 250 mL. Sperm density is approximately 100 million sperm/mL. Over 60% of spermatozoa should be both motile and normal.

Evaluation of herd fertility in pigs

Good records and good sow identification are essential prerequisites for the proper evaluation of herd performance. The veterinary surgeon's knowledge of general herd health is also important, as is information about diet changes etc.

Figures for the parameters of herd performance are discussed in Chapter 2 (Tables 2.4–2.8).

FURTHER READING

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11

Polysystemic diseases

Introduction

This important but diverse group of diseases can, as their name implies, affect a number of body systems. They include a number of viral diseases whose effects may reach epidemic proportions. Immunity to classical swine fever, African swine fever, foot and mouth diseases, and swine vesicular disease is low in areas where they are not endemic. Outbreaks of these diseases may involve many thousands of animals, as they spread rapidly through a population with low immunity. These diseases are notifiable to Department for Environment, Food and Rural Affairs (Defra) in the UK and are currently controlled by means of a slaughter policy.

Three new viral diseases have occurred in pigs in recent years that have caused serious economic loss, as they have spread rapidly in a number of countries including the UK. The first of these is porcine reproductive and respiratory syndrome (PRRS). Caused by a virus from the Arteriviridae group, it may initially cause poor reproductive performance in infected herds. It is endemic in a number of countries and recognized chiefly as a cause of chronic respiratory disease. Two more recently identified diseases are postweaning multisystemic wasting syndrome (PMWS) and porcine dermatitis and nephropathy syndrome (PDNS). Both are thought to be caused in part by *porcine circovirus* 2. PMWS in particular has caused severe losses in production in many herds. An effective vaccine has been developed for PRRS but not yet for the porcine circovirus diseases.

The polysystemic group of diseases also includes a number of bacterial infections. The incidence of anthrax in pigs has fallen dramatically in recent years since the withdrawal of animal offal products from the diet. *Clostridium novyi* infection is relatively uncommon, and the incidence of clinical disease caused by *Leptospira interrogans* serovars remains a threat but the incidence is relatively low. Swine erysipelas occurs in a number of clinical forms. These include sudden death in animals with poor immunity in the presence of high levels of infection, and also subacute and chronic forms. Tuberculosis is relatively uncommon. Avian tuberculosis can become a problem when pigs are in contact with poultry that are themselves infected by the disease. The rising incidence of bovine tuberculosis presents an increased risk of infection to the porcine population.

Aetiology of the polysystemic diseases

This group of diseases is caused by either viral or bacterial infection.

Epidemiology of the polysystemic diseases

This depends on the specific disease and is discussed in detail below for each disease in this group. In the case of the acute viral diseases, spread is usually rapid through a herd with little or no immunity. Some pigs may die suddenly, and the specific signs of the disease become evident in other animals. The infection may be present in a number of local herds or spread to these herds may follow. The consequences of infection may be devastating. In foot and mouth diseases, for example, there may be 100% morbidity in the herd. Mortality is variable, with the loss of up to 20% of young pigs but only 2% of adults. A very important epidemiological feature of the disease in pigs is the production and release into the air of large amounts of virus from infected animals. Windborne spread of the disease can occur, with plumes of infection being carried over a large area to pigs and other cloven-hoofed animals.

Although the incidence of anthrax in pigs is now very low, the epidemiology of the disease had an interesting pattern. Historically, a frequent source of infection was pig food contaminated with infected offal. Delivery of such food resulted in a number of cases of anthrax in an area showing a spectrum of the signs associated with the disease.

The epidemiology of the more chronic diseases such as PRRS follows a rather different course. The virus is usually brought on to a farm by an infected pig. Initially, signs of reproductive failure and skin discoloration are seen. The disease is spread by direct contact, by the use of contaminated hypodermic needles, and by flies. After some months, the disease becomes more chronic and signs of reproductive failure decrease, to be replaced by ill thrift and pneumonia. The incidence and severity of other diseases, such as enzootic pneumonia and sarcoptic mange, on the farm may increase. The lack of positive serological evidence of infection may suggest that virus ceases to be present in older sows that were infected at an earlier stage. These older animals can be used with care as foundation PRRS-free stock for a new herd.

Clinical signs of the polysystemic diseases

A spectrum of clinical signs is a feature of a number of the diseases in this group. In classical swine fever, for example, early signs may be non-specific and include pyrexia, depression, and anorexia. Sudden deaths may occur, and there is no response to antibiotic therapy prescribed before an accurate diagnosis has been made. In the course of the next few days, the high body temperature persists but more specific signs start to appear. These include incoordination, diarrhoea, and skin discoloration.

In swine erysipelas, a number of different syndromes occur either together or as distinct clinical entities. Sudden death in a number of pigs occurs in peracute erysipelas, and a number of acute cases may be found in other members of a group or herd. In cases of chronic erysipelas, the main clinical signs are either endocarditis or chronic arthritis. The conditions seldom occur together in the same animal, and there may or may not have been an earlier episode of clinical disease observed or a history of infection in the herd.

In the more chronic diseases such as PMWS, the owner may be aware of poor and uneven growth rates, anaemia, jaundice, pneumonia, and a generalized lymphadenopathy. With the passage of time, the extent and severity of the clinical signs may lessen slightly in subsequent batches of pigs.

Diagnosis of the polysystemic diseases

Diagnosis is based on the history of the farm and its location, the clinical signs seen and their severity, and the morbidity and mortality observed. The response to treatment, post-mortem lesions, and specific laboratory tests are important diagnostic parameters. The post-mortem signs seen may be highly suggestive of a disease. In classical swine fever, for example, these include the presence of petechial haemorrhages on the mucosal surfaces of the larynx and the bladder and the presence of button ulcers in the caecum. The presence of the diseases can be confirmed by virus isolation, serum neutralization, and enzyme-linked immunosorbent assay (ELISA) tests.

Treatment of the polysystemic diseases

In the case of the viral notifiable diseases, a slaughter policy is in place and treatment is not attempted. Treatment of the other viral diseases PRRS, PMWS, and PDNS is not usually effective. Antibiotic therapy may be prescribed to reduce the incidence of secondary bacterial disease. Nursing care is important, and its role in the prevention of these diseases is discussed below. Severely ill animals and those thought unlikely to thrive are euthanized. Antibiotic therapy is effective in leptospirosis and swine erysipelas, but early treatment is essential if severe tissue damage and death are to be avoided. The treatment of anthrax with penicillin may be effective in early cases. Although the condition is notifiable, confirmation of disease is normally possible only after death. Treatment of tuberculosis in pigs is not considered economically viable or desirable.

Control of the polysystemic diseases

This varies with the cause, epidemiology, and severity of the disease. In the case of the acute viral diseases, control is aimed at keeping infection out of a country or its herds. Strict biosecurity should be maintained at ports in an attempt to prevent these diseases entering the country. At a more local level, herd biosecurity is employed to prevent infection gaining access either through the arrival of infected pigs, contaminated food, visiting persons, and vehicles or through discarded meat offal. Once infection is diagnosed, a slaughter policy is used to eliminate infection locally and over wider areas. In some diseases, such as foot and mouth disease, vaccination of animals at risk near an infected premises may be employed. Effective vaccination is not available for all diseases in this group. Vaccines for classical swine fever and African swine fever, for example, are not very effective, especially in the face of high levels of infection.

In the case of PRRS, PMWS, and PDNS, efforts are made by stringent biosecurity, including the screening of potential replacements, to prevent the infections gaining access to a herd. Once the diseases gain entry into a herd, they are liable to become endemic, cause severe economic loss, and present a risk of spread to other units. A vaccine is available against PRRS but not against either PMWS or PDNS.

Biosecurity, vermin control, strict attention to cleanliness, and in some cases vaccination, all play a part in the control of the bacterial diseases in this group.

Details are given for each disease below. Swine erysipelas presents one of the greatest challenges in terms of control. The organism is found in the soil and is also carried by many pigs. The incidence of infection may be stress-related. Sudden hot weather or the recent weaning of a batch of sows can predispose to outbreaks of disease. A good vaccination policy can prove very effective but, despite this, clinical cases of disease including sudden death can occur if levels of stress or infection become very high. Tuberculosis in pigs is seldom diagnosed in life and is most commonly identified at meat inspection. The identity of the organism may help reveal its source. In the case of avian tuberculosis, the source of infection may be a flock of elderly hens, and effective control cannot be achieved until this source is eliminated or at least enclosed.

Classical swine fever

Also known as hog cholera, classical swine fever is a highly infective *pestivirus* infection of pigs that causes both acute disease and chronic disease.

Incidence

Classical swine fever is endemic in much of Western Europe, the Far East, and South America. Frequent epidemics of the disease occurred in the UK until its eradication in 1967 after the introduction of a slaughter policy. Until 2000, the last UK outbreak was in 1986 – then three primary outbreaks all came from the same imported meat source. In the 2000 outbreak, 16 infected units were identified in East Anglia. Control involved the slaughter of 41 000 pigs from infected farms and 34 000 'dangerous contacts'.

Aetiology

The cause is hog cholera virus, a pestivirus related to the bovine viral diarrhoea virus. A single virus is involved but with strains of varying clinical severity. The virus is susceptible to heat but can survive for 4 years in frozen meat and 3 months in salted meat.

Epidemiology

The disease is usually introduced by the import of infected pigs or waste food containing uncooked pig meat. A discarded sandwich from Eastern Europe containing porcine material was thought to be the source of the 2000 UK outbreak. Swill feeding was widely implicated in earlier outbreaks. Spread is mainly directly from pig to pig – all body excretions, especially urine, are highly infective. The virus can be spread by flies, vehicles, and people (including veterinary surgeons). Virus can be present on clothing, on instruments, and in contaminated injectable drugs. Spread is normally rapid and accompanied by severe signs. Spread may be slower when milder strains are present. Markets can be a serious cause of the spread of the disease. Sows affected with mild strains may show no signs, but virus spreads in utero to piglets, which are persistently viraemic when they are born. Infection gains access to the body via the mouth or the respiratory system. Virus localizes in the tonsil, and viraemia causes spread throughout the body within 24 h.

Clinical signs

These vary between acute, chronic, and congenital forms.

Acute disease

First signs are seen within 10 days of infection – occasionally more. Some sudden deaths are seen, especially in young pigs. Many pigs are severely depressed, with high temperatures (41.5–42.5°C). The pigs are anorexic and bury themselves in the straw. They are reluctant to move and may walk with swaying gait (Figs 11.1 and 11.2). Constipation may be seen initially, followed by diarrhoea and vomiting. Ocular discharge is present, and the eyelids may be stuck together (Fig. 11.3). Purple blotchy discoloration of skin (Fig. 11.4) is present, especially on the ventral abdomen, and there may be necrosis of ear tips. Ecchymotic haemorrhages may be seen on the skin (Fig. 11.5). Nervous signs include circling incoordination, muscle tremors, and convulsions.

Chronic disease

This is seen with low-virulence strains of virus. Affected pigs are dull, anorexic, and fail to thrive. Areas of skin discoloration and dermatitis may be seen. Pigs are susceptible to secondary bacterial infection and glomerulonephritis.

Fig 11.1 – Classical swine fever: weak ataxic sow leaving pen. (Courtesy of J.D. Mackinnon.)

Fig 11.2 – Classical swine fever: weak ataxic growers. (Courtesy of J.D. Mackinnon.)

Fig 11.3 – Classical swine fever: gilt ocular discharge and high fever. (Courtesy of J.D. Mackinnon.)

Fig 11.4 – Classical swine fever: dull weaner with cyanosis of ears and snout. (Courtesy of J.D. Mackinnon.)

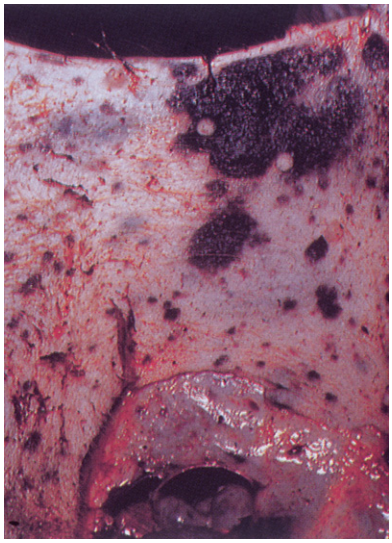

Fig 11.5 – Classical swine fever: ecchymotic haemorrhages on the skin. (Courtesy of J.D. Mackinnon.)

Congenital infection

Congenital infection usually involves low-virulence strains of virus. Affected sows show mild pyrexia and may abort or produce small litters with mummified piglets. Some piglets are born with viraemia. Piglets may show cerebellar hypoplasia and type A1 congenital tremor – myoclonia congenita. Other pigs grow initially but fail to thrive and die before maturity.

Diagnosis

This is based on history, clinical signs, post-mortem lesions, virus isolation, and serology. Pigs may show leucopenia.

Post-mortem – Post-mortem signs include petechial haemorrhages in kidney ('turkey egg') (Fig. 11.6), larynx (Fig. 11.7), and bladder. Enlarged haemorrhagic lymph nodes are seen (Fig. 11.8), and splenic infarcts (Fig. 16.16) and button ulcers in the caecum also occur (Fig. 11.9). Virus is detected by fluorescent antibody test (FAT), agar gel precipitation, and polymerase chain reaction (PCR). Serology includes fluorescent antibody neutralization test and ELISA tests.

Differential diagnosis

- *Erysipelas*. See skin lesions. Culture.
- *Salmonellosis*. Culture. Yellow diarrhoea and dyspnoea occur.
- *Pasteurella*. Culture. Pneumonia present.
- *PMWS and PDNS*. See below.

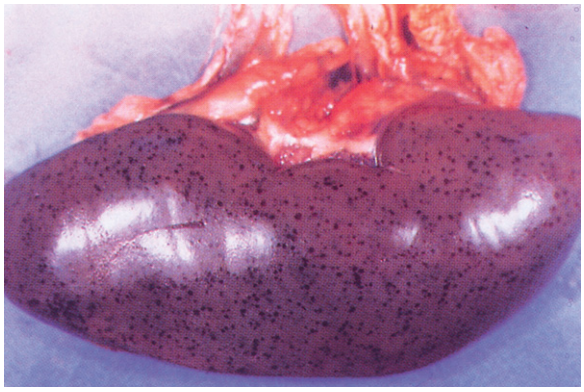

Fig 11.6 – Classical swine fever: haemorrhages in kidney – 'turkey egg' kidney. (Courtesy of J.D. Mackinnon.)

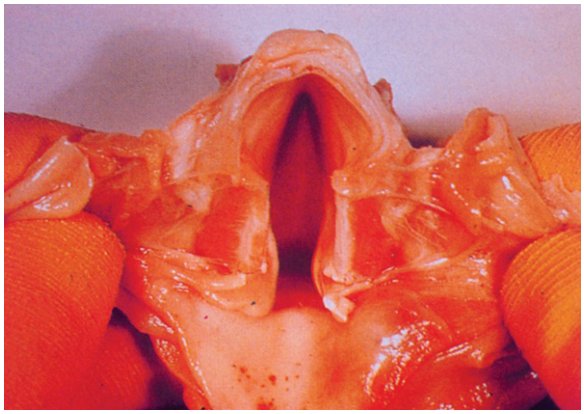

Fig 11.7 – Classical swine fever: petechial haemorrhages on the epiglottis (Courtesy of J.D. Mackinnon.)

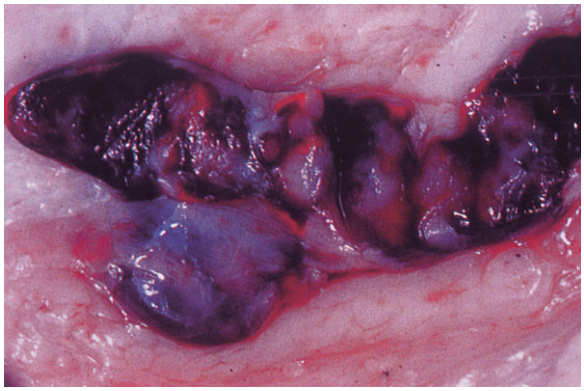

Fig 11.8 – Classical swine fever: haemorrhagic lymph nodes. (Courtesy of J.D. Mackinnon.)

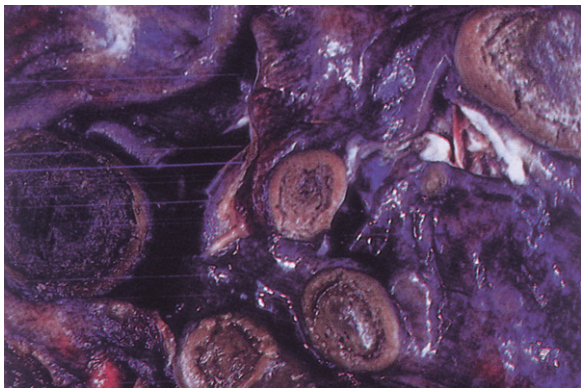

Fig 11.9 – Classical swine fever: button ulcers in the caecum. (Courtesy of J.D. Mackinnon.)

Treatment

None is effective.

Control

Classical swine fever is a **notifiable** disease in the UK. If suspected, Defra must be notified and precautions taken not to spread this highly infectious disease. There is a slaughter policy in the UK. Vaccination is used elsewhere, but vaccines vary in efficacy. Close markets and restrict pig movements.

African swine fever

Also known as African pig disease, this severe viral disease can be accompanied by high morbidity and mortality. It is mostly seen in its acute form, but recently milder forms have been described.

Incidence

The condition occurs in Africa but also in parts of Europe (in Spain and Portugal but not in the UK) and the Caribbean.

Aetiology

The cause is a large *icosahedral DNA virus* that replicates in the cytoplasm of infected cells.

Epidemiology

The disease spreads directly between pigs, mainly through the respiratory tract. Pigs that survive may be persistently viraemic. Tick and biting fly vectors are involved in transmission between domestic pigs and warthogs in Africa and Europe.

Clinical signs

Acute and chronic forms may be seen.

Acute

Pigs have pyrexia (temperature 41.5°C) initially for approximately 4 days, and then their temperature falls as other signs develop: severe depression, weak hind legs, ocular discharge, diarrhoea, and cough. Convulsions and death may follow within a few days.

Chronic

Pyrexia and depression are present. Pigs later become emaciated, with oedematous swellings over the joints. The disease may become enzootic, with recovery of some animals.

Diagnosis

Base diagnosis on post-mortem lesions, virus isolation, and serology.

Post-mortem – The virus causes thrombocytopenia with widespread ecchymotic haemorrhages. Occasional button ulcers are seen in the caecum. The virus is detected by FAT, and antibodies by an ELISA on serum. Affected pigs are leucopenic and lymphopenic.

Differential diagnosis

- *Classical swine fever.* This is milder. Use virus isolation.
- *Erysipelas*. Note response to treatment and serology.
- *Salmonellosis*. Use a bacterial culture.

Treatment

None is effective.

Control

African swine fever is **notifiable** in the UK and European Economic Community (EEC). A slaughter policy is recommended. Vaccines are not very effective.

Foot and mouth disease

This is a severe viral disease affecting pigs and ruminants.

Incidence

Foot and mouth disease is endemic in parts of the Far East, South America, Africa, Asia, and parts of Europe. There was a major UK outbreak in 2001 – the first case was detected in abattoir pigs.

Aetiology

The cause is an *apthovirus* that is a member of the Picornaviridae family. Seven strains are recognized, with a number of subtypes.

Epidemiology

The disease is highly contagious. Morbidity is often 100%. Mortality is lower, with 2% of adults and 20% of young animals dying. Economic consequences are severe and long-lasting. The disease spreads easily, with infection gaining access via the mouth and respiratory systems. The virus establishes itself in the oropharynx. The resultant viraemia carries the virus to the skin, causing most of the clinical signs. Large amounts of virus are excreted. Airborne infection by plumes of the virus has been blamed for spread between mainland Europe and the UK, especially if the wind direction is favourable. Infected meat products in swill are a major source of infection in pigs. Birds, wildlife, and movements of people can also be involved in transmission. In the 2001 UK outbreak, the initial diagnosis of foot and mouth disease was made in a group of pigs found to be lame while awaiting slaughter. Over 4 million animals, including 146 000 pigs, were slaughtered during this outbreak.

Clinical signs

Initial signs include pyrexia (temperature 40–41°C) with depression and anorexia. Lameness is observed, and vesicles develop on the snout and coronary band. Hooves may be shed. Vesicles coalesce, leaving areas of skin denuded of epithelium (Fig. 11.10). Younger stock are more susceptible than adults, and some sudden deaths occur.

Diagnosis

This is based on history, clinical signs, virus isolation, and serology. Virus from vesicles is identified by tissue culture, PCR, ELISA, and experimental transmission. Serology involves ELISA.

Post-mortem – Foot and mouth lesions are present, but one may also see lesions in pharynx, oesophagus, and trachea. Myocarditis may be seen at post-mortem in young pigs.

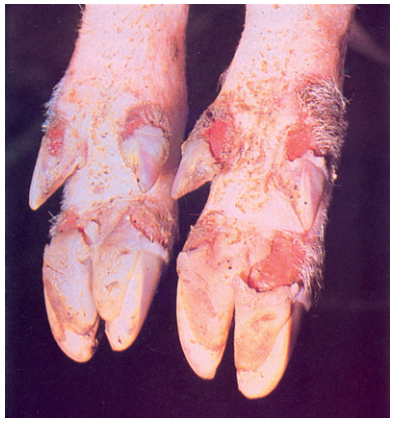

Fig 11.10 – Foot and mouth disease: ruptured vesicles on the coronary bands.

Differential diagnosis

In the UK, the differential diagnosis is chiefly swine vesicular disease. Exposure of the feet to strong disinfectant solutions may cause foot lesions without the pyrexia seen with foot and mouth disease. Work is progressing on an ELISA test to differentiate immunity to vaccination and that to viral exposure based on detection of non-structural proteins of the foot and mouth disease virus.

Treatment

None is effective.

Control

Control is by a slaughter policy in the UK. The disease is **notifiable**; it must be reported to Defra and the reporting veterinary surgeon must remain on the farm. Hand wash and clothing change should prevent spread by veterinarians between farms. In the future, vaccination may be used to ring fence infected pig herds.

Swine vesicular disease

This is a viral disease of pigs that was first identified in 1966 and produces clinical signs indistinguishable from those of foot and mouth disease. Pigs usually recover within 2–3 weeks.

Incidence

The disease was seen in several EEC states during the early 1990s, but UK pigs were serologically negative in a 1993 survey. The last confirmed UK case was in 1982.

Aetiology

The cause is an *enterovirus* of the Picornaviridae family.

Epidemiology

The disease is spread by direct pig to pig contact and through feeding uncooked pig products. It can also be spread by vehicles, people's boots, and other fomites. Unlike foot and mouth disease, airborne infection does not occur.

Clinical signs

These commence with a short episode of pyrexia (temperature 41°C) and anorexia. Evidence of lameness is then seen: pigs have their backs arched and are reluctant to move. Lameness is worse on solid surfaces. Vesicles are seen on the coronary band; skin pallor is present before vesicles appear. The vesicles rupture, leaving areas denuded of skin (Figs 11.11 and 11.12). Horn of hoof is occasionally shed. Lesions are less commonly seen on snout, tongue, and lips. Signs normally disappear within 2–3 weeks.

Diagnosis

This is based on history, clinical signs, and virus complement fixation test and FAT tests. Serology is by ELISA.

Differential diagnosis

Foot and mouth disease and other vesicular diseases are differential diagnoses. The clinical signs also occur in pigs whose feet have been exposed to irritant solutions.

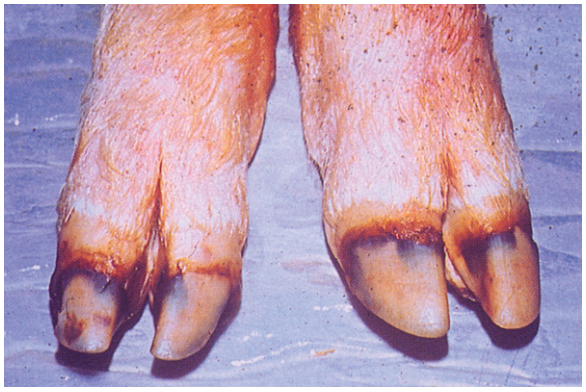

Fig 11.11 – Swine vesicular disease: coronary band lesions 5 days after infection.

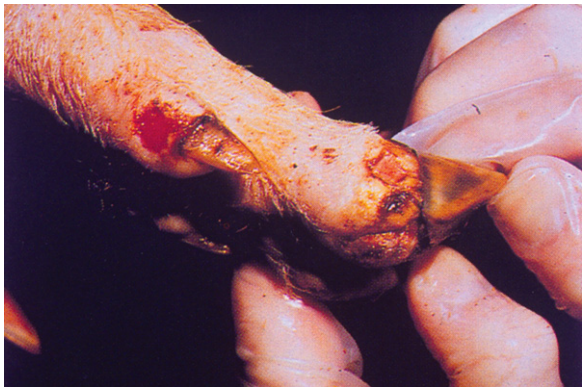

Fig 11.12 – Swine vesicular disease: coronary band lesions 12 days after infection.

Treatment

None is effective or necessary.

Control

This is a **notifiable** disease. It must be reported to Defra, and the reporting veterinarian must remain on the farm.

Malignant catarrh fever

This is caused by ovine herpesvirus 2 (PCR confirmation). A number of sporadic cases in pigs have been described in the literature. Clinical signs are rather like those in cattle, including ocular discharge, respiratory signs, and corneal opacity. There is no response to antibiotic therapy. In two Norwegian cases, affected pigs had been in close contact with sheep.

Porcine reproductive and respiratory syndrome

This is also known as blue-eared pig disease and as 'purrs' in the pig world. PRRS is an important and highly infectious pig disease that has been seen in many parts of the world during the past 20 years.

Incidence

The disease was first seen in North America in 1985. Since then, it has spread throughout the pig-keeping world. It was first seen in the UK in 1991 and is now endemic – often as a subclinical infection that predisposes to other infections – in many UK herds. Suboptimal performance in an infected herd can continue for years. There were 60 Veterinary Investigation Diagnosis Analysis (VIDA) diagnoses in the UK in 2000, 23 in 2002.

Aetiology

The cause is a virus belonging to the Arteriviridae family that is closely related to the equine arteritis virus. Several strains of varying virulence have been reported. The virus is labile but can survive for years in frozen material.

Epidemiology

The virus may enter a herd with infected pigs. It is present in semen and in faeces. Windborne spread may occur rarely, but there is limited experimental evidence of aerosol spread. Spread is by direct contact and within buildings and by contaminated needles and flies. The virus may take 5 months to spread through a herd, causing abortion and reduced litter size. Weaners are thought to be the main source of infection, as the virus is not excreted by older pigs. Respiratory disease caused by the virus may persist in an infected herd for years. Mange infestation on farm may become more severe.

Clinical signs

These are very variable, as is the severity of observed signs. Reproductive failure, with late abortion, stillbirth, and weak piglets, may persist for many months (Fig. 11.13). Sows develop good immunity after abortion and rarely abort again. Respiratory disease involves pyrexia, anorexia, cough, dyspnoea, skin discoloration, and ill thrift. In the worst-affected herds, up to 25% of young pigs die, often through secondary infection with, for example, *Haemophilus parasuis*. Respiratory signs may precede abortion in gilts. Some strains produce red cell anaemia in growing pigs and may predispose to pneumonia. Local haemorrhage from iron injection sites and elsewhere may be the result of thrombocytopenia.

Diagnosis

Herd history of breeding losses and respiratory disease may be suggestive of infection in new outbreaks. A number of serological tests are available. An indirect serum ELISA test gives good results. Herd serological profiles are useful to assess the extent of infection and confirm a diagnosis.

Post-mortem – Interstitial pneumonia is found in young pigs, including any weak neonates (Fig. 11.14). Using fluorescent techniques, virus is found in tissues. Serology involves indirect ELISA test.

Fig 11.13 – Porcine reproductive and respiratory syndrome: piglets born to an infected sow. (Courtesy of R.W. Blowey.)

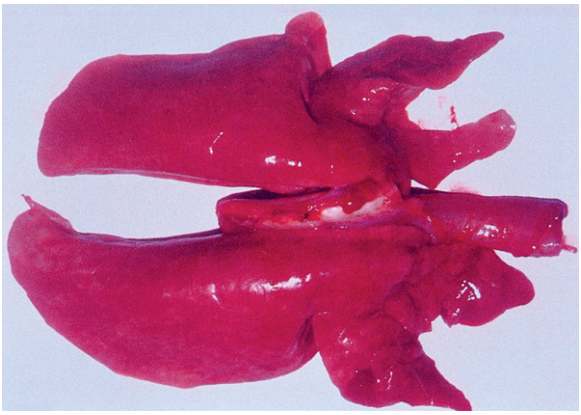

Fig 11.14 – Porcine reproductive and respiratory syndrome/ postweaning multisystemic wasting syndrome: lungs from a piglet with pneumonia. (Courtesy of R.W. Blowey.)

Differential diagnosis

Other causes of reproductive failure (e.g. parvovirus, leptospirosis, and brucellosis) are possible. Other causes of respiratory disease – for example enzootic pneumonia (*Mycoplasma hyopneumoniae*), *Pasteurella multocida*, *Streptococcus suis*, *Actinobacillus pleuropneumoniae*, and *H. parasuis* (Glasser's disease) – are other differential diagnoses. The clinical signs and post-mortem appearance of tissues may indicate which disease is present. Isolation of the causal organism or serological evidence of a recent infection may help to confirm the diagnosis.

Treatment and control

Maintain a closed herd if possible. Segregate and rear young pigs off site if possible. Antibiotic therapy may help control secondary infection. Depopulate and thoroughly clean weaner accommodation. A recent report from the USA describes successful elimination of PRRS from a herd of sows by blood testing (ELISA and PCR) and culling positive cases. Ten per cent of the herd should be tested. A second blood test may be advisable. Older animals may eventually become free of disease and could form the nucleus of a PRRS-free herd.

A live freeze-dried vaccine has been recently introduced for use on weaners and fatteners only from 6 weeks upwards. It is not to be used on breeding stock or on pigs coming in contact with breeding stock. The vaccine is given by intramuscular injection in a single dose. It may cause abortion in sows.

Postweaning Multisystemic Wasting Syndrome and Porcine Dermatitis and Nephropathy Syndrome

These two new diseases, which may be related, have recently (2000) become widespread in the UK – especially in East Anglia, where they are causing serious losses and much concern by adding to the problems of the pig industry. A major problem in their diagnosis is that some of their clinical signs resemble those of classical swine fever.

Postweaning multisystemic wasting syndrome

Incidence

Postweaning multisystemic wasting syndrome was first described in Canada in 1991. It was recognized in the UK late in 1999. PMWS has also been seen in the USA, Europe, and Ireland. It is widespread in East Anglia and Yorkshire but less common elsewhere in the UK. From December 1999 to March 2000, 159 new outbreaks were recorded by the Veterinary Laboratories Agency (VLA); there were none in Wales. In 2002, there were 286, including spread to Yorkshire. The disease was reported in UK wild boar in 2003.

Aetiology

The cause is possibly porcine circovirus 2, but Koch's postulates have not been fulfilled with this virus. Other trigger factors – including moulds in food or possibly another unidentified virus, PRRS, or parvovirus infection – have been suggested but not confirmed. Infection may be passed from sow to litter, although this is unproven. The incubation period may be as long as 3 months. It has been suggested that the presence of PMWS may worsen the effect of other viruses (e.g. Aujeszky's disease in Spain). It is not thought that *Mycoplasma hyopneumoniae* vaccine predisposes to PMWS infection. Immunosuppression caused by PMWS may predispose to infection by other pathological organisms.

Clinical signs

These are usually first seen in pigs aged 8–12 weeks. Affected animals show chronic wasting, anaemia with pallor of the skin, and enlarged lymph nodes, especially the inguinal nodes (Fig. 11.15). A low PCV and red blood cell count are present. The pigs often show jaundice (Fig. 11.16) and a decreased growth rate. Some animals

Fig 11.15 – Postweaning multisystemic wasting syndrome: affected group of pigs showing great variation in size and condition. (Courtesy of Merial and Chen 2000.)

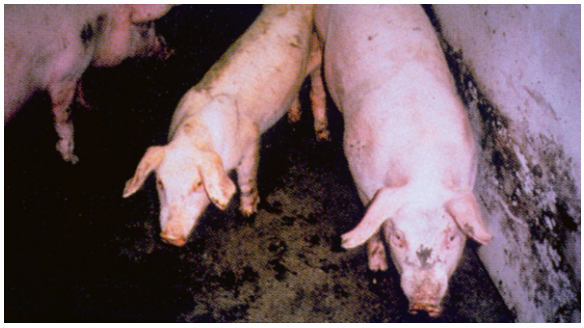

Fig 11.16 – Postweaning multisystemic wasting syndrome: jaundiced ill-thriven pig with a normal pig of similar age. (Courtesy of Merial and Ellis and Allan 2000.)

show signs of diarrhoea and respiratory signs. An expiratory dyspnoea may be observed, but there is no cough. Some pigs develop gastric ulcers. At least 30% of affected pigs die. Other pigs in a group in which infected pigs are seen may be normal.

Diagnosis

Base diagnosis on signalment of affected animals, clinical signs, and lesions. Serology is examined.

Post-mortem – The carcass is emaciated and jaundiced. The spleen and lymph nodes are enlarged, the kidneys may be swollen and have visible white spots, and the lungs are rubbery and mottled (Figs 11.17–11.22).

Differential diagnosis

All other causes of wasting must be eliminated. The advice of Defra must be sought if the clinical signs resemble those of classical swine fever. Virus isolation and serology should confirm or refute the presence of classical swine fever.

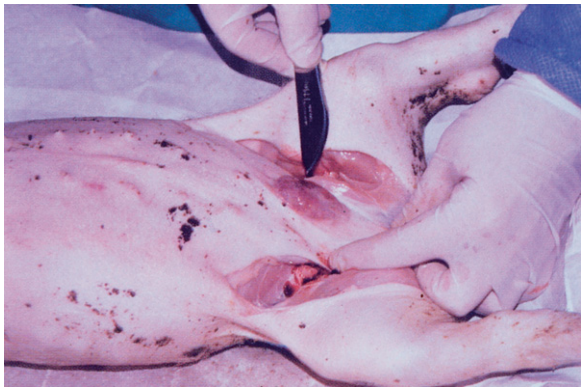

Fig 11.17 – Postweaning multisystemic wasting syndrome: enlarged non-haemorrhagic inguinal lymph nodes.

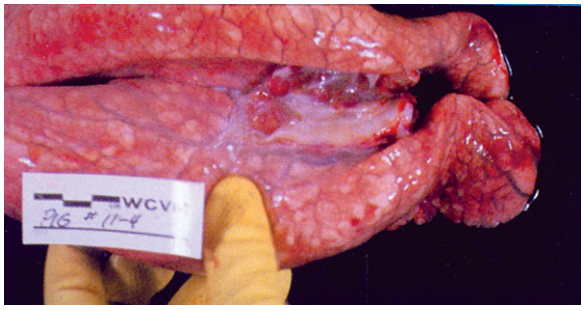

Fig 11.18 – Postweaning multisystemic wasting syndrome: mediastinal lymph node adenopathy and interstitial pneumonia. (Courtesy of Merial and Ellis and Allan 2000.)

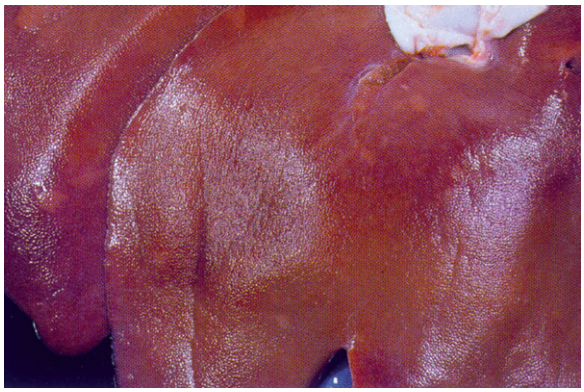

Fig 11.19 – Postweaning multisystemic wasting syndrome: atrophy of the liver. (Courtesy of Merial and Ellis and Allan 2000.)

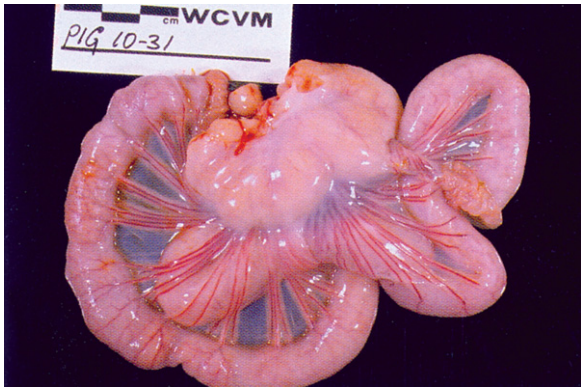

Fig 11.20 – Postweaning multisystemic wasting syndrome: mesenteric lymphadenopathy. (Courtesy of Merial and Ellis and Allan 2000.)

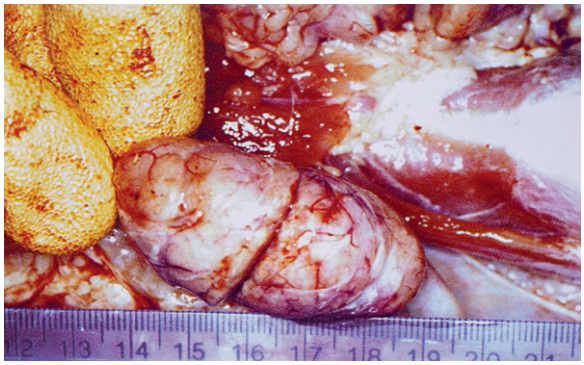

Fig 11.21 – Postweaning multisystemic wasting syndrome: enlarged pale submandibular lymph node. (Courtesy of Merial and Guilmoto and Wessel-Robert 2000.)

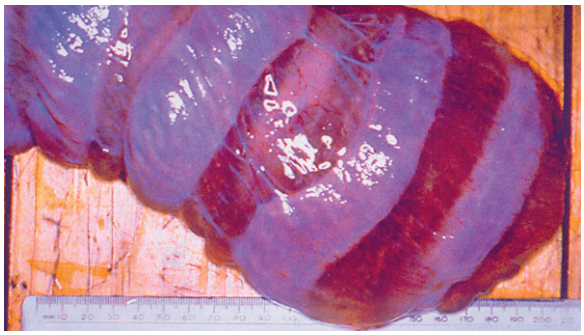

Fig 11.22 – Postwearing multisystemic wasting syndrome: oedema of colonic mesentery. (Courtesy of S.H. Done.)

Treatment and control

No specific treatment and no commercial vaccine are currently available. Serum treatment was described in 2002. Harvesting serum from recovered pigs and giving it by intraperitoneal injection to piglets at weaning was claimed to help prevent disease. The serum used must be harvested from the farm of origin. Great care must be taken to prevent spread to and from other farms by vehicles, personnel, etc. Recent VLA advice is as follows.

- As most transmission occurs shortly after weaning, delay weaning and reduce mixing of piglets at weaning. Do not mix until pigs weigh 30 kg or are 10 weeks old. It is best to keep them in their original litter group. Cross-fostering should be avoided.
- Minimize stress, including moving, mixing, inappropriate management, or poor food.
- All in, all out management is best.
- Reduce stocking density and control the environment to give optimal environmental conditions.
- Clean, disinfect, and rest pens between batches of pigs.
- Treatment is mostly unrewarding. There are some reports of better recovery with broad-spectrum antibiotics plus non-steroidal anti-inflammatory drugs (NSAIDs) or steroids. Many pigs die despite treatment – euthanasia may be required for any that are severely affected. The VLA advise that identification of any secondary pathogens is helpful – sensitivity testing and also possible use of vaccine can help considerably. Common secondary infections include enzootic pneumonia, salmonellosis, and Glasser's disease.
- Moving affected pigs to comfortable low-density hospital pens seems to help but must be done early in the disease to be effective. Some farmers advise euthanasia of any affected animals and avoid hospital pens. Survivors may be uneconomic. Good colostrum intake also seems to help avoid disease.

Porcine dermatitis and nephropathy syndrome

Incidence

An important new disease, PDNS causes illness in growers and finishers and seldom affects adult pigs. It was first reported in Scotland in 1993, becoming widespread in East Anglia in 1999. From December 1999 to March 2000, the VLA recorded 139 new outbreaks in East Anglia. There were 49 in the VIDA list for 2002.

Aetiology

Pasteurella multocida has been found in many cases in Scotland. The disease was thought to be caused by a toxin

or an antigenic substance of bacterial origin. Porcine circovirus 2, which is associated with PMWS, has been found in some but not all cases. The exact cause of PDNS is unclear. PDNS may develop in about 15% of cases of PMWS, but the association is not proven.

Epidemiology

This is currently unknown.

Clinical signs

Affected pigs are depressed and reluctant to eat or move. Some are pyrexic. Some breathe heavily. Mortality is about 15%, and surviving pigs may be unthrifty. Affected pigs show an extensive dermatitis over the chest, abdomen, thighs, and forelegs. The skin in affected areas has purplish red bumps of varying size and shape (Figs 11.23 and 11.24). Some pigs die of glomerulonephritis without showing skin lesions. Cases of severe skin lesions are now less common than when the disease was first described.

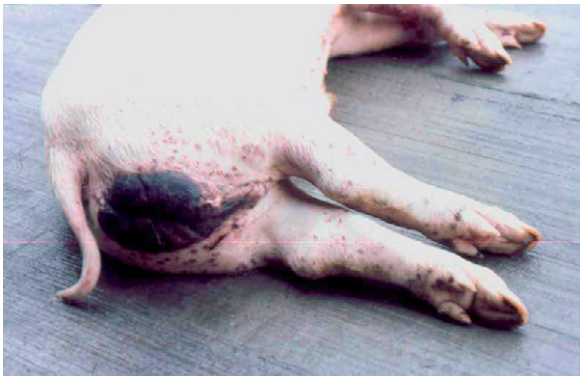

Fig 11.23 – Porcine dermatitis and nephropathy syndrome: skin lesions on scrotum. (Courtesy of J.R. Thomson).

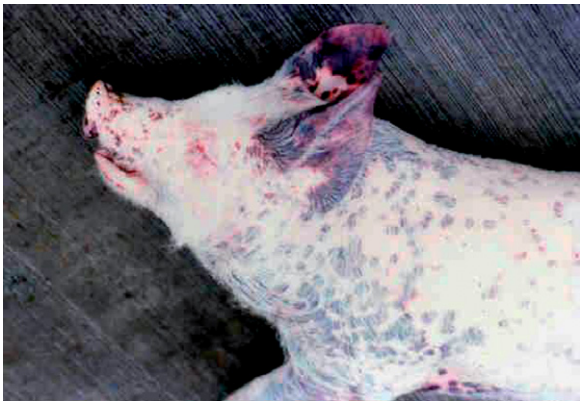

Fig 11.24 – Porcine dermatitis and nephropathy syndrome: skin lesions on head. (Courtesy of J.R. Thomson.)

Diagnosis

This is based on the clinical signs and post-mortem findings, both of which can resemble those of classical swine fever. Classical swine fever must be excluded (if suspected) by Defra advice and laboratory tests, which may take up to 7 days.

Post-mortem – The kidneys are mottled, with numerous small haemorrhages (Fig. 11.25). There may be some free fluid in the abdomen, and there may be oedema of the colonic mesentery. The lymph nodes of the caudal abdomen and the inguinal lymph nodes are haemorrhagic, enlarged, and red (Fig. 11.26). Other findings are inconsistent.

Fig 11.25 – Porcine dermatitis and nephropathy syndrome: normal and grossly enlarged kidneys.

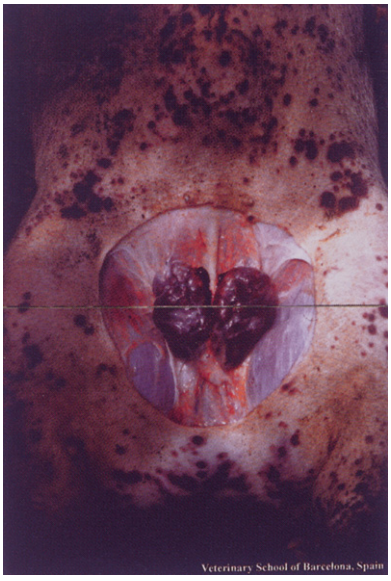

Fig 11.26 – Porcine dermatitis and nephropathy syndrome: enlarged haemorrhagic inguinal lymph nodes. (Courtesy of Merial and Segalés and Domingo 2000.)

Treatment

None is effective; it may be best to euthanize pigs on welfare and economic grounds.

Control

Control is difficult in the absence of a clear indication of cause (see also under *Postweaning multisystemic wasting syndrome*).

Anthrax

Anthrax is an acute infectious disease occurring in pigs, ruminants, and horses. It is potentially zoonotic.

Aetiology

The cause is *Bacillus anthracis*.

Incidence

Anthrax is worldwide but relatively uncommon now in the UK. A severe and long-lasting outbreak occurred in Wales in the early 1990s. Most cases involve small numbers of pigs, but 19 pigs were involved (including piglets) in the Welsh outbreak.

Epidemiology

The source of pig infection is mainly imported contaminated feedstuffs, especially when the inclusion of products such as blood and bonemeal was permitted. Infection can be endemic in areas where anthrax spores are widespread in the soil.

Clinical signs

Three main forms of the disease are recognized in pigs: pharyngeal, enteric, and septicaemic. All forms may be seen in related outbreaks.

Pharyngeal form

The pig is mostly pyrexic (temperature 42°C); the pharyngeal area and upper neck are unilaterally or bilaterally swollen and oedematous. The pig may be dyspnoeic and dysphagic through local pressure on the trachea or pharynx. The swollen area is non-painful. Depression, vomiting, and death may follow in 24 h.

Enteric form

One may see diarrhoea, dysentery, and death.

Septicaemic form

Sudden death may be seen in younger pigs with pulmonary lesions.

Diagnosis

Diagnosis is based on clinical signs and isolation of the organism. The clinician must avoid opening the carcass whenever possible to avoid contamination of the environment by spores. Smears of blood, oedematous swellings, and peritoneal fluid are examined for the causal organism.

Post-mortem

- *Pharyngeal form*: bloody oedema of the subcutaneous area of the neck.
- *Enteric form*: severe inflammation of intestine, with intensely bright red petechiae in the mesentery and bowel wall.
- *Septicaemic form*: general congestion of body tissues and enlarged spleen.

Differential diagnosis

Acute clostridial disease and peracute erysipelas causing sudden death are possible, also lightning strike.

Treatment

The disease is **notifiable** in the UK and must be reported to Defra. It will respond to high doses of penicillin if seen and treated soon after infection.

Control

The carcass should not be opened but disposed of under Defra guidance.

In-contacts can be treated with penicillin. Warn people of possible zoonotic consequences. A vaccine was available when the disease was more widespread. Since the Welsh outbreak, vaccine has been available from Defra. Control responsibilities are set out in the Anthrax Order of 1991; Defra rather than the local authority is now responsible for cleaning up and disinfecting.

Clostridium novyi infection

Incidence

This is relatively uncommon but can be responsible for the sudden deaths of adult or heavy finishing pigs. It is seen in intensive indoor units and also on outdoor units.

Aetiology

Clostridium novyi type B is responsible.

Epidemiology

This is not known; there may be possible activation of spores in the liver when oxygenation is reduced. This may also be present in cases of sudden death caused by infections such as peracute swine erysipelas.

Clinical signs

Sudden death occurs.

Diagnosis

Base diagnosis on clinical signs and post-mortem.

Post-mortem – There is rapid decomposition of the carcass. The liver is pale and friable, with a 'foam rubber' appearance. The organism is identified by FAT and PCR, but diagnosis is often hampered by rapid decomposition.

Differential diagnosis

Other causes of sudden death, especially peracute erysipelas and anthrax, are possible.

Treatment

If the infection is suspected in life, large doses of antibiotics such as crystalline penicillin may be given.

Control

This involves vaccination using 8 in 1 clostridial vaccines, which are not licensed for use in pigs.

Leptospirosis

This is a group of infectious diseases that have a worldwide distribution. The organisms are sometimes associated with disease, but in other cases serological evidence shows subclinical exposure to infection. This group of diseases is also discussed in Chapter 10 (*Obstetrics and reproduction in pigs*).

Incidence

Leptospirosis has worldwide distribution. It was diagnosed as a cause of abortion on four occasions in 1997, although the cause of some abortion problems remains undiagnosed.

Aetiology

In the current classification, pathogenic leptospires are grouped together as *L. interrogans*. There are numerous serovars broadly classified as those that are either host-adapted or non-host-adapted (accidental infection) for a particular species. In pigs, *L. pomona*, which does not occur in the UK, is considered to be host-adapted. *L. canicola*, *L. icterohaemorrhagiae*, and *L. australis* species show serological evidence of activity in the UK. *L. bratislava* and *L. muenchen* are the most common members of the *L. australis* group in the UK.

Epidemiology

Infection is often contracted from a host-adapted species. Such species are readily and widely infected but show few signs of disease. The non-host-adapted species is less easily infected. Once infected, however, the animal may show severe signs of disease. The brown rat is the host-adapted species for *L. icterohaemorrhagiae*, and this organism can produce severe disease in pigs, in which it is not host-adapted. Infection gains access through the mouth, skin wounds, transplacental transmission, or in some cases the venereal route. Excretion of organisms, especially in the urine, leads to rapid spread of infection, especially in intensive units.

Clinical signs

The disease may be seen as acute infection, subclinical infection, and reproductive disease (chiefly abortion).

Acute infection

This is associated with *L. canicola* and *L. icterohaemorrhagiae*. Affected pigs are pyrexic (temperature 40°C), dull, and anorexic. They may show diarrhoea and jaundice; the latter is said to be rare and may be accompanied by haemolysis (Figs 11.27 and 11.28).

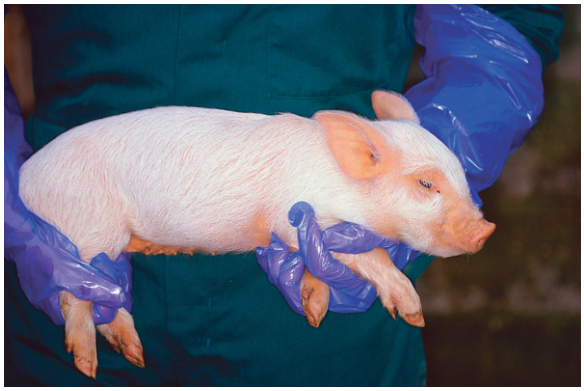

Fig 11.27 – *Leptospira icterohaemorrhagiae*: jaundiced piglet.

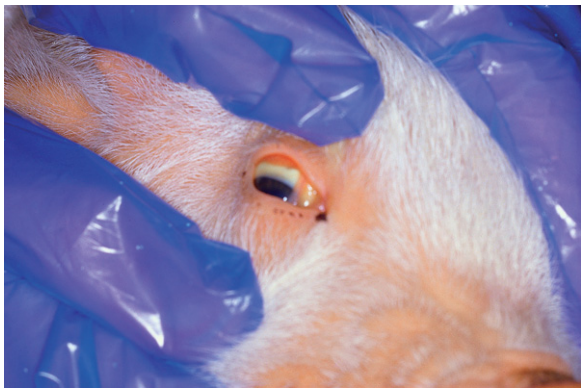

Fig 11.28 – *Leptospira icterohaemorrhagiae*: yellow sclera of jaundiced piglet.

Subclinical infection

This is quite widespread in outdoor pig units. There is serological evidence of infection but no clinical signs.

Reproductive disease

This causes abortion and neonatal mortality of unthrifty pigs. Affected sows may show pyrexia, agalactia, and jaundice. Aborted fetuses may be autolysed.

Diagnosis

Diagnosis is by demonstration of the organism or its culture from body fluids and tissues. Serology includes agglutination tests, ELISA, and DNA probes.

Differential diagnosis

Differential diagnoses are other causes of abortion: swine erysipelas and parvovirus PRRS. In pyrexic pigs, other acute infections, both bacterial and viral, are possible.

Treatment

Antibiotic therapy, especially penicillin–streptomycin, is given in cases of acute infection. Early treatment produces the best results. Antibiotics can also be used to eliminate infection in carriers.

Control

Hygienic measures and control of vermin aid the control of infection.

Vaccination can be used against serovars such as *L. icterohaemorrhagiae*, and in cases of severe herd disease depopulation may be necessary.

Swine erysipelas

This is an important infectious disease of pigs seen in peracute, acute–subacute, and chronic forms. High mortality is seen in untreated animals.

Incidence

Swine erysipelas has worldwide distribution and is common in unvaccinated animals.

Aetiology

The cause is *Erysipelothrix rhusiopathiae (insidiosa)*. A number of different serotypes have been recognized.

Epidemiology

The organism is found in the bodies of normal pigs, especially in the tonsils and other lymphoid tissue. It is also widespread in the soil in pig-keeping areas, where it can survive for variable periods. Sick pigs excrete the organism in their urine, saliva, nasal discharges, and faeces. Cases of acute erysipelas often occur following stress – sudden changes of weather, recent weaning or service, and following movement. The disease also occurs where pigs are overstocked in unhygienic conditions. Its incidence may rise if vaccination is abandoned.

Clinical signs

Peracute, acute, and chronic forms are recognized.

Peracute erysipelas

Pigs are found dead, with bloating of the carcass and purple-reddish discoloration of the skin. Several pigs in a group may be affected, with others being acutely ill with

pyrexia (temperature 42°C), dullness, and sometimes collapse. Other pigs in the group may show signs of acute erysipelas (see below).

Acute erysipelas

This is sometimes termed *subacute erysipelas*. Affected pigs are dull, depressed, and pyrexic (temperature 41–42°C). Characteristic diamond-shaped and elevated skin lesions are seen on the skin within 24 h of the onset of disease (Fig. 11.29). Lesions are palpable, especially on the dorsal surface of the neck and shoulders, before they are visible. In black pigs, the lesions are less easily seen but readily palpable. Skin lesions are initially pink then turn red and finally black in untreated cases. Individual lesions may occasionally coalesce and become necrotic. In such cases, the dead skin forms a hard shield-like layer of rigid tissue penetrated by hairs and underlain by a suppurating skin surface. Early cases may not show the pathognomonic skin lesions but only a very high temperature. In most cases, following treatment the skin lesions become pale and disappear. Sows in late pregnancy may abort. Suckling and unweaned piglets on an infected sow rarely show signs of the disease.

Chronic erysipelas

This is seen in joint and cardiac forms, which occasionally occur together. It may also have a general adverse effect on reproduction.

- *Chronic arthritis* is now thought to follow acute infection but may also have an immune response to persistence of the antigen at the site. Lameness and unwillingness to walk are seen. Temperature is often normal. Joints are hot and swollen but usually without distension of the joint capsule (Fig. 3.11). Movement of joints is reduced, and some new bony growth may be seen on the epiphyses of bones making up the joints. All limb joints may be affected but especially the hip, stifle, hock, and carpus.
- In *endocarditis*, vegetative lesions develop on the heart valves, especially on the tricuspid. Sudden deaths may occur in undetected cases. Exercise intolerance, tachycardia, and cyanosis of mucosae (and the skin in white pigs) are seen in some cases (Fig. 8.3). Auscultation of the heart may reveal a cardiac murmur. An ultrasonographic scan of the heart may reveal valve lesions in compliant animals (Fig. 8.4).

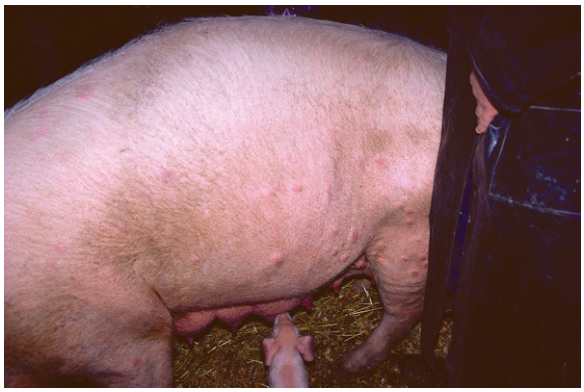

Fig 11.29 – Swine erysipelas: skin lesions in a subacute case.

Diagnosis

This is based on history and clinical signs.

Post-mortem – In acute cases, numerous ecchymotic haemorrhages may be seen. The spleen may have rounded edges, and the organism may be seen in impression smears of the cut surface of the spleen stained with methylene blue. Joint and heart lesions are characteristic in chronic cases (Fig. 8.5). Culture of the organism from the heart blood and also serology are useful diagnostic aids.

Differential diagnosis

In acute cases, other causes of pyrexia – including classical swine fever, leptospirosis, streptococcal septicaemia, salmonellosis, and anthrax – must be considered. In sudden death cases, acute clostridial infection may occur. Other causes of abortion if this has occurred, for example parvovirus, are seldom pyrexic. Arthritis in pigs can also be caused by *H. parasuis* and also *Mycoplasma hyorhinis* (especially 3–10 weeks) and *Mycoplasma hyosynoviae* (especially 10–26 weeks). Endocarditis can also be caused by streptococcal infection.

Treatment

The organism is responsive to penicillin. The maximum therapeutic dose is given at12-h intervals in acutely ill pigs. NSAIDs may help in severe cases. Treatment should continue for 3–5 days to prevent chronic infection. Pyrexic sows often show subnormal temperatures on the day after their first treatment; in other pyrexic diseases, temperature returns to normal. Arthritis cases may be helped temporarily with, for example, aspirin in the food or water. Endocarditis treatment is ineffective, and cases have a grave prognosis.

Control

Vaccination with killed vaccines is effective. Annual boosters are given to breeding sows when they are not pregnant. In farms with a high risk of infection, several booster doses of vaccine may be required to produce an effective immunity.

Tuberculosis

Tuberculosis is a relatively uncommon infectious disease of pigs whose incidence may rise with rising levels of infection in the cattle population. A number of mycobacteria can infect pigs, but *Mycobacterium avium-intracellulare* is the most common in the UK.

Mycobacterium bovis affects pigs in some countries and could become a problem in the UK if the level of the disease in cattle rises further.

Mycobacterium avium-intracellulare

Incidence

This is relatively uncommon in the UK. There are mostly sporadic cases, but occasional herd infections have been reported.

Epidemiology

Infection is by ingestion of the organism brought by wild or domestic birds or contaminated bedding – especially deep litter, sawdust, peat, and earth floor systems. Pig to pig transmission can also occur.

Clinical signs

These are often subclinical, but infected nodes are found at slaughter – mostly in the nodes of head, neck, and mesentery. Nodes are granulomatous or caseous (Fig. 11.30). There are usually no clinical signs, but generalized infection with weight loss and diarrhoea is seen occasionally.

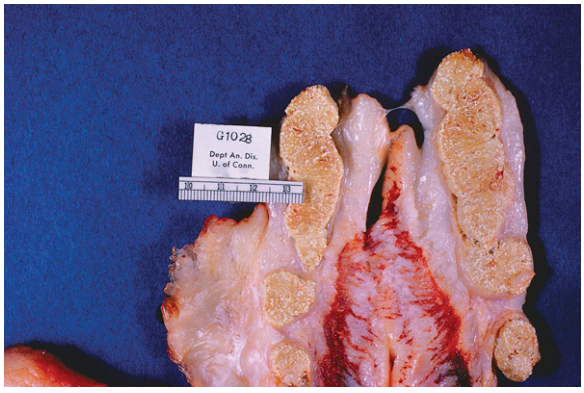

Fig 11.30 – Tuberculosis: caseous lymph nodes. (Courtesy of P.S. Bridge.)

Diagnosis

Base diagnosis on findings at slaughter. PCR identification of the organism is quicker than culture.

Differential diagnosis

Other species of tuberculosis are possible – identify by PCR testing. Perform tuberculosis testing as shown below (see under *Control*).

Treatment

None is available.

Control

Intradermal tuberculosis testing with avian and mammalian tuberculin is carried out by injection at the base of an ear. The test is read 48 h later. Not all infected animals respond to the test, but local swelling and erythema at the site of injection may be seen in positive cases. A contaminated environment must be disinfected and the source of infection eliminated. Owners should be warned of the zoonotic risk of human (often asymptomatic) infection.

Mycobacterium bovis

Incidence

This is uncommon in the UK but could rise with increasing levels of cattle infection.

Epidemiology

The infection mostly originates from land where infected badgers are found. Infection is mostly by inhalation. The organism often locates in the cervical lymph nodes, often with no clinical signs. It may occasionally ulcerate to the skin surface.

Clinical signs

There are usually none. Diagnosis is based on findings in the submaxillary, retropharyngeal, mesenteric, and mediastinal lymph nodes after slaughter.

Differential diagnosis

Other species of tuberculosis are possible. Perform PCR on the organism to identify the species.

Treatment

There is none.

Control

Eradicate the source of infection (e.g. cattle). Beware of zoonotic spread. Tuberculin testing with mammalian tuberculin or with avian and mammalian tuberculin can be carried out.

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12

Outdoor pigs and organic herds

Conventional outdoor pig units

Characteristics of outdoor pig units

The number of breeding sows kept outdoors has rapidly increased in recent times and is still increasing, with 35–40% of breeding herds currently kept outside. Compared with intensive indoor pigs, there are lower capital start-up costs and there is a perception that it is a more 'natural' environment and welfare-friendly. This assists in the marketing of the product to the consumer. There is a need for light, free-draining soil, and low rainfall. Most of production in the UK is concentrated in the south of England, East Anglia, and along the east side of Britain as far north as Aberdeen, where these conditions are met (Fig. 12.1). These areas also allow rotations with arable crops, which are desirable. The performance can rival that of indoor systems, but this requires good stockmanship and management. However, on average, outdoor sows require more food, produce 1.5 piglets/sow per year fewer, and have a 10% higher sow replacement rate. The sows need to be bred to tolerate cold, heat, and sunburn; have good mothering ability; and be hardy. Duroc crosses, Tamworths (Fig. 12.2), Gloucester old spots, and large blacks (Fig. 12.3) have been used for these reasons, although they are prone to become fat and hence have a lower carcass quality. It is possible to convert to organic production with an outdoor unit.

Paddock systems and housing

Paddocks are used for farrowing sows and in-pig dry sows, and service paddocks for weaned sows and boars. There are currently two main systems of arranging paddocks on outdoor pig units: conventional paddocks and the more recent radial system, which has a central handling facility. The service paddocks are composed of a number of sows with a group of boars. Small groups of sows are added to the service paddock on a weekly basis for a period of 3–4 weeks. This group of sows then becomes a stable dry sow in-pig group until they are remixed with sows at the same stage of pregnancy just prior to farrowing to form the farrowing group. Electrified fences are used to contain sows (Fig. 12.4). Housing is in the form of kennels, arcs (Fig. 12.5), tents, or huts, which are relatively inexpensive. The hut is moved to a clean site between litters.

The piglets are normally weaned at 4–5 weeks and are either moved off the field into permanent buildings or into specialist weaner accommodation set up in the field until they reach 10–12 weeks, then they are finished indoors. Outdoor finishing systems are rare. Accommodation for weaners usually comprises a monopitch structure with an insulated roof and a back vent. There is no floor, and an outside run is made from hurdles. A deep straw bed is provided for a group of 30–50 weaners. Feed and water are available on an ad lib basis.

Lower stocking densities result in lower incidence of enteric and respiratory diseases. Potentially, endoparasites may be a problem but annual rotation and anthelmintic programmes have minimized this problem. Nose ringing is now discouraged. Hot air balloons flying above the site can cause considerable stress to the sows, and formal arrangements are usually made to avoid this. Supervision is more difficult in outdoor units, and records regarding whether a dead piglet was stillborn or born alive are usually inaccurate. One feature of outdoor units is that the spread of a contagious pathogen may be relatively slow because of environmental factors and lower stocking densities.

Site selection

Correct site selection is important for welfare, efficiency of production, and minimizing environmental impact. Pigs kept out of doors need a comfortable dry lying area, easy access to adequate food and water, effective prevention and control of injury, conditions that allow the expression of normal behaviour, inspection at least twice a day by a competent stockperson, and facilities that minimize the effect of extremes of temperature.

High winter rainfall results in chilling and, when combined with poor drainage (clay-based soils), results in muddy areas (Fig. 12.6). In muddy conditions, feed is more easily trampled into ground and wasted. The mud can get trampled into the huts, making the bedding wet. Wet conditions increase piglet chilling and the incidence of hypothermia. Waterlogging makes access and movement difficult for pigs and stockpersons. Wet hoof horn becomes soft, and penetration by foreign bodies more common – particularly on stony (flinty) soils – with an increase in the incidence of lameness (Fig. 12.7).

Environmental issues are nutrient losses and soil erosion. Manure deposits can result in high levels of

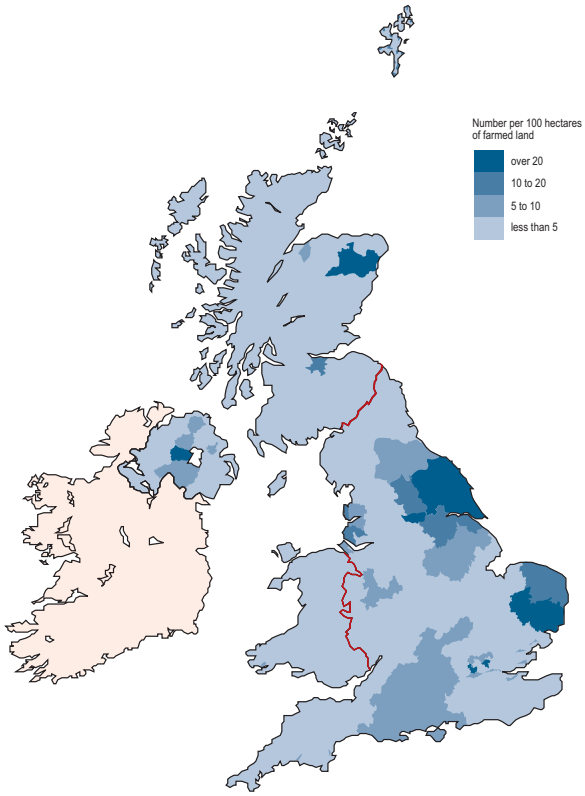

Fig 12.1 – Distribution of pigs in the UK: breeding pigs (sows and gilts). (With permission from Department for Environment, Food and Rural Affairs 2004 Online. Available: http://www.defra.gov.uk.)

Fig 12.2 – Tamworth sow. (Courtesy of R. Potter, deceased.)

Fig 12.3 – Extensive pigs.

Fig 12.4 – Electric fences are used for the paddocks. (Courtesy of R. Potter, deceased.)

Fig 12.5 – Outdoor arcs. (Courtesy of D. Chennells.)

nitrate and phosphorus. Typical annual nutrient loading from outdoor pig units are about 400 kg/ha of nitrogen and 200 kg/ha of phosphate. Nitrate and phosphorus can leach into streams or other watercourses. Sandy and free-draining soils, although advantageous in other respects, are susceptible to leaching. Some areas in the UK have regulations to limit this problem. Soil erosion with loss of top soil is increased with high rainfall and sloping sites. Sloping field may increase the risk of overlying, although gentle slopes can have a positive effect on drainage (Fig. 12.8). Ideally, outdoor pig units should be sited in areas with less than 800 mm of rainfall. Wind and temperature

Fig 12.6 – Appropriate site selection is important to avoid adverse conditions in winter. (Courtesy of R. Potter, deceased.)

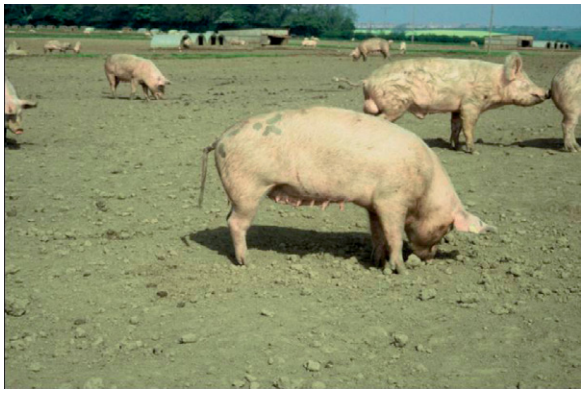

Fig 12.7 – Stony ground without grass may increase stone swallowing. (Courtesy of D. Chennells.)

Fig 12.8 – Arcs on a slope will increase the risk of piglet overlay. (Courtesy of R. Potter, deceased.)

interact to produce chilling, so using a site with some form of natural shelter may be desirable. The maintenance and/or the provision of suitable ground cover facilitates drainage, protects the soil from erosion, and allows grazing. Nose ringing should be discouraged as a management tool to preserve ground cover. The practice of inserting rings

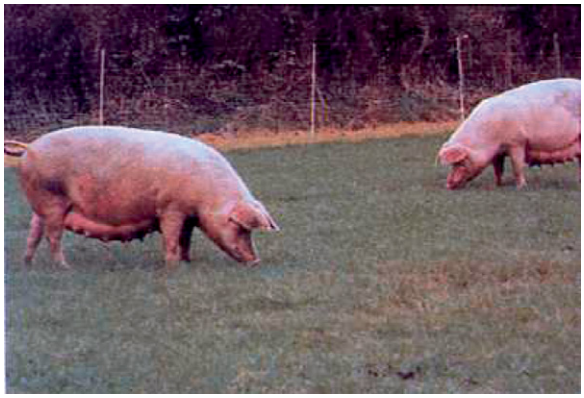

Fig 12.9 – Grass will avoid erosion. (Courtesy of R. Potter, deceased.)

is invasive, painful, and prohibits a normal behaviour. Natural hazards such as recurrent flooding should be avoided. Water supplies and visual impact are also factors to consider.

Management of the site

In ideal conditions, stocking densities up to 25 sows/ha (10 sows/a.) can be used. Reduction of stocking density may be necessary if the quality of the site deteriorates.

Ground cover needs to be well established before pigs are introduced (Fig. 12.9). It is important that outdoor pigs are regularly rotated on to clean sites at least every 2 years, with a break of at least 2 years before returning to the original site, to allow recovery of the site.

Stock should always have huts that are dry and provided with bedding. In muddy and wet conditions, a straw 'door mat' outside the huts can reduce contamination inside the hut. Farrowing paddocks should be sited in sheltered areas to avoid chilling, with the opening of the huts positioned away from the prevailing wind. Draughts can be reduced by the use of plastic curtains. The huts should be sited on level ground and well anchored to avoid being blown over. Shade should be provided, and wallows should be sited and constructed with care to avoid creating excessively muddy conditions. There should be the provision of suitable, isolated hospital pens for ill or injured pigs.

Clinical conditions of outdoor pigs

Highlighted below are some of the conditions that are associated with outdoor pigs. These are discussed in greater detail elsewhere in the book.

Replacement breeding stock

Replacement stock have usually been reared indoors. A period of quarantine, during which the animals can get

acclimatizatized to the harsher conditions and familiar with electric fences before joining the herd, will help reduce the risk of lameness and acute disease after arrival.

Mycoplasmal arthritis

Sometimes, clinical signs appear following the stress of transport and introduction. Affected stock show reluctance to stand, with severe pain and lameness often associated with distension of the hock joint capsule. Individual cases usually respond well to tiamulin or lincomycin by injection. A persistent herd problem may require in-feed antimicrobials (e.g. chlortetracycline).

Osteochondrosis

Degenerative joint disease is a common cause of lameness in outdoor herds. Initial cartilage damage may already be present, and the dramatic increase in exercise often triggers acute lameness. Osteochondrosis can affect any joint, but in outdoor pigs the hip, stifle, hock, and elbow joints are most commonly involved, particularly in animals with upright limb conformation. Reducing growth rates and acclimatization may reduce the incidence.

Swine erysipelas

Although bought-in stock are usually vaccinated, or should be, the vaccine does not protect against erysipelas arthritis, which can be acquired from spores in the environment by newly acquired stock. Affected animals are usually acutely lame, and penicillin by injection is the treatment of choice. Unvaccinated stock may also have the systemic form with skin lesions and vegetative endocarditis.

Penis damage

High libido will cause boars to ride each other, leading to penile damage that will haemorrhage when the boar serves. Young boars managed in outdoor groups are particularly prone to damage to the penis and associated haemorrhage. Treatment is isolation and rest of affected animals, although reintroduction can be a problem. Animals with persistent haemorrhage may have to be culled. Prevention is by grouping young boar teams with in-pig gilts during their acclimatization period and distancing them from gilts in oestrus before they are put to work.

Sunburn and heat stress

Young or new stock delivered from indoor multiplication herds in summer are at high risk from sunburn, although this condition may affect all ages (Fig. 12.10). Sunburnt pigs show reddening of the skin, with severe pain over their back, causing gilts and boars to dip their backs as they walk. Sunburnt gilts will not stand for mating. Heat stress may lead to abortions, infertility, long returns to oestrus, and sows found not in pig. Prevention is by the provision of well-bedded sunshades (Fig. 12.11) and wallows (Fig. 12.12). Open-backed huts will increase the airflow and reduce the internal temperatures.

Fig 12.10 – A piglet with sunburn. (Courtesy of R. Potter, deceased.)

Fig 12.11 – Shades are important to avoid sunburn. (Courtesy of R. Potter, deceased.)

Sows in the service and dry sow paddocks

The majority of outdoor herds rely on unsupervised matings, with boars running with dry sows in paddocks. The lifespan of a boar in an outdoor unit is about 2.5 years, reflecting the workload and often inappropriate condition of the paddocks with uneven ground.

Seasonal infertility

Seasonal infertility occurs during summer and early autumn. The condition is a common and economically important problem. The incidence varies between herds and years. It is multifactorial, resulting from infectious and non-infectious causes. Factors include hot weather, decreasing day length, and leptospirosis infection. It is more common in older sows. Older larger boars may also suffer more from heat stress than younger smaller boars.

Fig 12.12 – Pigs appreciate wallows in summer.

The larger animals lose heat less readily because of increased fat cover and the relatively smaller skin surface area.

Aetiology

Hot weather is known to reduce spermatogenesis and reduce boar libido. In the sow, heat stress can also result in embryonic loss. In affected sows, farrowing rates can drop by 10–15% to 60% or less, with a reduction of pigs per litter. In hot weather, there is a reduction in the appetite of the sow, resulting in a poor condition score at weaning in some cases. Decreasing day length results in increasing melatonin concentrations, which may reduce the luteinizing hormone support of early pregnancy. Serological evidence indicates that exposure to *Leptospira bratislava* is widespread in outdoor pig units. This pathogen can cause embryonic loss, abortion, and reduced numbers of piglets born alive. Porcine reproductive and respiratory syndrome and swine influenza have the potential to cause reduced fertility and may be responsible in some herds. Hot weather also encourages cross-suckling. If fenders are inadequate or in a poor state of repair, piglets can escape and cross-suckle. This makes abrupt weaning more difficult if other piglets continue suckling, hence delaying the return to oestrus. Other factors, such as a change in management or stockperson, should not be overlooked and attributed to this syndrome.

Clinical signs

Regular and less commonly irregular returns to service, sometimes accompanied by a vaginal discharge, occur in this condition. Other manifestations include sows not in pig as they approach their anticipated farrowing date, or sows in pig but at a later date than anticipated because of an unobserved return to oestrus. In some problem herds, pregnancy failure may be a more important factor than failure to conceive. The normal farrowing rate of 80–85% may drop to 65% or less. There is also a reduction in pigs born alive of 0.5 pigs/litter. The primary effects may last 3–4 months. The farrowing pattern, flow of pigs, and production system can be severely disrupted. The boars have increased workloads because of the increasing number of returns. There is increased culling of sows with poor fertility.

Control

Control of seasonal infertility is difficult because of the multifactorial and poorly characterized nature of the condition. The manipulation of the photoperiod to reduce the effects of seasonal infertility is not practicable in outdoor pigs. Heat stress can be reduced by providing adequate shade and wallows, optimizing ventilation of the huts, and reducing the amount of bedding straw. Early morning feeding before the heat of the day may maintain or increase the food intake. Ensuring that there are adequate young mature boars for the summer breeding period and increasing the number of breeding gilts to compensate may reduce the impact of seasonal infertility. Managing the farrowing sows and paddocks to ensure abrupt weaning and reduce the opportunities for cross-suckling may be helpful. Providing extra feeding during the first 4 weeks in the June–September period following mating has been used to improve fertility. Detection and recording of returns and/or pregnancy failures will assist in identifying problem sows. Positive serology to leptospirosis in the absence of vaccination, in association with vaginal discharges and returns to service, may indicate a causal link. In-feed chlortetracycline for 1–2 months can be used to treat infection with leptospirosis on a herd basis.

Reproductive performance

The principles to achieve good performance are the same as for indoor herds. Management factors, sow condition score, sow comfort, stress, and disease all have an impact.

Lameness

Lameness in outdoor adult stock is usually traumatic (Fig. 12.13). Frozen ground and severely poached heavy soil are risk factors. The prevalence of hoof problems is usually fairly low, although this increases in wet conditions because of softening of the horn. Conditions that are fairly commonly encountered include fractures, muscle ruptures, interdigital hyperplasia, penetrating solar lesions, sand cracks, bush foot, and broken toe.

Stony sites with large stones are usually not a problem, but solar injuries are the most common in areas with small flint fragments over a hard subsoil with wet conditions on

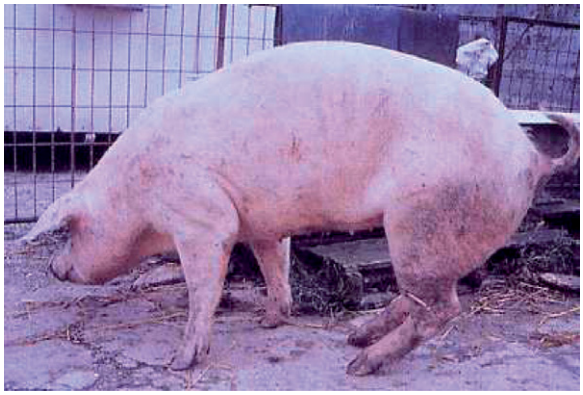

Fig 12.13 – Lameness can be a problem in replacement stock. (Courtesy of R. Potter, deceased.)

Fig 12.14 – Outdoor sows sometimes swallow stones, which may cause gastrointestinal problems.

top. Sand cracks are most common on very dry sandy sites in summer during the transition period from a wetter spring. Chronic osteoarthritis is recognized in boars and may be signified by an unwillingness to work.

Treatment

Early recognition, accurate diagnosis, and appropriate treatment are important to increase the chances and speed of recovery. Treatment, which includes antimicrobial therapy, analgesia, and paring to facilitate drainage, will increase the chances of recovery. With chronic unresponsive lameness, euthanasia should be considered on welfare grounds.

Prevention

The quality of the horn may be improved by biotin supplementation, which may reduce the incidence of sand cracks or broken toes. Reducing the stocking density and site maintenance to reduce obstacles or dangerous fittings may help. Selection of stock with good conformation, followed by an appropriate period of acclimatization, is advised. Initial site selection and management are important.

Necrotic hepatitis

This condition is recognized as a cause of sudden death in outdoor pigs. Necrotic hepatitis is caused by *Clostridium novyi*. Diagnosis is by gross pathology of fresh liver, which has an Aero chocolate appearance, and a positive indirect fluorescent antibody test for the pathogen. Control is by vaccination.

Pyelonephritis

Pyelonephritis is caused by *Actinobaculum suis* and may cause high levels of mortality in some herds. Signs include sudden death, depression, anorexia, and losing condition, and – in severe cases – subnormal temperature with blood or pus in the urine. Treatment in clinical cases is often unrewarding. If there is a herd problem, feeding chlortetracyclines or potentiated sulphonamide (boars) for 4–8 weeks is usually helpful. The boar is responsible for spreading the disease.

Abdominal catastrophes

Gastric and intestinal rupture associated with accumulations of stones and sand is common on some sites (Fig. 12.14). Outdoor sows often chew and swallow stones compulsively. Small sharper stones are a greater risk than rounded pebbles. Sandy sites when floor feeding in wet conditions can lead to the ingestion of large quantities of sand, with destabilization of the gut and risk of torsion. Sows on well-grassed sites are rarely affected, probably because grazing results in better gut fill and contentment. Control measures include regular access to good-quality straw and placing food over a wider area on dry surfaces.

Skin conditions

Mange and lice

Mange and lice are common problems and can lead to polishing of the huts by persistent rubbing. The advent of an ivermectin in-feed premix has improved the control of mange in outdoor herds. Lesions endued by pruritic rubbing are most commonly found at the base of the ears and flanks.

Ringworm

Ringworm (*Trichophyton mentagrophytes*) complicated by staphylococcus dermatitis and forage mites is not uncommon and is often associated with poor-quality straw and rodent contact. The condition is not usually pruritic and appears as a greasy brown discoloration of the skin and hair. Specific therapy is often unnecessary, as the condition is largely self-limiting.

Enilconazole has been used topically in severe cases, but there is no product specifically licensed for use in pigs in the UK. Vermin control in straw stacks and improved straw quality will limit the incidence. Ringworm is a zoonosis.

Bird trauma
Magpies and crows on some sites develop the habit of pecking holes in the skin on the dorsum of the back of outdoor stock, producing a sinus that fills with serum and blood that the birds persistently feed from (Fig. 12.15). Bird control and packing the sinus with Stockholm tar can assist control.

Photosensitization
Severe sunburn may result from skin contact with photosensitizing toxic plants (phytophotodermatitis) such as parsley. Pigs are sometimes allowed into parsley aftermath paddocks. Snout, muzzle, and ventral abdomen are affected.

Intestinal parasites
On permanent sites with no rotation, without anthelmintic therapy intestinal parasites can be a problem, particularly *Ascaris suum*. Fascioliasis can also infect pigs. However, with regular annual or biannual moves to clean ground, combined with in-food worming of adult stock on a twice-yearly basis, internal parasites are an uncommon problem. Screening of pooled sow faeces for worm eggs gives reassurance that parasite control programmes are working.

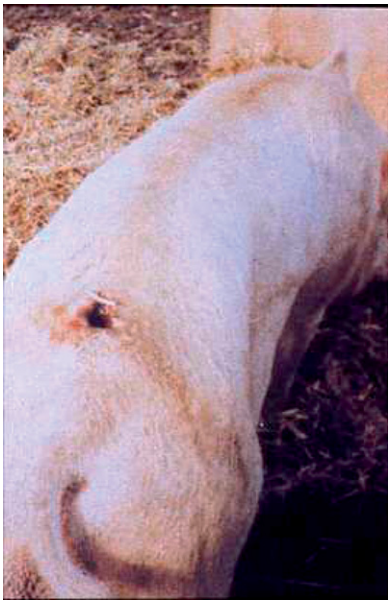

Fig 12.15 – Bird trauma causing a skin sinus. (Courtesy of R. Potter, deceased.)

Granulomatous mastitis
Chronic granulomatous mastitis, sometimes with open sinuses, is a common condition in outdoor sows. Risk factors include trauma to the teat by the piglets or poorly designed farrowing arcs. A course of penicillin treatment by injection at weaning may be helpful.

Vulval biting
This condition is usually sporadic but can occur in small outbreaks, usually associated with underfeeding, overstocking, or the presence of a bully sow (or sows).

Conditions affecting farrowing sows

Vulval biting
The enlarged vulva of late pregnancy increases the risk of vulval biting. Attention should be given to management factors and factors causing close contact and competition between the sows, such as the feeding routine. Recurrent offending sows should be culled.

Cannibalism
This is a sporadic problem, but an offending sow can cause havoc. Offenders must be identified and culled. Attention should be given to reducing the stress of lactating sows.

Dystocia
This is uncommon, although – because there is often a delay in intervention as a result of reduced accessibility and observation – the prognosis for the sow in some cases may be poor if toxaemia has developed.

Agalactia and mastitis
Mastitis–metritis–agalactia and mastitis are usually uncommon. Some outbreaks of mastitis have been associated with infected wallows. Exercise, higher-fibre diets and lower-energy densities, and reduced pathogen challenge may reduce the risk. However, peracute mastitis is sometimes associated with hot weather, when there is a reduced food intake. In-feed medication may be required if the prevalence is high, in addition to reducing the heat stress and encouraging the intake of food. Poor milk let-down with or without mastitis is a common problem in hot weather. Over-fat sows with a high-energy diet and a high feed intake are most at risk. Lush grass and oestrogenic clovers may also contribute. Ergot (*Claviceps purpurea*) on headed rye grass in late summer may also be a risk factor, as the alkaloid ergotometrine can produce agalactia if ingested in quantity.

Piglet conditions
Piglets are kept outdoors until weaning (Figs 12.16 and 12.17).

Fig 12.16 – Sow and piglets. (Courtesy of D. Chennells.)

Fig 12.17 – Sow and piglets inside an arc. (Courtesy of D. Chennells.)

Overlying and starvation

This is a major cause of mortality in the first 24 h of life, although this condition may be frequently misdiagnosed. Differential diagnoses include born dead, hypothermia, *Escherichia coli* diarrhoea, and recording errors. Extremes of weather, arcs on slopes, sow comfort, and disturbances are risk factors. Excessive provision of straw may ensnare the piglets, reducing their movements to avoid the sow and prevent piglets escaping into the corners or behind the rails. Some sows may dig a hollow as a nest in the arc, particularly where the soil is soft, which may result in piglets rolling in and being inadvertently crushed. Ringing has been used, but a more acceptable alternative is the provision of a hard floor covering.

Some sows show excessive nesting behaviour accompanied by agitation and restlessness, which increases the risk of overlay. Increased sow movement also occurs with discomfort such as mastitis or piglet teeth injuries. Lack of insulation of the arc results in piglets spending more time in the central area to avoid chilling in winter and overheating in summer. This may increase the chance of overlay. Ventilation and draught avoidance by the use of adjustable rear vents and door flaps is advisable. The relative size of the arc in relation to the size of the sow or gilt may have an influence. A large sow in a small arc may increase the chance of overlay. A small gilt in a large arc may encourage chilling and/or increased movement.

Some arcs have farrowing rails to provide piglet protection, but these easily get blocked with straw and may hinder piglet attempts to escape the sow. In very wet and poached areas, floorboards covered with straw can reduce overlay but they must be turned or disinfected between litters. The arc should not be positioned on a slope, otherwise there is migration of bedding and piglets and an increase in overlay. The arc should be positioned to avoid cold prevailing winds in winter and reduce direct sunlight exposure in summer. Records can be misleading and inaccurate. Establishing the true extent of the problem may be difficult.

Piglet enteritis

Piglet enteritis in general is less of a problem than in indoor herds. Isolation of litters in individual arcs, routine relocation of farrowing groups, and new straw bedding between litters are thought to be the reason.

Clostridial enteritis

Oubreaks of haemorrhagic enteritis caused by *Clostridium perfringens* type C are reported on outdoor units and may cause high levels of loss in the first week of life. Older litters may be affected by types A and B. Sow vaccination is possible to protect against types B and C. Antitoxin may be useful in a new outbreak to give protection to young litters. In newly established herds, this is a relatively common condition and prophylactic vaccination should be considered. Protection of the litter is dependent on colostral intake. Prefarrowing booster vaccination at least 3 weeks before farrowing is required.

Colibacillosis

Some outdoor herds do have a problem with *E. coli* enteritis. Attention to hygiene in the farrowing huts is necessary. In recurrent cases, sow vaccination should be considered.

Coccidiosis

Caused by *Isospora suis*, this is a common cause of scour in outdoor litters from 10 days of age up to weaning. Signs include a pasty grey, non-fatal diarrhoea that results in variable weaning weights. It is usually more severe in summer. Oocysts can build up in wallows and farrowing huts. Diagnosis is dependent on histopathology and speciation of the oocysts. Differential diagnoses include *E. coli* and clostridial enteritis. Prevention and control in affected herd is best achieved with in-feed coccidiostats,

although they are not licensed in pigs in the UK. The sows and the piglets should be treated to reduce the output of pathogenic oocysts. Potentiated sulphonamides reduce the incidence of the condition but are less effective, and attention to hygiene in the farrowing huts is advocated – this includes disinfecting the arcs and fenders using an appropriate disinfectant.

Joint ill

Joint ill is a relatively uncommon condition in outdoor litters, although there is an increased incidence in farrowing huts with solid floors. Teeth clipping and docking techniques should be reviewed and attention paid to bedding quality and dryness at farrowing. Treatment of affected litters is by appropriate antimicrobials by injection.

Recently, *Streptococcus suis* type 14 has been identified as a cause of polyarthritis in piglets from 2 weeks upwards. The incidence can be high in naive herds until herd immunity rises. The response to injectable amoxycillin is usually good. In severely affected herds, in-feed medication of sows has reduced the incidence.

Tetanus

Tetanus occurs sporadically in outdoor litters and is usually associated with poor hygiene when teeth are clipped and tails docked.

Exudative dermatitis (greasy pig disease)

This is uncommon in outdoor litters. Risk factors include poor teeth-clipping technique and competition between piglets after weaning.

Sunburn

Sunburn can occur in overexposed piglets.

Predation

Predation by foxes can cause serious losses in some herds (Fig. 12.18). Control by trapping or shooting, combined with an electric fence around the parameter, will reduce losses (Fig. 12.19).

Others

Outbreaks of disease caused by *S. suis* types 1 and 2, *Haemophilus parasuis*, and erysipelas in piglets of 2–3 weeks of age have been reported.

Outdoor weaners

The principles of rearing are the same as indoors, with similar potential health problems, but the system of management can increase the risk of certain conditions. Temperature and ventilation are often suboptimal and large fluctuations may occur, especially in spring and autumn. Recurrent use of the same housing and floor space encourages the build up of potential pathogens. Vices are not common. Interruptions of water supplies because of adverse weather conditions can result in salt poisoning. The bedding straw can generate high levels of dust.

Fig 12.18 – Predation by foxes can result in piglet losses. (From Pritchard et al (2005), with permission.)

Fig 12.19 – Electric fences can be used to keep predators out but do not always keep piglets in.

Respiratory disease

Aetiology

Porcine reproductive and respiratory syndrome and swine influenza, *Actinobacillus pleuropneumoniae*, *H. parasuis*, pasteurellae, *Bordetella*, streptococci, and *Mycoplasma hyopneumoniae* have been diagnosed in outdoor weaner units.

Predisposing management and environmental factors

Set stocking of weaner arcs results in overstocking as the weaners grow, and the need for increased ventilation is often overlooked. Mixing different litters of different ages increases the risk of respiratory disease. It is often difficult to balance the need to avoid chilling with the needs for ventilation when the weather is extremely changeable.

Control and prevention

Reducing the stocking density and group size, use of insulated arcs with good management of ventilation, reduction of cross-contamination between kennels and runs by physically separating subpopulations of weaners, and early detection of sick animals will help to prevent respiratory disease. Treatment by using in-feed medication or water medication may be necessary in the short term to control an outbreak. Post-mortem assessment and accurate diagnosis should be encouraged followed by appropriate control and prevention measures, which may include vaccination.

Other conditions include postweaning multisystemic wasting syndrome, porcine dermatitis and nephropathy syndrome, mange, and ringworm (*T. mentagrophytes*).

Veterinary health plans for outdoor pigs

Health plans have been covered in detail in Chapter 2 (*Population medicine*).

The following are of particular importance to outdoor units.

- Vaccination programmes:
  - porcine parvovirus – gilts prior to service, with booster doses if necessary
  - erysipelas – all stock before or on arrival, followed by boosters
  - clostridial diseases – all stock
  - *Escherichia coli* – if necessary.
- Anthelmintic programmes
- Rotation of site:
  - rotation of fields – limit use to 12 months
  - alternation of service area – rotate to ensure good condition.
- Coccidiosis control
- Acclimatization protocols
- Biosecurity protocols
- Arc or hut management protocols
- Movement of arcs regularly: between every litter for farrowing arcs
- Removal and disposal of straw beds: at time of moving arc

Organic pig production

Aims of organic pig farming

Organic production requires an outdoor free range system, and all the desirable characteristics described earlier apply. The stated aims of the principles and methods used in organic farming are to help sustain or build soil fertility, protect and enhance farm conservation and wildlife, protect animal welfare, and ensure the ethical treatment of animals. This includes sustainable crop rotations, use of manures and vegetable waste, and avoidance of mineral fertilizers and pesticides. In addition, animal husbandry techniques that meet the animal's physiological, behavioral, and health needs are encouraged. Optimization rather than maximization is emphasized. Statutory requirements required by law apply to organic farms. There are approximately 53 000 organic pigs in the UK. Supermarkets are responsible for the sale of more than 70% of organic pig meat. Some supermarkets are still importing significant amounts of pig meat (up to 75%), particularly pork from Germany and bacon from Holland.

Veterinary inputs in the form of antibiotics, antiparasiticides, and vaccines are acceptable in the treatment of clinical disease or where there is an established risk of disease. When prophylactic or metaphylatic (whole group) treatments are used on a herd basis, the prescribing veterinary surgeon is required to justify their use by identifying specific risk factors and to explain why they cannot be reduced by other means such as management changes. The prophylactic use of antibiotics, organophosphates, and avermectins are prohibited. The use of alternative or complementary medicines is encouraged, and withdrawal times of conventional (allopathic) drugs are extended. There is a limit to the number of allopathic treatments an animal can receive per annum before losing organic status. Recording systems are required for stock movements, disease events, and mortalities. All veterinary and non-veterinary treatments must be recorded in detail, including statutory and organic withdrawal periods. A veterinary health plan is required. If meat is to be marketed as organic, the animals must be slaughtered and butchered by organic-registered establishments that are subject to an annual inspection.

Organic certification

The Compendium of Organic Standards (Defra) is the standard for organic food production and must be complied with in the UK. It is based on and complies with the European Council Regulation (EEC) no. 2092/91. Farm assurance and annual farm inspection are carried out by private registered organic certification bodies. These bodies can develop their own stricter standards than the Compendium of Organic Standards and/or those set down in the European Council regulation. It is essential that the veterinarian is conversant with the requirements of the organic certification body used by the producer.

Conversion

Farms must go through a conversion period when the land is managed to full organic standards. This period is normally 2 years.

Organic standards

Below are some examples of the organic standards that are required by the organic certification bodies.

Organic pigs

- Conventionally reared pigs can never be used for organic meat production.
- With an existing non-organic herd, sows can produce piglets classified as organic if the sows are managed to full organic standards on organic land from service onwards. The sow can never be sold as organic.
- Animals that are non-organic can never achieve organic status themselves.
- Non-organic boars can be bought in for breeding provided that they are fed to full organic standard. Artificial insemination is allowed.
- There is a 10% maximum replacement rate of sows from non-organic sources.

Feeding

- Pigs must be fed organically produced feed (100%).
- All bought-in concentrate must come from an approved source.
- Synthetic amino acids cannot be used in organic pig rations.
- The daily ration must contain some roughage in the form of fresh grass, hay, or silage.
- Antibiotics, coccidiostats, medicinal substances, growth promoters, and any substance intended to stimulate growth or production are not allowed.

Disease prevention and veterinary treatment

- A veterinary health plan is required. It is a fundamental part of organic livestock management. It must identify all potential animal welfare issues and put in place a comprehensive framework to ensure that flock or herd health is maintained and improved, while the use of any routine veterinary products is reduced. It is strongly recommend that this is drawn up in consultation with the farmer's veterinarian.
- Disease prevention is based on breed selection, nutrition, and free range conditions (exercise, appropriate stocking densities, and rotation).
- Herbal medicine, homeopathy, and trace elements and vitamins should be used in preference to chemically synthesized allopathic veterinary medical products, provided that they are effective.
- Allopathic medicines can be used only under veterinary supervision.
- Vaccination is permitted in the case of a known risk identified by a veterinarian.
- Monovalant vaccines are preferred if available.
- Preventive use of allopathic medicine and antibiotics, growth promoters (including copper supplements and probiotics), and hormonal products to synchronize or induce oestrus are prohibited.
- All treatments, diagnoses, and withdrawal periods must be recorded.
- Withdrawal periods for allopathic drugs are generally as follow:
  - 48 h where no withdrawal is specified,
  - 7 days when a withdrawal period of 0–48 h is specified,
  - three times the withdrawal period when the specified withdrawal period is 2–18 days,
  - 56 days for specified withdrawal times of between 18 and 27 days,
  - twice the withdrawal period for specified withdrawal periods of 56 days or more, and
  - withdrawal times three times the statutory withdrawal periods.
- If a sow or boar is treated more than three times a year with allopathic treatments (courses of treatment), the organic status of the animal is lost. (Vaccinations, parasite treatments, and statutory disease control measures do not count.)
- Grazing management and strategic pasture rotation should be used as part of an endoparasite control programme.
- Faecal egg counts and abattoir checks on milk spot liver and lung lesions should be used to monitor the success of endoparasite control programmes.
- Advice regarding the permitted use of or restrictions that may apply following the use of certain allopathic drugs can be obtained from the certifying body.

Husbandry

- Tail docking, teeth clipping, teeth grinding, castration, nose ringing, and prophylactic use of iron injections are prohibited.
- Artificial insemination is allowed.
- Pigs must have continuous access to pasture or an open air exercise area or open air run.
- The final fattening stage (maximum one-fifth of life) can take place indoors.
- Sows must be kept in groups except in the late stages of gestation and during suckling.
- Piglets must not be kept on flat decks or in cages.
- The recommended weaning age is 8 weeks, although 40 days is the accepted minimum. Weaning at less than 40 days of age is allowed only in an emergency.
- Over the summer months, wallows and shade must be provided.
- Organic straw is recommended for bedding, but currently non-organic straw can be used.
- Rotation is recommended, with pigs spending no longer than 6 months on one plot of land with a 4-year rotation. Woodlands can be used.

- There is an annual limit on manure application rates, expressed as nitrogen/ha. These can be expressed in stocking rate equivalents, for which tables are available.

The veterinary health plan

All converting farms should establish a herd health plan as part of the livestock conversion plan. This should be done in collaboration with a veterinary surgeon. If a specialist in complementary and alternative medicine is involved in health planning on the farm, the veterinary surgeon and the adviser in complementary medicine should collaborate in the formulation of the health plan. This collaboration should identify the areas where complementary medicine would be used instead of conventional medication.

A herd health plan should include at least the following aspects.

- A description of the herd (production system, number of animals, and production targets).
- A description of all routine health and husbandry measures to prevent disease or injury in stock (including management of passive and active immunity).
- Identification of changes that are required to remedy identified problems, such as rotation plan, appropriate stocking densities, improved hygiene practices, and improved ventilation of housing.
- An outline of a feeding plan, rotation plan, and grazing policy.
- Definition of the housing needs and breeding management.
- An assessment of existing recording systems and plans to improve them if necessary.
- Establishment of a feedback system for post-mortem data from the abattoir.
- An assessment of disease levels and stock condition. This should identify all persistent mineral deficiencies, disease, and parasite health problems.
- An assessment of the animal welfare status on the farm, using the five freedoms. The five freedoms are:
  - freedom from hunger and thirst;
  - freedom from discomfort;
  - freedom from pain, injury, and disease;
  - freedom to express the most normal behaviour; and
  - freedom from pain and distress.
- A plan to reduce disease or injury levels or routine medication if considered necessary, with clear targets and review dates.
- A plan to improve the animal welfare situation on the farm if considered necessary, with clear targets and review dates.

Herd recording and health monitoring

Herd recording is often poor in outdoor units in comparison with indoor systems, where observation and surveillance are easier. The organic standards require that detailed records should be kept of all veterinary or complementary treatments carried out. It is, however, important that farrowing records are also kept in good order, so as to recognize problems early when they arise.

To meet the organic standard requirements, a pig producer must have a recording system for disease treatments, indicating drugs used, with quantities and withdrawal periods included, and outcomes of treatment. Treatments should include any prophylactic or metaphylactic use of drugs as well. For the purposes of positive health care and herd health planning (also required in the organic standards), a pig producer should record farrowing data and entries and exits of sows, with reasons for culling or death clearly indicated. All organic pig producers should also establish a routine feedback of post-mortem information from the abattoir after each batch of pigs is slaughtered.

Checklist for outdoor pigs

General

- Suitability of the land
  - Drainage
  - Level
  - Shelter
  - Rotation
  - Proximity of roads, people, and pig units
- Breed
  - Sows
  - Boars
- Nose ringing
- Electric fences
  - Condition
  - Training and acclimatization
- Protection from sunlight
  - Shade
  - Wallows

Farrowing paddocks

- Stocking density
- Ground cover
- Vermin and predator control
  - Foxes, rats, and birds
- Accommodation
  - Type (huts or arcs)
  - Size
  - Siting (level, prevailing wind direction)
  - Standard
    Construction state of repair: huts or arc, fenders
    Solid floor or earth
    Bedding type: quality (dry or wet) and quantity (too much or too little)

  - Air flow (draughts, condensation, door flaps, vents)
  - Lying patterns
- – Management
  - Moving after each farrowing
  - Straw (replacement, burning)
  - Disinfection protocol
- – Shade and wallows
- Feeding
- – Troughs: siting, state of repair, wastage
- – Feed quantity, quality, frequency, and method
- Water
- – Nipple or trough
- – Supply
- Fencing
- Sows
- – Condition score

Farrowing and weaning

- – Reproductive indices
- – Disease indices
  - Lameness
  - Mastitis–metritis–agalactia
  - Culling rates
- – Mortality
- Piglets
- – Born dead
- – Mortality rates
- – Predatory losses
- – Overlying
- – Management at farrowing
- – Iron injections
- – Teeth clipping
- – Tail docking
- – Savaging
- – Weight at weaning
- – Missing (stolen?)

Dry sow in-pig paddocks

- Stocking density
- Ground cover
- Accommodation
- – Type (huts or arcs)
- – Size
- – Siting (level, prevailing wind direction)
- – Standard
  - Construction state of repair: huts or arc
  - Solid floor or earth
  - Bedding type, quality (dry or wet) and quantity (too much or too little)
  - Air flow (draughts, condensation, door flaps, vents)
- – Management
  - Moving
  - Straw (replacement, burning)
  - Disinfection protocol
- – Shade and wallows
- Feeding
- – Troughs: siting, state of repair, wastage
- – Feed quantity, quality, frequency, method
- Water
- – Nipple or trough
- – Supply
- Fencing
- Sows
- – Returns to service
- – Pregnancy diagnosis
- – Condition score

Mid pregnancy

- – Reproductive indices
- – Disease indices
- – Mortality

Boars and service paddocks

- Management
- Boar usage
- Boar condition score
- Group sizes
- Stocking density
- Number of boars
- Age of boars
- Accommodation
- Feeding and water
- Fencing
- Ground cover and condition
- Performance
- Artificial insemination usage

Gilt paddocks

- Weight and condition score
- Feeding and water
- Accommodation
- Management
- Training and acclimatization

Weaners

- Accommodation
- – Stocking density
- – Bedding
- – Water
- – Feeding
- – Ventilation
- – Quality of fittings
- Weaners
- – Condition
- – Growth rate
- – Weight on entry and exit
- – Feed consumed
- – Mortality

Hospital pens

- Environment
  - Well ventilated but not draughty
  - Straw bedding to assist thermoregulation
  - Well lit
  - Clean fresh water should be available at all times
  - There should be an effective cleansing and disinfection policy
  - Isolation pens (infectious conditions)
- Health
  - Presumptive diagnosis
  - Treatment plans and records
  - Defined end points
  - Signs
  - Morbidity
  - Mortality
  - Post-mortem results

Diseases checklist

Replacement breeding stock

- Mycoplasmal arthritis
- Osteochondrosis
- Swine erysipelas
- Penis damage
- Sunburn and heat stress

Service and dry sow paddocks

- Seasonal infertility
- Lameness
- Necrotic hepatitis
- Pyelonephritis
- Abdominal catastrophes
- Skin conditions
  - Mange and lice
  - Ringworm
- Bird trauma
- Photosensitization
- Intestinal parasites
- Granulomatous mastitis

Piglet conditions

- Overlying and starvation
- Piglet enteritis
  - Clostridial enteritis
  - Colibacillosis
  - Coccidiosis
- Joint ill
- Tetanus
- Exudative dermatitis (greasy pig disease)
- Sunburn
- Predation

Conditions affecting farrowing sows

- Vulval biting
- Cannibalism
- Agalactia and mastitis
- Mastitis–metritis–agalactia
- Endometritis

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FURTHER INFORMATION

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13

The pet pig

First, the torpedo shape of the pig defies all attempts to grip it during a rugby tackle. Secondly, pigs, if successfully restrained, possess the best panic alarm of all, producing a scream that can induce instant tinnitus. Thirdly, any sedation of the pig is purely temporary; the tricky bit is that there is virtually no way of ascertaining when this temporary period will end, and that at any time without warning the pig will elect to part the scene, not infrequently leaving a trail of clattering instruments in its wake. Finally once they have gained a head of steam you'll never stop a moving pig.

Dr Reg Goodwin, quoted by David Chennells (2004)

Advise clients that it is normal for pigs to squeal when handled.

Steve Borsberry (2004)

Introduction

Pet pigs are increasing in popularity as both indoor and outdoor pets. Pet pigs have included breeds such as Landrace and Tamworth breeds, and there is an increasing trend for keeping exotic breeds such as the Vietnamese pot-bellied pig (Fig. 13.1). The Vietnamese pot-bellied pig is currently the most popular breed, followed by the kune-kune breed from New Zealand. The Yucatan breed from Mexico is also kept in the UK. Vietnamese pot-bellied pigs originated in the forests of South-East Asia and were originally black. They can live 15–20 years and when fully grown at 18 months of age may weigh over 65 kg, although there is a wide weight range. Sexual maturity is around 2 months of age in the male and 3 months in the female in Vietnamese pot-bellied pigs. Weaning age is usually 6–8 weeks of age. The kune-kune are small short pigs having a colour range from black to marble and cream (Fig. 13.2). They generally have a pleasant temperament. Pet pigs are kept indoors and out of doors. They can be house-trained like a dog. Clients often treat their pet pigs as other companion animals, and they will have special needs in comparison with commercial pig farmers.

Legal requirements

The information in this section is used as a guide to alert the reader to aspects of pig keeping they may not have considered in England. All the details should be checked with the Department for Environment, Food and Rural Affairs (Defra) or other relevant regional and national government organizations to ensure accuracy. In order to reduce the risk of disease spreading, tracing of pig movements is essential and much of the legislation is concerned with the movement of pigs.

In England, premises where pigs are to be kept require a county parish holding number before pigs are moved on to the property. This can be obtained from the Rural Development Service. Pig movement usually takes place under a General Licence, which sets out the condition of movement. These licences can be obtained from the Defra web site or the local Animal Health Divisional Office. Pig movements must be accompanied by this movement document. Pig movements to another holding, a market, or slaughter require an Animal Movement Licence 2 (AML2) form to be completed. A copy is sent to the local trading standards animal health department. This provides details of where the animal has come from and where it is going to. An Individual Movement Licence is required to move pigs from a market and is issued at the market by a local authority trading standards officer. The person selling the pigs will be responsible for supplying an AML2.

Once a pig arrives on the property, the holding is under a standstill. In the case of pigs already on the holding, no movement of pigs can occur for 20 days. In the case of cattle, sheep, and goats on the holding, there is a 6-day standstill. Cattle, sheep, and goats moving on to a holding will impose a 6-day standstill on any pig on the holding. Once the pigs are on the holding, they have to be registered with Defra at the local Animal Health Divisional Office. When the pigs are registered, a herd mark is allocated. Whether the owner keeps one pig or a commercial herd of pigs, the owner must be registered with Defra.

Farm records need to be kept that indicate the date of movement, the identification mark, the number of pigs, the holding from which moved, and the holding to which

Fig 13.1 – The Vietnamese pot-bellied pig. (Courtesy of K. Mueller.)

Fig 13.2 – The kune-kune pig. (Courtesy of K. Mueller.)

moved. This has to be done within 36 h of the movement taking place. These records have to be kept for 6 years after pigs are no longer kept on the holding. At least once a year, a record must be made of the number of animals on the holding.

Pig identification rules

All pigs under 1 year of age moving direct to slaughter are required to be identified with their Defra herd mark. Pigs under 1 year of age moving to another holding should be marked with a temporary paint mark (e.g. a red line, blue cross, or blue circle), which must last until the pig reaches its destination. Combined with the movement document, the temporary mark identifies the holding from which the animal has moved.

All pigs over 1 year of age moving to any destination require identification with the Defra herd mark of the dispatching premises. The Defra herd mark should be slapped on to each shoulder. Alternatively, the pigs may be identified with an approved ear tag or ear tattoo containing their herd mark.

Identification methods

The slap marker consists of slap marker plates. Each plate consists of a series of letters and numbers that make up the herd mark. Each figure is made up of pins that, when covered in ink and slapped on the shoulder of the pig, leave a long-lasting mark. It is important that sufficient ink is applied and that the figures on the slap marker are replaced if the pins become damaged. The slap marker should be kept clean. It is important that a clear herd slap mark is created on both shoulders of the pig.

The tattoo should be on the outside of the ear and easy to read. The letters should be clean and sharp. If the characters are broken or worn, they should be replaced. The ear should be cleaned with surgical spirit or methylated spirit to remove dirt. Ink should be applied to the pins and the ear (a toothbrush works well). The ear is placed between the jaws of the tattoo pliers, avoiding any prominent veins. The imprint is made quickly and firmly by closing the pliers and the ear rubbed vigorously to ensure that the ink is embedded in the puncture wounds. Signs of infection, including discomfort, should be treated promptly.

Pig-walking licences

A pig-walking licence is necessary in order to take a pet pig for a walk and is issued by the Animal Health Divisional Office. The route has to be approved, and a Defra veterinarian will visit and inspect the route to perform a risk assessment. The walk will have to avoid certain areas, such as fast food outlets or other animal holdings, that may present a risk of disease spread or be at risk of disease spread. The licence must be renewed annually.

Feed

It is illegal to feed catering waste or animal by-products to a pig because of the risk of disease. Swill feeding has been banned. The term *catering waste* includes all waste food – including used cooking oil – originating in restaurants, catering facilities, and kitchens (even those preparing vegetarian food), including household kitchens.

Behaviour

Pet pigs may grind and chomp their teeth at any time, and this does not necessarily indicate pain, hunger, or distress. They are curious animals and will investigate the environment around them. Indoors, pigs are very inquisitive and may chew through cables and plastic pipes – a problem prospective owners may not have considered. Edible treats should not be used as rewards, as the pig can become obsessive. They have an acute sense of smell but only moderate hearing and eyesight. To maintain a comfortable temperature, pigs must have access to bedding when

the weather is cold and access to shade and wallows when the weather is hot. Provision of appropriate housing is essential. They can be successfully housetrained if kept indoors. They have a strong rooting instinct; if given an opportunity, they will destroy lawns and flowerbeds. They can also burrow under fences.

There is a dominance hierarchy, which is evident when there are limited resources such as food or preferential resting places. Fighting will occur when new pigs are introduced or reintroduced after even a short period of absence. Azaperone can be used to prevent excessive fighting in these situations.

Boars produce a potent-smelling pheromone in their saliva that is distinct and unpleasant. The pheromone is no longer produced following castration, and the smell disappears. Boars may salivate profusely and stomp their feet to indicate their dominance. This can be a prelude to aggressive behaviour, which may be directed towards the owner or a pet. Annual trimming of the boar's tusks is advisable. Some boars kept together will mount each other.

During oestrus, the sow will adopt a rigid stance in the presence of a male or with pressure applied to the lumbar area. During this time, the vulva becomes enlarged and a slight clear vaginal discharge may be observed. The female animal may be difficult to control around this time. Nest building will be observed in pregnant females close to farrowing. A previously tranquil well-behaved animal may become aggressive before, during, and following parturition. This protective mothering instinct will persist until after weaning of the piglets. Care is therefore needed when handling or approaching her. Some gilts (and even sows) may savage the newborn piglets during farrowing because of fear or stress. Removal of the piglets until farrowing is complete may be the best course of action if excessive aggression is observed. Azaperone can be used to sedate the mother during or following farrowing when reintroducing the piglets. Azaperone does not interfere with farrowing.

Husbandry

Reproduction

In pot-bellied pigs, sexual maturity is usually attained at 2–4 months, although the preferred age for first breeding is 6–7 months. The oestrous cycle is 19–22 days. Standing heat is 12–18 h in the gilt and 24–48 h in the sow. Gestation length is 109–114 days, and the litter size is usually 4–6 (range 1–12). Total or partial failure of lactation may occur, particularly in obese animals. Cow colostrum can be used as a colostrum substitute. Commercial piglet milk substitutes are available. The neonatal piglet should be fed every 3–4 h with approximately 10–15 mL of replacer by bottle. Weak piglets can be given 2–5 mL of 40% dextrose by mouth 6–8 times a day. By 1 week of age, the piglets can usually drink from a flat pan but it should be weighted down with a brick to prevent it being tipped over by the piglet. The piglets are usually weaned at 6–8 weeks of age, with creep feed being provided from 1 week onwards.

Feeding

Pet pigs should ideally be fed several small meals a day but should not be allowed to become over-fat, particularly the Vietnamese pot-bellied breed. For mature adults, a 12% protein mix fed at 2–3% of body weight per day is recommended. Treats such as fruit and vegetables make a useful addition. Chocolate and crisps should be avoided. Most commercial diets will meet the needs of a pet pig, although a source of fibre is recommended such as hay, straw, or root vegetables. Feeding should be according to condition score and demand. Close observation of the body condition and fat deposition is recommended to avoid obesity. Pot-bellied pigs deposit fat in the jowl area and around the tail head. The ideal condition for a pot-bellied pig is when the ribs can be felt but not seen.

During gestation and lactation, the sow needs more energy, protein, and minerals. Commercial rations are available that are specially formulated to meet the demands of pregnancy and lactation. The pregnant animal should be fed 2–2.5% of her body weight daily until the last month of pregnancy, when this should be increased to 3%. The sow should gain approximately 20% of her body weight during gestation, with over half of this gain occurring in the last 5 weeks. A lactation ration should be fed from farrowing to weaning.

With piglets, solid food (creep feed) is usually introduced at 1 week using an ad lib starter ration. Piglets are usually weaned at 6–8 weeks of age on to a grower ration. The grower ration usually contains 14% protein, but a switch to 12% at 4 months of age is better for pet pigs as rapid growth is not desirable. The growers are fed approximately 3–5% of body weight daily. Mature weights should be attained at approximately 8–10 months of age. Boars should be fed a 12% protein ration at 2–3% of body weight daily. Careful monitoring of the weight and condition score of the boar is desirable to avoid the animal becoming overweight.

Nutritional deficiencies

Commercial diets usually contain adequate amounts and correct ratios of minerals and vitamins for larger pigs. It has been suggested that vitamin E and selenium supplementation may be advisable (avoid overdosing with selenium). Iron deficiency can occur when pigs do not have access to soil, and iron injections to young piglets by

intramuscular injection into the hamstring muscles are recommended.

Exercise

Exercise should be encouraged. One hour a day in the form of two exercise periods is recommended. This provides stimulation, keeps the animal fit, reduces excessive weight gain, and provides additional wear for the horn of the feet. Collars and leads are required when taking the pig for a walk. A harness that passes around the neck and thorax provides the best control.

Housing

A dry, clean, draft-free environment is required. This may be indoors or outdoors. Pigs are sensitive to extremes of hot and cold. The zones of thermal comfort are as follow: adults, 15–20°C; suckling piglets, 25–30°C; weaned piglets (6–12 weeks), 25–30°C; growing pigs (12–16 weeks), 15–21°C; and older pigs (16–26 weeks), 13–18°C. Keeping animals in groups and supplying them with straw enable animals to be thermally comfortable in lower ambient temperatures.

Outdoor pigs are often kept in weatherproof wooden shelters or arcs. Straw is the most common bedding material used in the shelters. Replenishment is required, as some straw will be consumed by the pigs. Replacement will be required periodically, as the quality of the bedding declines because of soiling and increasing moisture content. The floor should be insulated. In hot weather to avoid hyperthermia, shades, mud wallows, water pools, and fans can be used to keep the animal cool. Shades and wallows will also help to avoid sunburn. There should be a bedded area for resting and an area for defecating. Housing should be waterproof and easy to clean and disinfect. Slippery floors should be avoided. Perimeter fences should be robust and extend well below ground level to discourage burrowing.

Pigs kept indoors are odourless (apart from a mature boar), and blankets can be used for bedding. Pigs can be trained to use a litter box or trained to go outside to urinate or defecate. Pigs often urinate when drinking, so the water supply should be placed near the litter box.

Other considerations

Prospective owners should be made aware of the appreciable amounts of faeces and urine that pigs can produce. An 80-kg pig may produce 4 L of urine a day and a kilogram of faeces, which can result in offensive odours. Noise can also be considerable, particularly where several animals are kept.

The clinical examination (see Chapter 2)

History

The age, sex, breed, gender (whether the animal has been neutered), vaccination status, diet, housing, recent contacts, and exercise should be established. The duration, progression, signs observed, and severity of the disease should be established.

Handling

Catching an outdoor pig can be difficult, and food may be helpful. If an arc is being used to house the pig, always block up the arc doorway once the pig is out to avoid the animal returning. Catching a pig by the hind leg or the forelegs is not recommended. The hips can dislocate because of lighter musculature. Careless handling and manipulations may also cause back and limb injuries. Pig boards, snares, and chemical sedation can all be used to good effect, although the response to the snare is often unpredictable with exotic pigs. They sometimes move forwards rather than back away, which may not provide the restraint anticipated. Grabbing the pig around the chest immediately behind the forelegs is recommended in pigs less than 50 kg. Pigs of this size can also be restrained by lifting them upright on to their hind legs by elevating the forelegs with the pig positioned between the operator's legs. Snares are useful in larger, more aggressive animals that have tusks. If one is brought to the surgery, then a small pig may be placed on the examination table in the consulting room to improve accessibility or on to a padded rug on the floor.

Pigs that are difficult to handle can be restrained using a Panepinto sling. This is like a stretcher with five holes in it – four for the legs and one at the base of the neck for blood sampling. Once the pig has the legs through the holes, the stretcher can be raised and suspended from a frame. The pig is suspended in mid air with its four legs protruding clear of the floor. Once in this position, the pig is unable to move. The hole over the base of the neck is used to facilitate blood sampling.

Physical examination

This has been covered in detail in Chapter 1 (*Investigation of clinical problems on pig farms*). The following is a brief checklist.

- Weight
- Condition score: palpation of ribs and spine (fat or thin)
- Temperature, heart rate, rhythm, respiratory rate, character
- Mucous membranes: colour
- Eyes and eyelids (entropion?)
- Oral and vulval mucosa (anaemia or jaundice)

- Ear (dark brown wax: swab *Sarcoptes*?)
- Skin (lesions: location and type)
- Palpation and manipulation of joints (arthritis?)
- Check for hernias (umbilical and inguinal)
- Feet (overgrown or misshapen)
- Auscultation: heart and lungs

Normal values for pot-bellied pigs

Table 13.1 shows the normal physiological values for pet pigs.

Sampling and further investigations

Skin scrapings and samples of blood, faeces, urine, vaginal and tonsillar swabs, and earwax can be collected. Often a sow will urinate after she has stood up, enabling a urine sample to be collected. Alternatively, the urethra can be catheterized using sedation, a speculum, and a dog catheter. Ultrasonography and radiography are useful, but sedation will usually be required in the adult. Blood samples for haematology and serum biochemistry can be taken.

Common diseases

A survey of pot-bellied pig owners in the UK on the range of conditions and procedures performed on 102 pot-bellied pigs was reported (Carr 2004). About a quarter of the pigs were pigs kept alone. Locomotory disorders accounted for 33% of the problems reported, with overgrown feet, lameness, and deformed feet being the most common. Respiratory disease, skin disease, and intestinal disease accounted for 10%, 33%, and 6% of the other disorders, respectively. Eight female pigs were reported to have behavioural problems related to the oestrous cycle. Five pigs had eye problems related to excessive loose skin around the eyes. Eight male pigs had been castrated, and two female pigs had had an ovariohysterectomy.

Table 13.1 Normal physiological values for pot-bellied pigs

Variable	Normal value
Rectal temperature	38.5–40.0°C
Resting heart rate	
Newborn	200 beats/min
Adult	70–80 beats/min
Respiratory rate	
Young	20–50 breaths/min
Adult	13–20 breaths/min
Puberty	2–4 months
Oestrous cycle	19–22 days
Standing heat	
Gilt	12–18 h
Sow	24–48 h
Gestation	109–114 days
Litter size	1–12 piglets

The same report indicated the conditions seen in 26 pigs by a veterinary consultant. Of the 26 pigs, 10 presented with locomotor problems (three with overgrown feet, seven with lameness). Six presented with skin problems. Individual cases of pneumonia, ileitis, behavioural aggression related to oestrus, hepatitis, cystitis, chronic mastitis, middle ear infection, and obesity were reported. The consultant also performed procedures on 29 pigs. Among these were 15 castrations (six with a unilateral scrotal hernia) and four ovariohysterectomies.

Obesity is common in pet pigs and contributes to cardiac (heart failure), respiratory (breathing difficulties), and lameness (osteoarthritis) problems. Owner education with regard to appropriate diets and exercise is essential. In severe obesity, a long-term diet plan should be implemented, with gradual weight loss over several months.

The louse (*Haematopinus suis*) is a large light brown parasite that is visible with the naked eye usually on the body of the pig. Eggs can be found on the hairs. Affected pigs are pruritic. Pigs with mange (*Sarcoptes scabiei* var. *suis*) are usually very pruritic with constant rubbing. There is thickened skin and loss of hair, often from the head, neck, and dorsum of the back. The mite may be identified by deep skin scrapings, although it may be difficult to find. Wax samples from the ears can be obtained by using swabs. They are easier to collect and the mites more numerous. The wax is mixed with potassium hydroxide and examined under the microscope. Ivermectin treatment of all the pigs on the property on two occasions 14–21 days apart will normally eliminate the problem. This drug is available in oral form, which is easy to administer. The drug is also effective against nematodes. Removal of bedding and thorough cleaning of shelters may also assist in the elimination of the parasite.

Sunburn and hyperthermia are relatively common conditions in the summer in pet pigs. Wallows and shades must be provided to prevent these conditions.

Skin fold pyoderma and dermatitis are common, especially in obese animals. Parenteral and topical antimicrobials in addition to cleaning the affected area with antiseptic solution usually results in resolution. Regular cleaning with antiseptic solution of at-risk areas and dieting will help to prevent recurrence.

Escherichia coli diarrhoea and colisepticaemia are encountered in colostrum-deprived piglets in association with dirty environments. Intraperitoneal injections of 5% dextrose can be given to severely dehydrated or hypoglycaemic piglets. Oral electrolytes can be given by stomach tube or bottle. Alternatively, piglets may lap from a flat dish. Antibiotics can be given by injection or orally (e.g. spectinomycin). Milk should not be withheld for more than 24 h because of the high metabolic demands for energy and protein.

Piglets that are reared indoors may develop iron deficiency anaemia, which will become apparent from 3 weeks

onwards. Pale mucous membranes and a prominent apex beat are presenting signs. Parenteral proprietary intramuscular iron injections into the semitendinosus and semimembranosus (hamstring) muscles should be given at 3–4 days of age to prevent this condition. Access to soil will ensure oral supplementation.

Streptococcal infections arising from navel contamination or bite wounds have caused bacterial arthritis in piglets. Penicillin has been used with good results in suspected cases.

Erysipelas causing skin lesions, endocarditis, chronic arthritis, and sudden death has been recognized in pet pigs. Vaccination is highly recommended.

Atrophic rhinitis is rarely recognized in pet pigs, but there is a potential risk from animals returning from shows to introduce the disease into a multipig household. Vaccination of these animals may be a wise precaution.

Lameness or abnormal gait is a common presenting sign. Overgrown hooves, dyschondroplasia, and degenerative joint disease have been most commonly diagnosed in pet pigs. Regular hoof trimming, avoidance of obesity, and use of appropriate commercial diets are recommended.

Abscessation can occur in many locations. Drainage, flushing, and antimicrobial therapy are the usual treatments.

Entropion is a common condition and is sometimes more severe in obese animals. There may be corneal ulceration with secondary infection. Surgical correction by skin flap resection is the treatment of choice.

Gastric ulceration has been reported in pet pigs. Predisposing factors may include insufficient fibre in the diet, meal that is too finely ground, and stress. The presenting signs depend on the severity. Sometimes, a perforated ulcer may lead to generalized peritonitis and death. Ulcers can also haemorrhage, with severe loss of blood and melaena. Less severe cases result in abdominal pain over the stomach and inappetance. Pyelonephritis must be ruled out as a differential diagnosis. Pyelonephritis can be recognized by localization of the pain to the renal area, demonstration of a high blood urea nitrogen, and the presence of pus or blood in the urine. Ultrasonography and peritoneal fluid analysis may be helpful in the investigation. Antimicrobials, non-steroidal anti-inflammatory drugs (NSAIDs), and ranitidine or cimetidine by mouth have been used to treat gastric ulcers. The potential predisposing factors should be identified and eliminated.

Miniature breeds of pig are prone to lower back and hind leg problems if handled or lifted incorrectly by the owner. Rest and NSAIDs may be indicated. More severe injuries may require investigation using radiography.

In addition to the conditions highlighted, pet pigs can be affected by all the conditions described for commercial pigs too.

Therapeutics

Antimicrobial therapy is discussed in Chapter 14 (*Antimicrobial therapeutics*). Analgesia is described in Chapter 15 (*Analgesia, anaesthesia, and surgical procedures in the pig*). Useful anthelmintic and antiparasiticide agents include ivermectin and doramectin.

Although unlicensed in the pig in the UK, natamycin and enilconazole are effective topical antifungal agents for ringworm. Gonadotrophins may be used in anoestrous sows and gilts to induce oestrus and to treat cystic ovaries. Azaperone can be used to prevent aggression when mixing pigs and to prevent savaging of a litter by agitated sows.

Routine procedures

Vaccination

Vaccines are sold in multidose packs, and it is more economical if several pigs can be vaccinated around the same time to make vaccination more affordable. If the pigs are to visit the surgery, direct contact between pigs from different households should be avoided by scheduling the pigs at different times. Alternatively, good communication with a specialist pig practice may enable single doses to be obtained on request from multipacks.

Erysipelas vaccination is recommended for all pet pigs. For breeding animals, *E. coli* and parvovirus vaccination are recommended. For all outdoor pigs, multivalent clostridial vaccination that includes *Clostridium perfringens* (types B, C, and D), *C. novyi* (types B and C), *C. septicum*, and *C. tetani* should be used. Atrophic rhinitis vaccination and *Mycoplasma hyopneumoniae* vaccination may be a wise precaution if the pig is to attend agricultural shows or may come into contact with other potentially infected pigs. Other vaccines should be considered dependent on individual circumstances and risk assessment.

Routine endoparasite and ectoparasite control

Ascaris suum, *Hyostrongylus rubidus*, *Oesophagostomum dentatum*, and *Metastrongylus apri* (lungworm) can be prevented by treating twice a year with ivermectin either by injection or by using in-feed medication. For control of lice and sarcoptic mange, a second dose 14 days after the first is required to eradicate all the life stages of the parasite.

Routine foot care and tusk trimming

It is advisable that the feet be checked regularly and trimmed if overgrown. Boar tusks pose a considerable risk to people and pets who come into close contact with a pet boar. The tusks should therefore be trimmed under seda-

tion or anaesthesia using embryotomy wire or a hacksaw. The teeth should be cut close to the gum margin and a file used if the sharp edges may cause problems. Annual trimming is recommended.

Chemical restraint and anaesthesia

Food should be withheld for 24 h and water for 4 h if general anaesthesia is to be used. Hypothermia can complicate recovery, particularly following prolonged procedures. Heating pads, blankets, and hot water bottles can be used to good effect in preventing or treating this problem. Intravenous anaesthesia can be used for short procedures. Gaseous anaesthesia following induction is advisable for more prolonged procedures. Anaesthesia and analgesia are described in detail in Chapter 15.

Intubation and venepuncture are two major challenges when dealing with pet pigs. In pot-bellied pigs, the ear vein may or may not be appreciated as it runs dorsally along the anterior margin of the ear. A rubber band placed at the base of the ear will raise the vein and increase its visibility. The vein can be catheterized using a 21- to 22-gauge butterfly needle if required. Alternatively, it is possible to cut down over the cephalic vein in the sedated pig.

Intubation is difficult and requires the use of a laryngoscope to depress the tongue and epiglottis. Placement of the intubation tube is sometimes assisted by the use of a plastic stylet. The stylet is placed inside the tube to direct the tube through the larynx and then removed. If the pig is quietly restrained, isoflurane using a tightly fitting mask around the pig's snout can be used for short procedures requiring anaesthesia. For longer procedures, the animals should be intubated. The sensitivity of pot-bellied pigs to halothane-induced malignant hyperthermia is not known.

For intramuscular injections in growers and adult pigs, 18-gauge 3.8-cm ($^1/_2$ inch) needles are recommended to ensure that the injection is intramuscular and not into the thick subcuticular layer of fat. The sedative azaperone is the only sedative or anaesthetic drug licensed in pigs in the UK. Ketamine (4 mg/kg) and xylazine (4 mg/kg) have been used in combination intramuscularly as a short-acting anaesthetic. Further information is provided in Chapter 15.

Surgical procedures

These are described in Chapter 15 (*Analgesia, anaesthesia, and surgical procedures in the pig*).

Piglet castration is a commonly performed procedure in male pet pigs to minimize aggression and avoid the undesirable smell of the pheromone in the saliva present in mature boars. It can be performed as in commercial piglets. The inguinal region should be carefully checked for pre-existing inguinal hernias that may need concurrent repair. These are relatively common in miniature piglets. Ultrasonography may be helpful in hernia identification. There may be an increased risk of postoperative inguinal herniation, and a closed castration is advisable. In addition, some veterinarians place a deep mattress suture across the inguinal ring to reduce the risk of herniation.

Most breeds of pet pig are smaller than commercial breeds, which limits the assistance that may be given during a dystocia. Caesarean section may have to be performed. Ultrasonography may be useful in appraising the viability, number, and position of the fetuses. Radiography can also indicate the position and number of fetuses. General anaesthesia or sedation and local anaesthesia are used. A flank approach parallel to the mammary glands is the recommended approach to minimize postoperative piglet interference.

Other common surgical procedures include inguinal hernia repair, umbilical hernia repair, entropion skin resection, laceration repair, and ovariohysterectomy.

Blood sampling

See Chapter 2 (*Investigation of clinical problems on pig farms*).

In young pigs, the anterior vena cava site can be used with the pig in dorsal recumbency. In adult pigs, the ear vein may be used if it can be detected on the margin of the ear. A rubber band around the base of the ear is required in addition to good restraint. Local anaesthetic gel is useful over the proposed site of venepuncture. Alternatively, the jugular at the base of the neck, which cannot be visualized or palpated, may be used. If only a small quantity of blood is required for a blood smear, the infraorbital venous sinus in the medial canthus has been described, but restraint must be good. The coccygeal vein or tail vein has been used for small volumes. The cephalic vein is an alternative site, and lifting the forelegs of the pig is a good restraint position for this approach.

Euthanasia

This is described in Chapter 16 (*Sampling and post-mortem examination of the pig*). Intravenous barbiturate overdose with or without sedation into the lateral ear vein in the large pig, or into the anterior vena cava in the piglet or weaner, is the most acceptable method in pet pigs.

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14

Antimicrobial therapeutics

Introduction

The appearance in human pathogens of multiple resistance to antibiotics and the adverse effects of residues in meat for human consumption have focused attention on the need for appropriate selection and usage of antibiotics in production animal veterinary medicine (Fig. 14.1). The pig industry in the UK uses more antibiotics on an annual basis than are used in any other species. The current consumption by the UK pig industry is approximately 200 tonnes per annum, a high proportion of which is delivered as in-feed or in-water medication. The veterinarian has an important part to play in ensuring that the meat is as free as possible from antibiotic residues, resistant zoonotic bacteria, and resistant bacteria that may transfer resistance to human bacteria.

When the decision is made to use antimicrobial therapy, veterinarians should strive to optimize therapeutic efficiency and minimize resistance to antimicrobials to protect public and animal health. Antimicrobials should not be used to replace other methods of reducing the risk of disease, such as nutrition, housing, hygiene, management, and vaccination. The aims are to minimize antibiotic usage without compromising animal welfare, to maximize therapeutic efficiency, and to minimize the selection of resistant organisms. Antimicrobial therapy can be very effective in reducing the duration of infection, limiting further spread of the disease and lowering the severity of the disease.

Therapy may fail because:

- the owner or keeper of the animals did not comply with the instructions,
- the diagnosis was incorrect,
- the dose prescribed was insufficient,
- the dose was given for too short a period of time to be effective,
- the pathogen was not sensitive to the antibiotic,
- the antibiotic failed to reach therapeutic levels in the organ or site of infection,
- immunosuppression reduced the host response, or
- the antibiotic was not stored correctly.

Food residues may result as a consequence of:

- using incorrect dose rates,
- treating the wrong animals,
- inappropriate withdrawal times,
- inaccurate records of treatment,
- incorrect or no labelling of feed hoppers containing medicated feed,
- inadequate cleaning of the hoppers used for medicated feed, or
- contamination of the environment with antibiotic (e.g. by spillage).

A knowledge of the national regulations regarding which antimicrobials can be used in pigs and the data sheet information regarding the specific conditions the product is licensed to treat, the dosage, the contraindications, the route of administration, and the withdrawal times are essential. Appropriate storage in terms of legal and temperature requirements should be implemented.

Factors in the selection of an appropriate antibiotic will include an accurate diagnosis, bacteriological culture results and in vitro antibiotic sensitivity results, previous responses on the farm to specified antimicrobials, and which licensed antibiotics are available for the specific disease within that species. If a licensed product is not available for that species, it may be possible, subject to national regulations, to select a product licensed in another farm animal species. In this case, evidence for efficiency in well-performed field trials should be sought.

The affected animals or animals at high risk may be selected for treatment.

Antimicrobials

Sensitivities

Antibiotics should be used only when it is known or suspected that an infectious agent is present that will respond to antibiotic therapy. Ideally, the sensitivity of the causal organism should be ascertained before therapy is started. In cases where the disease is severe and/or the spread is rapid, treatment should be started with an antibiotic with predicted sensitivity to the clinical diagnosis. Samples should be taken and sensitivity in vitro of isolates from outbreak of disease established in case of incorrect diagnosis or poor response to the chosen antibiotics. The response to antibiotic therapy should be monitored. In vivo factors affecting response include the ability to reach the site and attain high enough concentrations,

Fig 14.1 – Appropriate selection and usage of antimicrobials is an important function of the veterinarian.

persistence at the site of infection, the nature of the pathological process, and the immune response of the host. It is useful to know the pharmokinetics and tissue distribution of the drug. Pharmokinetic parameters such as bioavailability, tissue distribution, half-life, and minimum inhibitory concentration (MIC) 50 and MIC 90 can be used to compare different drugs. The route of administration should be considered. Prolonged oral use may raise concerns about the selection of resistant bacteria that inhabit the gut.

Antimicrobial group characteristics

Sulphonamides

- Good oral bioavailability (available as in-feed and in-water additives).
- Good distribution throughout the body, including central nervous system (CNS) and joints.
- Low concentrations in milk.
- Hepatic acetylation followed by renal excretion.
- Crystalluria may occur with overdose or water deprivation.
- Necrotic tissue and pus reduce efficacy.
- Bacteriostatic; combined with trimethoprim, bacteriocidal.
- Environmental contamination may occur because of excretion in faeces and urine.
- Susceptible: *Erysipelothrix rhusiopathiae*, *Streptococcus* spp., *Staphylococcus* spp., *Escherichia coli*, and *Haemophilus* spp.
- Highly resistant: *Mycoplasma* spp. and *Rickettsia*.

Penicillins

- Bacteriocidal.
- Penicillin G and phenoxymethylpenicillin have mainly Gram-positive activity.
- Penicillin G can be given only parenterally (it is degraded by gastric acid).
- Penicillin G and phenoxymethylpenicillin
  - Gram-positive: drug of choice for infections with *Erysipelothrix* spp., *Clostridia* spp., and *Streptococcus* spp.
  - Gram negative: *Pasteurella multocida* and *Haemophilus* spp.
- Phenoxymethylpenicillin and semisynthetic ampicillin, and amoxycillin can be given orally.
- Semisynthetic ampicillin and amoxycillin have a wide range of Gram-positive and Gram-negative activity, including against *Salmonella* spp. and *Escherichia coli*.
- Well distributed, with the exception of the CNS and joints.
- Low concentrations in milk.
- Hypersensitivity reactions may occur.
- Bacteriocidal, with noted anaerobic capability.
- Susceptible to staphylococcus penicillinases.

Tetracyclines

- Well absorbed following parenteral or oral administration.
- Bacteriostatic.
- Significant activity against Gram-positive and Gram-negative bacteria.
- Well distributed in tissues.
- Eliminated through bile and kidneys.
- Susceptible: *Erysipelothrix rhusiopathiae*, *Haemophilus* spp., *Pasteurella* spp., and *Streptococcus*.
- Variable susceptibility: *Staphylococcus* spp., *Escherichia coli*, *Salmonella* spp., *Clostridium* spp., and *Mycoplasma* spp.

Macrolides (tylosin, erythromycin, tilmicosin, and tulathromycin)

- Well absorbed orally.
- Erythromycin and tylosin are well distributed in tissues, particularly milk, with the exception of the CNS (variable distribution).
- Tilmicosin and tulathromycin are found in neutrophils and macrophages at sites of inflammation.
- They are bacteriostatic.
- Elimination is mainly by liver, some by kidney.
- Spectrum of activity: mainly Gram-positive, with some Gram-negative activity.
- Sensitive organisms include *Staphylococcus* spp., *Streptococcus* spp., *Leptospira* spp., *Haemophilus* spp., *Pasteurella* spp., and mycoplasmas.
- Tilmicosin and tulathromycin have demonstrated activity against *Actinobacillus pleuropneumoniae*, *Mycoplasma hyopneumoniae*, and *Pasteurella multocida*.

- Tulathromycin has prolonged activity following parenteral administration.
- Erythromycin has limited activity against mycoplasmas.

Aminoglycosides (spectinomycin and apramycin)

- Bacteriocidal.
- Excreted by kidneys.
- Poor CNS penetration.
- Mainly Gram-negative activity, including *Escherichia coli*, *Salmonella* spp., *Pasteurella* spp., and *Bordetella bronchiseptica*, but with some activity against *Staphylococcus aureus*, *Brachyspira hyodysenteriae*, and some mycoplasmas.

Cephalosporins (ceftiofur)

- Bacteriocidal.
- Given parenterally.
- Eliminated through kidneys.
- Activity
  - Gram-negative: *Actinobacillus pleuropneumoniae*, *Pasteurella multocida*, *Salmonella* spp., and *Escherichia coli*.
  - Gram-positive: including *Streptococcus suis*.

Lincosamides (lincomycin)

- Oral absorption good.
- Good tissue distribution (with the exception of the CNS).
- Eliminated mainly through liver.
- Bacteriostatic.
- Activity
  - Gram-positive: including *Staphylococcus* spp., *Streptococcus* spp., and *Erysipelothrix rhusiopathiae*.
  - Anaerobic bacteria: including *Brachyspira hyodysenteriae*, *Clostridium* spp., and *Fusobacterium* spp.
  - Mycoplasmas.

Diterpines (tiamulin)

- Good oral absorption.
- Good tissue distribution.
- High concentration in milk.
- Mainly excreted through the liver.
- Extensively used for swine dysentery (*Brachyspira hyodysenteriae*), mycoplasma pneumonia, and leptospirosis.
- Activity also against *Actinobacillus pleuropneumoniae* and *Erysipelothrix rhusiopathiae*.
- Gram-negative activity is limited.

Table 14.1 indicates the characteristics of a selection of pig pathogens that are important when considering their antimicrobial sensitivities. Table 14.2 provides information about the likely sensitivities of the major antimicrobial groups relative to the characteristics listed in Table 14.1.

Other antibacterial drugs

Spectinomycin

- Bacteriocidal.
- Active against some Gram-negative and Gram-positive bacteria.
- Not active against *Brachyspira hyodysenteriae*.
- Anaerobic bacteria mainly resistant.

Trimethoprim

- Bacteriostatic.
- Active against aerobic Gram-negative and Gram-positive bacteria.

Table 14.1 Characteristics of some pig bacterial pathogens that are important when considering their antimicrobial sensitivities

Bacteria	Location	Gram stain	Oxygen affinity
Actinobacillus	Extracellular	Gram-negative	Facultative anaerobe
Bordetella	Extracellular	Gram-negative	Aerobic
Campylobacter	Extracellular	Gram-negative	Aerobic
Clostridia	Extracellular	Gram-positive	Anaerobic
Arcanobacter	Facultative intracellular	Gram-positive	Facultative anaerobe
Escherichia coli	Extracellular	Gram-negative	Facultative anaerobe
Erysipelothrix	Extracellular	Gram-positive	Facultative anaerobe
Fusobacterium	Extracellular	Gram-negative	Anaerobic
Haemophilus	Extracellular	Gram-negative	Facultative anaerobe
Klebsiella	Extracellular	Gram-negative	Facultative anaerobe
Leptospira	Extracellular	n/a	Aerobic
Mycobacterium	Facultative intracellular	Acid-fast	Aerobic
Mycoplasma	Attached to cell membrane	n/a	n/a
Pasteurella	Extracellular	Gram-negative	Facultative anaerobe
Salmonella	Facultative intracellular or extracellular	Gram-negative	Facultative anaerobe
Staphylococcus	Extracellular or facultative intracellular	Gram-positive	Facultative anaerobe
Streptococcus	Extracellular	Gram-positive	Facultative anaerobe

n/a, not applicable.

Table 14.2 General antimicrobial group sensitivities

Antibiotic	Action	Aerobic		Anaerobic		*Mycoplasma*	Penetration
		Gram-positive	Gram-negative	Gram-positive	Gram-negative		
Aminoglycosides	Cidal	+	+	–	–	(+)	Extracellular fluid; synovial, peritoneal, and pleural fluid
Spectinomycin	Cidal	+	+	–	–	–	
Lincosamides	Static	+	–	+	+	+	Lung, liver, spleen, reproductive tract, skin, and bone
Macrolides	Static	+	–	+	+	+	Lung, liver, spleen, reproductive tract, skin, and bone
Tetracyclines	Static	+	+	+	+	+	Lung, liver, spleen, kidney, urine, and milk
Quinolones	Cidal	+	+	+	+	+	Lung, liver, and kidneys
Sulphonamides	Static	+	+	–	–		–
Trimethoprim	–	+	+	–	–	–	–
Potentiated sulphonamides	Cidal	+	+	+	+	–	Pleural, peritoneal, synovial, and ocular fluids, and cerebrospinal fluid
Cephalosporins (third generation)	Cidal	+	+	+	+	–	Soft tissue; bone; synovial, pleural, and pericardial fluids; cerebrospinal fluid; urine; and bile
Penicillin	Cidal	+	–	+	+	–	Soft tissue, bone, bile, urine, and peritoneum
Synthetic (e.g. ampicillin)	Cidal	+	(Some)	+	+	–	
Potentiated with clavulanate	Cidal	+	+	+	(+)	–	

Table 14.3 Comparative antimicrobial resistance of some porcine bacterial pathogens

Antimicrobial and disc antimicrobial content	*Escherichia coli*	*Salmonella*[a]	*Pasteurella multocida*	*Actinobacillus pleuropneumoniae*	*Streptococcus suis*	*Arcanobacter pyogenes*
No. of isolates	365	309	161	54	16	20
Ampicillin 10 µg	43	61	3	4	0	0
Penicillin 10 IU	–	–	–	–	0	0
Tetracycline 10 µg	85	84	9	22	94	0
Trimethoprim-sulphonamide 25 µg	52	63	8	13	6	10
Neomycin 10 µg	11	7	–	–	–	–
Apramycin 15 µg	12	5	–	–	–	–
Enrofloxacin 5 µg	8	–	0	4	0	0
Ceftiofur 30 µg	–	–	–	0	0	0

(From Veterinary Laboratories Agency 2004 and [a]Veterinary Laboratories Agency 2003, quoted by Burch D 2005 Problems of antibiotic resistance in pigs in the UK. In Pract 27:37–43.)

Resistance

There is resistance on UK farms and elsewhere in many bacterial species but in particular with strains of *Escherichia coli*, *Salmonella* (mainly *Salmonella typhimurium*), *A. pleuropneumoniae*, and *Brachyspira hyodysenteriae*. Table 14.3 indicates the antimicrobial resistance of some porcine UK isolates. Further information regarding pharmokinetics and antimicrobial resistance can be found in Burch (2003) and Burch (2005).

Immunocompetence

Bacteriocidal antibiotics may be preferred in immunosuppressed animals (e.g. postweaning multisystemic wasting syndrome) or severely ill animals, as the successful use of bacteriostastic antibiotics relies on an active immune system to control infection.

Spectrum of activity

Ideally, the antibiotic will target only the causal pathogen and have minimal effect on other micro-organisms. Generally, broad-spectrum antibiotics lead to the development of resistance in non-target micro-organisms more rapidly than narrow-spectrum antibiotics do, because of increased selection pressure. Therefore where an appropriate narrow-spectrum antibiotic is available, this may be preferable. Antibiotics that have an important function

in human medicine with few alternatives should be used only after careful consideration and justification. Individual treatment would be preferable to mass mediation with these antibiotics.

Decision analysis

Data sheet instructions regarding species, disease indications, contraindications, dosage regimens, withdrawal periods, and storage conditions should be understood. The dosage regimen and duration of treatment should be carefully considered to avoid administration of subtherapeutic doses and suboptimal time exposure, leading to therapeutic failure and chronic disease. Group medication will be necessary in some cases to treat clinically affected animals within the group, reduce the spread, and prevent clinical disease developing in incubating or subclinical animals. Strategic medication to healthy animals should be justified by a risk assessment indicating that the animals are at high risk and that other methods of reducing risk have been implemented or considered. Questions you should ask when considering antimicrobial therapy are as follow.

- Does the diagnosis warrant antibiotic therapy?
- What pathogenic organism is involved?
- What is the in vitro antibiotic sensitivity of the organism?
- What organ(s) is involved?
- Is there likely to be secondary infection (e.g. respiratory infections)?
- Is the organism extracellular or intracellular?
- Will the antibiotic penetrate these tissues?
- What is the spectral range of the antibiotics?
- What are the antibiotic or antimicrobial sensitivities of the pathogen?
- What dose, frequency, and length of course will be required?
- Are there any side effects to consider?
- What route of administration is appropriate?
- Which animals should be treated? (Clinical cases only or at-risk animals as well?)
- What are the withdrawal times and residue implications?
- Is it the most cost-effective therapy?

Antimicrobials for specific diseases

Tables 14.4–14.7 provide information regarding the selection of particular antimicrobials for specific pig pathogens. Table 14.8 indicates the range of antimicrobials that are available in the UK and their method of administration.

Antimicrobial medication

The five basic questions when the decision has been taken to use antimicrobial therapy are as follow.

1. Which antibiotic to use?
2. What dose rate to use?
3. Which route to use?
4. How long to continue medication for?
5. What is the withdrawal period?

Routes for administration include topical, oral, intramuscular, and subcutaneous. Intravenous injections are rarely used. Intramuscular injections are usually given in the semitendinosus and semimembranosus muscles (hamstring muscles) in small piglets. In older pigs, the neck muscles are the site of choice to minimize carcass damage. Subcutaneous injections may be given behind the shoulder, below the ear, or – for small volumes of non-irritant substances – in the loose skin in the posterior lower flank.

The oral route may include individual oral dosing of piglets or mass medication by inclusion of antimicrobials in the feed or in the water supply. Sick pigs are more likely to continue drinking than eating, although they may do neither. Severely sick animals are best treated individually by injectable products. Mass medication is used to effect in preventing and controlling diseases in large groups of animals. However, inevitably some healthy animals will be treated unnecessarily.

In-feed medications are prescribed by gram of active ingredient per tonne of feed and require a medicated feedstuff prescription in the UK. This concentration is derived from the dose rate required and the predicted daily intake. This varies, with small pigs eating 4–5% of their body weight and adult sows eating 1–2%, therefore higher inclusion rates would be required for sows and boars. There can be a delay in the manufacture and delivery of medicated feed. A dedicated bin for medicated feed is important to avoid residues contaminating other feed. Automated feed lines may make targeted delivery more difficult. An alternative is to top dress the ordinary feed with a mediated feed supplement to administer the antibiotic. Top dressing is a suitable method with small groups of pigs and individuals, but with larger groups of pigs there is extreme variation in the intake.

Water medication requires that the daily dose required is drunk by the pigs each day. The amount of antibiotic required for a group can be added to the header water tank supplying the target group and the supply to the header tank switched off until it is drunk. Provided that the capacity of the header tank is less than the daily requirement of the pigs (a rough estimate is 100 L/tonne pig/24 h), the pigs will receive the dose required. Table 14.9 indicates the estimated daily intake of pigs at different weights. The solubility and palatability of the

Table 14.4 Pathogen antimicrobial therapy possibilities

Pathogen	Aminocyclitols	Aminoglycosides		Cephalosporins	Diterpines	Fluoroquinolone[b]	Lincosamides	Macrolides				Penicillins		Sulphonamides		Tetracyclines	
	Spectinomycin	Gentamicin	Neomycin	Ceftiofur	Tiamulin	Enrofloxacin	Lincomycin	Tilmicosin	Tulathromycin	Tylosin tartrate	Valnemulin	Ampicillin	Penicillin	Sulphonamides	Trimethoprim/ Sulfamethoxazole	Florfenicol	Tetracycline
Actinobaculum suis				■		■	■					■	■	■	■	■	■
Actinobacillus suis	■			■		■		■	■		■	■		■	■	■	■
Actinobacillus pleuropneumoniae	■			■		■	■	■	■		■	■	■	■	■	■	■
Arcanobacter pyogenes				■	■	■	■					■	■	■	■	■	■
Bordetella bronchiseptica		■				■			■						■	■	■
Borrelia spiralis				■			■					■	■	■	■	■	■
Brachyspira hyodysenteriae					■		■	■		■	■						
Brachyspira pilosicoli					■		■	■		■	■						
Brucella suis	■	■	■		■		■	■		■	■						
Clostridium difficile																■	
Clostridium perfringens				■								■	■				
Escherichia coli cystitis				■		■						■			■	■	■
Escherichia coli diarrhoea		■	■	■		■						■			■		
Escherichia coli bowel oedema F18 Ste2x		■	■	■		■						■			■		
Eperythrozoon suis														■	■	■	■
Erysipelothrix rhusiopathiae				■			■			■		■	■				
Haemophilus parasuis	■	■	■	■	■	■	■	■	■			■	■	■	■	■	■
Isospora suis[a]														■			
Lawsonia intracellularis	■				■		■	■		■	■					■	■
Leptospirosis		■	■													■	■
Mycoplasma hyopneumoniae					■	■	■	■	■	■	■					■	■
Mycoplasma hyosynoviae					■	■	■	■	■	■	■					■	■
Pasteurella multocida (toxigenic)	■			■	■	■		■	■	■	■	■	■	■	■	■	■
Pasteurellosis	■			■	■	■		■	■	■	■	■	■	■	■	■	■
Salmonellosis	■	■	■	■		■									■	■	■
Spirochaetal colitis					■	■	■	■		■	■						
Staphylococcus hyicus				■		■	■								■		■
Streptococcus abscess	■	■	■	■		■	■					■	■	■	■	■	■
Streptococcus arthritis	■	■	■	■		■	■					■	■	■	■	■	■
Streptococcus suis joint ill	■	■	■	■		■	■					■	■	■	■	■	■
Streptococcus suis meningitis	■	■	■	■		■	■					■	■	■	■	■	■
Toxoplasma gondii	■	■	■	■	■	■	■	■	■	■	■	■	■	■	■	■	■

Shaded, sensitivity recognized.
[a]For *Isospora suis*, use toltrazuril.
(Courtesy of Dr John Carr, Iowa State University, Ames.)

Table 14.5 Antimicrobial drug selection for enteric and respiratory diseases

System	Condition	Aetiological agent(s)	Comments	Suggested	Alternatives with proven activity
Enteric	Colibacillosis	*Escherichia coli*	Resistance common	Gentamicin Trimethoprim-sulphas Ceftiofur Enrofloxacin	Apramycin Neomycin
	Coccidiosis	*Isospora suis*	Early treatment more effective	Toltrazuril	Amprolium Decoquinate Sulphonamides
	Colitis	*Brachyspira pilosicoli*	–	Tiamulin Lincomycin Tylosin Carbadox	–
	Proliferative enteropathy	*Lawsonia intracellularis*	Early treatment more effective	Tylosin Tiamulin Lincomycin Tetracycline	Enrofloxin Carbadox
	Salmonellosis	*Salmonella typhimurium*	Oral antibiotics may prolong shedding and promote resistance	–	–
	Swine dysentery	*Brachyspira hyodysenteriae*	Resistance to tylosin and lincomycin common and increasing to dimetridazole	Tiamulin Carbadox	Lincomycin Tylosin Dimetridazole
	Clostridial enteritis	*Clostridium perfringens* type C	Treatment of piglet clinical cases unrewarding; treat sows to reduce shedding	Bacitracin	–
Respiratory	Pasteurellosis	*Pasteurella multocida*	Marked resistance to tylosin and spectinomycin	Trimethoprim-sulphas Tetracycline Tulathromycin	Ceftiofur Enrofloxacin
	Atrophic rhinitis	*Bordetella bronchiseptica*, *Pasteurella multocida*	–	Tetracycline Trimethoprim-sulphas	Enrofloxacin
	Pleuropneumonia	*Actinobacillus pleuropneumoniae*	Resistance to tetracyclines and tylosin; sick pigs have reduced food and water intake	Procaine penicillin Ceftiofur Ampicillin Tulathromycin	Tiamulin
	Enzootic pneumonia	*Mycoplasma hyopneumoniae*	Often complicated by secondary bacterial pneumonia; tylosin and tiamulin are mycoplasmastatic	Tetracyclines Lincomycin Tylosin Tiamulin Enrofloxacin Tulathromycin	–

(After Friendship RM 2000 In: Prescott JF, Baggot JD, Walker RD [eds] Antimicrobial drug usage in swine in antimicrobial therapy in veterinary medicine. Iowa State Press, Ames, pp 602–610.)

Table 14.6 Antimicrobial drug selection for polysystemic, neurological, and musculoskeletal conditions

System	Condition	Aetiological agent(s)	Comments	Suggested	Alternatives with proven activity
Polysystemic	Erysipelas	*Erysipelas rhusiopathiae*	Insufficient length of treatment can result in chronic form	Procaine penicillin G Tylosin	Lincomycin Ceftiofur Trimethoprim-sulphas
	Glasser's disease	*Haemophilus parasuis*	Early treatment important	Procaine penicillin G Tetracyclines	Trimethoprim-sulphas
	Mycoplasma serositis	*Mycoplasma hyorhinis*	Poor response to treatment	Tylosin Lincomycin Tetracycline	–
	Salmonellosis	*Salmonella cholerae-suis*	Sensitivity testing important; treat clinical systemic parenterally	Ceftiofur Enrofloxacin Ampicillin Trimethoprim-sulphas	Apramycin Carbadox
	Actinobacillus suis	*Actinobacillus suis*	–	Ampicillin Trimethoprim-sulphas Ceftiofur Enrofloxacin	–
Neurological	Streptococcal meningitis	*Streptococcus suis*	–	Procaine penicillin G Ampicillin	Trimethoprim-sulphas Ceftiofur
	Oedema disease	*Escherichia coli*	Prognosis poor	Apramycin	Carbadox
	Otitis media (middle ear disease)	*Staphylococcus* *Streptococcus* *Arcanobacter pyogenes*	Response variable	Trimethoprim-sulphas Ampicillin	–
	Tetanus	*Clostridium tetani*	Prognosis poor	Procaine penicillin G	–
	Listeriosis	*Listeria monocytogenes*	–	Procaine penicillin G Trimethoprim-sulphas Tetracycline	–
Musculoskeletal	Neonatal polyarthritis	*Streptococcus* *Staphylococcus* *Escherichia coli* *Arcanobacter pyogenes*	Severe cases unresponsive	Procaine penicillin G Lincomycin Tylosin	–
	Suppurative arthritis	*Streptococcus* *Staphylococcus* *Arcanobacter pyogenes*	Severe cases unresponsive	Procaine penicillin G Lincomycin Tylosin	–
	Mycoplasma arthritis	*Mycoplasma hyosynoviae*	–	Lincomycin Tylosin Tiamulin	–
	Foot rot	*Fusobacterium necrophorum*	–	Procaine penicillin G	Tetracyclines Sulphonamides

(After Friendship RM 2000 In: Prescott JF, Baggot JD, Walker RD [eds] Antimicrobial drug usage in swine in antimicrobial therapy in veterinary medicine. Iowa State Press, Ames, pp 602–610.)

Table 14.7 Antimicrobial drug selection for urinary, cardiovascular, and skin conditions

System	Condition	Aetiological agent	Comments	Suggested	Alternatives with proven activity
Urinary	Pyelonephritis and cystitis	*Actinobaculum suis*	–	Procaine penicillin G Ampicillin	Ceftiofur Tetracyclines
	Leptospirosis	*Leptospira* spp.	Carrier state not eliminated with antimicrobials	Tetracyclines Streptomycin	–
	Brucellosis	*Brucella suis*	National control policies apply	Tetracyclines Streptomycin Trimethoprim-sulphas	–
Cardiovascular	Eperythrozoonosis	*Eperythrozoon suis*	Control lice to decrease spread	Tetracyclines	–
Skin	Exudative epidermitis (greasy pig disease)	*Staphylococcus hyicus*	–	Procaine penicillin G Lincomycin	Ceftiofur Enrofloxacin

(After Friendship RM 2000 In: Prescott JF, Baggot JD, Walker RD [eds] Antimicrobial drug usage in swine in antimicrobial therapy in veterinary medicine. Iowa State Press, Ames, pp 602–610.)

Table 14.8 UK drugs licensed for use in pigs

Antimicrobial group	Drug	Injection	Water	Feed	Oral doser	Topical spray
Aminocyclitols	Lincospectinomycin		X	X		
	Spectinomycin				X	
Aminoglycosides	Apramycin		X	X		
	Procaine penicillin/dihydrostreptomycin	X				
	Neomycin	X	X	X		
Cephalosporins	Cefquinome	X				
	Ceftiofur	X				
Diterpines	Tiamulin	X	X	X		
Florfenicols	Florfenicol	X				
Lincosamides	Lincomycin	X	X	X		
Macrolides	Tylosin	X	X	X		
	Tulathromycin	X				
	Valnemulin			X		
	Tilmicosin			X		
Penicillins	Ampicillin	X				
	Amoxycillin	X	X	X		
	Clavulanic/amoxycillin	X				
	Procaine penicillin	X				
	Phenoxymethyl penicillin			X		
	Procaine penicillin/dihydrostreptomycin	X				
Polymixins	Colistin		X			
Quinolones	Danofloxacin	X				
	Enrofloxacin	X			X	
	Marbofloxacin	X				
Sulphonamides	Trimethoprim/sulphonamide	X	X	X		
Tetracyclines	Tetracycline	X	X	X		X

(From National Office of Animal Health 2006 National Office of Animal Health [NOAH] data sheets compendium. Online. Available: http://www.noah.co.uk/.)

Table 14.9 Estimated daily intake of water by growing pigs

Average weight of pig (kg)	Water intake (L)
8	1.0
10	1.2
15	1.7
20	2.2
30	3.0
40	4.0
50	5.0
60	6.0
70	7.0
80	8.0
90	9.0

antibiotic powder may mean that a greater dilution is required. Provided that the quantity of water to achieve this does not exceed the daily intake, the correct quantity of the antibiotic could be divided across two header tanks of water. It is important to stir the water in the header tank vigorously following the addition of the antimicrobial to ensure that it forms a solution. It is also important to monitor the header tank to ensure that the water supply is turned on again once the tank is empty. Salt poisoning has occurred in the past where the water supply to the header tank has been left switched off for prolonged periods following medication.

The arrangement of the water supply may make targeting a specific group difficult, and alternative arrangements regarding the water supply to this group may be needed for the duration of the treatment. A wastage of approximately 20% of the water can be anticipated due to spillage with nipple drinkers. Temporary dedicated medicated water tanks are available that can be plumbed into the target pens when required. This may be the best option in some cases. Proportioners, which add a measured amount of drug into the water line, are also available.

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National Office of Animal Health 2006 National Office of Animal Health (NOAH) data sheets compendium. Online. Available: http://www.noah.co.uk/

15

Analgesia, anaesthesia, and surgical procedures in the pig

Introduction

The cost and welfare of the animal should be carefully considered before surgery. Surgery on individual commercial pigs is frequently not cost-effective, and euthanasia is the preferred option. Boars and sows of high genetic merit may be the exceptions. Pet pigs often have a greater value to the owner, and more expensive complex procedures are possible (Fig. 15.1).

Owner communication

The potential risks and prognosis should be clearly explained to the owner. All the options should be made explicit. Written consent should be obtained. The costs should be discussed and put in writing.

Preanaesthetic evaluation

An appropriate assessment of the patient should be made. This should include a physical examination. Haematology and clinical pathology may be indicated in high-risk or valuable animals.

Legislation

The current UK legislation regarding on-farm routine surgical procedures in pigs is given in Table 15.1.

Premedication

Atropine sulphate at a dose rate of 0.02–0.04 mg/kg i.m., s.c., or i.v. 30 min before general anaesthesia will reduce the risk of bradycardia and reduce salivation. Ophthalmic ointment can be instilled into both eyes immediately following induction of anaesthesia to provide corneal protection, particularly in pet pigs.

Analgesia

Pain management is important in pigs undergoing surgical intervention. Preoperative, intraoperative, and postoperative pain management should be considered in relation to the intensity of pain the animal is likely to suffer. Non-steroidal anti-inflammatory drugs (NSAIDs) can be administered for mild pain, α_2 agonists or opioids for moderate and severe pain (Table 15.2). Opioids can be used intraoperatively and postoperatively to treat acute pain.

Anaesthesia

Mask induction using isoflurane followed by intubation is a commonly used option in smaller pigs, particularly pet pigs that are brought to the surgery. For small and large pigs of all sizes, on-farm intramuscular injections are preferable for sedation or anaesthesia. Intramuscular injections are most effectively given into the neck muscles. Sedation and general anaesthesia can be used in combination with local anaesthesia at the surgical site to reduce the doses required for effective analgesia.

Preparations, precautions, and aftercare

Food should be withheld for 24 h and water for 4 h if general anaesthesia is to be used. Thermoregulation in the pig under sedation or general anaesthesia is relatively poor, and the animal should be kept in a comfortable ambient temperature to prevent hypothermia or hyperthermia both during and after the procedure. Heating pads, blankets, and hot water bottles can be used to good effect in preventing or treating this problem.

The sensitivity of pot-bellied pigs to halothane-induced malignant hyperthermia is not known. Halothane and other stresses can trigger malignant hyperthermia in genetically predisposed pigs. The clinical signs include a sudden increase in body temperature with muscle rigidity, tachycardia, and tachypnoea. Gaseous anaesthetics should be discontinued and the pig cooled by the use of ice packs.

The pig, particularly the pet pig, has a higher proportion of body fat relative to muscle and may have prolonged recovery periods when barbiturates are used.

Fluid therapy should be used where appropriate, using the ear vein.

During the anaesthetic recovery phase, pigs should be confined to a pen with a non-slippery surface to prevent uncoordinated, abnormal movements, which can result in torn muscles. The most common of these is lateral abduction of both hind legs with both legs being splayed outwards into the 'splits' position, with tearing of the adductor muscles. Confinement following surgical procedures with

healing wounds for 10–14 days is advisable to avoid wound breakdown. Reintroduction of a pig to a group must be done with extreme care and close supervision to avoid fighting. The use of azaperone sedation may be advisable.

Intubation and venepuncture

Intubation and venepuncture are two major challenges when dealing with pigs.

In pot-bellied pigs, the ear vein may or may not be detected as it runs dorsally along the anterior margin of the ear. A rubber band placed at the base of the ear will raise the vein and increase its visibility. The vein can be catheterized using a 21- to 22-gauge butterfly needle if required. Pigs flick their ears when the ear is pricked with a sharp needle. The ear needs to be held securely to minimize this problem. The use of topical local anaesthetic gel on the skin over the vein to be punctured is helpful in this regard. Alternatively, it is possible to cut down over the cephalic vein in the sedated pig.

Intubation is difficult and requires the use of a laryngoscope to depress the tongue and epiglottis. Placement of the intubation tube is sometimes assisted by the use of a plastic stylet. The stylet is placed inside the tube to direct the tube through the larynx and is then removed. If the pig is quietly restrained, administration of isoflurane using a tightly fitting mask around the pig's snout can be used to anaesthetize the pig for short procedures. For longer procedures, the animal should be intubated.

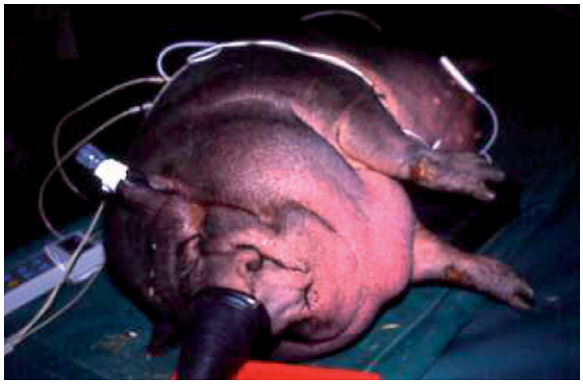

Fig 15.1 – General anaesthesia in a pot-bellied pig to repair severe bilateral entropion.

Table 15.2 Analgesic drugs used to relieve pain in pigs

Drug	Dose rate (mg/kg)	Route	Frequency of administration (h)
Aspirin	10.0	p.o.	4
Flunixin[a]	1.0	i.m.	24
Ketoprofen[a]	3.0	i.m.	Once
Butorphanol	0.1	i.m.	Once or as required
Buprenorphine	0.005–0.01	i.m.	12
Morphine	0.2–0.9	i.m.	Once or as required
Xylazine	1.0	i.m.	12
Paracetamol[a]	30	In feed	24
Meloxicam[a]	0.4	i.m.	Once
Tolfenamic acid[a]	2.0	i.m.	Once

[a]Licensed products available in the UK.

Table 15.1 Current UK legislation regarding routine surgical procedures in pigs

Operation	Age of animal	Conditions	Veterinary surgeon must perform procedure?	Anaesthetic required by law?
Castration	First week of life	–	No	No
	> 7 days	–	Yes	No
	> 4 weeks	–	Yes	Yes
Tooth clipping	First week of life only	When there is evidence that injuries to the sow's teats or other piglets have occurred or are likely to occur as a result of not carrying out this procedure. A remedial action plan is required.	No	No
Docking of tails	First week of life	Routine tail docking is not permitted. If tail biting is a problem, tail docking should only be used as a last resort when other environmental improvements have proved ineffectual.	No	No
	> 7 days old	When in the opinion of the veterinary surgeon the operation is necessary for reasons of health or to prevent injury from the vice of tail biting	Yes	Yes
Vasectomy	Any age	–	Yes	No – anaesthetic requirement decided by veterinary surgeon

Table 15.3 Some drug regimens that have been recommended for sedating or anaesthetizing pigs

Use	Drug	Dose (mg/kg)	Route	Onset (min)	Duration (min)
Sedation	Acepromazine	0.2–0.5	i.m.	20–30	30–60
	Azaperone				
	Boar	1	i.m.	5–15	60–120
	Others	2	i.m.	5–15	60–120
	Combined[a]				
	Medetomidine	0.08	i.m.	5–8	15–30
	Ketamine	10	i.m.		
Anaesthesia					
Induction only	Combined[a]				
	Xylazine	0.5	i.m.	5–10	5–15
	Ketamine	5	i.m.		
Short procedures	Thiopental (thiopentone)	10–20	i.v.	Immediate	2–10
	Pentobarbital (pentobarbitone)[b]	10–30	i.v.	Immediate	15–45
	Combined				
	Diazepam	1–2	i.m.	10	20–40
	Ketamine	10–15	i.m.		
	Combined				
	Midazolam	0.1–0.5	i.m.	5–10	20–40
	Ketamine	10–15	i.m.		
	Combined[a]				
	Xylazine	4	i.m.	5–10	15–30
	Ketamine	4	i.m.		
Longer procedures	Combined[a,c]				
	Medetomidine	0.08	i.m.	1–5	60–120
	Xylazine	0.2	i.m.		
	Ketamine[c]	2	i.m.		
	Combined[a]				
	Azaperone	2	i.m.	5–8	40–60
	Xylazine	0.2	i.m.		
	Ketamine	2	i.m.		

[a]Combinations can be mixed in a syringe and given as a single injection.
[b]Should be used with caution in young pigs because of their reduced hepatic function.
[c]Emergency reversal of this combination is by i.m. injection of atipamezole at a dose rate of 50 mg/kg.

Anaesthesia and sedation by intramuscular injection

For intramuscular injections in growers and adult pigs, 18-gauge, 3.8-cm ($1\frac{1}{2}$-inch) needles are recommended to ensure that the injection is intramuscular and not into the thick subcuticular layer of fat. The sedative azaperone is the only sedative or anaesthetic drug licensed in pigs in the UK. Some drug regimens for pigs are given in Table 15.3.

Lumbosacral epidural

General anaesthesia or sedation with or without local anaesthetic is the preferred option in pet pigs. Lumbosacral epidural anaesthesia is the most commonly used regional anaesthesia in commercial pigs and provides regional anaesthesia of the abdomen and hindquarters depending on the dose administered. This enables umbilical, scrotal, abdominal, or rectal surgery to be performed. Physical or chemical restraint is needed to prevent movement caused by the forelimbs. Sedation may also be necessary to control the noise caused by the pig.

The lumbosacral epidural injection site is just caudal to an imaginary line drawn between the two tuber coxae indicated by a depression in the spinous processes. A 6- to 8-cm 20-g lumbospinal needle is used in pigs up to 30 kg in weight, a 10-cm 18-g needle in pigs of 30–90 kg, and a 12- to 16-cm 18-g needle in pigs over 90 kg. The injection is usually given while the animal is standing restrained in a chute and with a snare in place. The needle is inserted at a slight angle caudal to the perpendicular, and advanced between the last lumbar and the first sacral vertebrae. The needle penetrates the skin, back fat, muscle, and then the interspinous ligament, where there is a slight increase in resistance before a pop is felt as the end of the needle enters the lumbosacral space. The stylet is removed and an empty syringe attached. Negative pressure is applied to the syringe barrel by attempting to pull back on the plunger. If blood or clear fluid is withdrawn, the needle

should be repositioned. On removing the empty syringe, a small drop of 2% lignocaine (lidocaine) can be placed on the needle barrel. If the needle is correctly positioned, the fluid will rapidly disappear. The dose of lignocaine should then be injected slowly and the needle removed. The dose rate is 1 mL/9 kg of body weight of 2% lignocaine. Analgesia should be present within 10 min and lasts up to 120 min. The pig should not be placed on a slope with the herd pointing downwards, otherwise anaesthesia may progress too far anteriorly and compromise respiration. Xylazine has been given by epidural injection at a dose rate of 2 mg/kg in 5 mL of saline to provide immobilization and analgesia for 120 min. It has also been used in combination with 10 mL of 2% lignocaine at a dose rate of 1 mg/kg to provide anaesthesia and analgesia of the hindquarters.

Once the surgical procedure is complete, the hind legs should be cast using a figure of eight rope around the hocks to prevent uncoordinated hind leg movements before complete sensation returns. Uncoordinated movement can result in muscle or tendon ruptures. The floor should also be non-slip to facilitate standing.

Intravenous regional anaesthesia of the lower limb

A tourniquet is placed above the hock in the hind limb or above the carpus in the forelimb. Identification of a superficial vein distal to the tourniquet may be difficult, and palpation may be necessary. The lateral saphenous in the hind leg is usually the most prominent and easiest to locate. Using a 19-g needle, 10 mL of 2% lignocaine without adrenaline (epinephrine) is injected into the vein. Ten minutes should be allowed for diffusion of the local anaesthetic to desensitize the lower limb. Desensitization should be tested by deep pinching of the lower limb with forceps. More time should be allowed if there is a response. There will be no increase in effect after 15 min has elapsed from the time of injection. If the procedure is abandoned soon after injection, the tourniquet should be released gradually over several minutes to avoid an intravenous bolus of anaesthetic being circulated.

Surgical procedures

Amputation: digital

Digital amputation may be considered when severe infections of the foot such as osteomyelitis and septic joints are unresponsive to other forms of treatment. The problem must be confined to one digit. A high digital amputation through the distal end of phalanx 1 will remove the third and second phalanges and the proximal and distal interphalangeal joints.

This procedure is performed under general anaesthesia or sedation and intravenous regional anaesthesia. The skin of the limb is cleaned and prepared for surgery. A tourniquet is placed just proximal to the carpus in the forelimb and the tarsus in the pelvic limb to avoid haemorrhage during the procedure. A skin incision is made through the interdigital skin and continued to encircle the digit to be amputated in an elliptical arc as high as distal end of phalanx 1 laterally. A length of embryotomy (obstetrical) wire with a handle on each end is placed into the wound between the claws. The leg is held firmly by an assistant and the digit is amputated by sawing an angle laterally in the groove of the incised skin. The stump of phalanx 1 is sprayed with antibiotic and a non-stick bandage applied. A pressure bandage is applied to the limb. The tourniquet is then removed. Some clinicians prefer to saw through the proximal end of phalanx 2 then remove the stump of phalanx 2 and attempt to cover the stump with skin. Persistent synovial fluid production may increase the risk of postoperative infection using this method. The animal should be kept in a well-bedded pen for 3–4 weeks. The dressing can be removed in 10 days. A course of analgesia and antibiotics is recommended.

Amputation: uterus

This is a salvage procedure to preserve the sow until her piglets are weaned. The sow should be culled when the uterus is severely and extensively damaged or is impossible to replace. The aim is to surround the base of the uterus to be amputated with overlapping mattress sutures to facilitate haemostasis. The overlapping mattress sutures should penetrate the full thickness of the uterine wall and be tied tightly. Monofilament nylon is usually used. Before the distal portion is amputated, two anchor pins can be placed through the uterus at right angles just proximal to the suture line. The distal uterus is then amputated using a scalpel distal to the mattress suture line and the cut edge carefully checked for bleeding vessels. If found, these should be carefully ligated. Once haemostasis is complete, the anchor pins can be removed and the uterine stump replaced into the pelvic cavity. Some clinicians place a subcutaneous purse string suture round the vulva.

Atresia ani

In this condition, there is no patent anal orifice. Abdominal distension occurs in the first few days or weeks of life, which may be accompanied by vomiting. In the female, there may be a rectovaginal fistula with defecation occurring via the vulva. General anaesthesia provides an immobile operation site; alternatively, local infiltration with good physical restraint is required. A bulge in the skin below the tail, caused by the retained faeces, indicates the appropriate site to create the missing anus. A small

circular skin incision is made over this bulge. The passage of faeces indicates that the rectum is now patent. In some cases, where the atresia is less superficial, there may be no bulge in the perineal skin and no faeces forthcoming following an exploratory incision over the site of the anus. In these cases, euthanasia is recommended.

Aural haematoma

This condition of the ear is caused by head shaking, fighting, or biting. Trauma results in the rupture of a blood vessel, and the subcutaneous space between the skin and the cartilage of the external pinna fills with blood. The animal presents with a large fluctuant swelling of the ear. The swelling is almost invariably on the medial aspect of the ear. A needle aspirate of the swelling reveals serum or unclotted blood. The haematoma will become fibrosed, leaving an unsightly crumpled external pinna. In commercial pigs, this condition is rarely treated. Attention should be given to the underlying cause of the problem. In some cases, early drainage of the haematoma using a needle and syringe and using a snare for restraint may result in resolution. This may need to be repeated if the haematoma reforms. More usually, the skin over the haematoma is incised and the serum, fibrin, and clot are removed. The dead space is then obliterated by suturing the skin to the pinna cartilage using a series of mattress sutures of monofilament nylon. The sutures are placed either side of the incision wound. The wound is left open to facilitate drainage. A pressure bandage may be applied to the ear by using a roll of bandage placed on the medial aspect of the ear, wrapping a dressing around the ear, and securing the roll of bandage with Elastoplast. A course of antibiotics is recommended to avoid any secondary infection.

Bladder: retroversion

This is a rare condition. The bladder becomes displaced laterally and caudally to lie beside the vagina. The condition may occur in late gestation or during parturition. The condition may induce a vaginal prolapse. The sow may strain in an attempt to pass urine if the urethra is blocked by the abnormal position of the bladder. Vaginal and rectal examinations reveal a distended viscus lying beside or above the vagina. Catheterization of the bladder may be possible to relieve the straining and allow the sow to farrow. Once decompressed, vaginal or rectal manipulation may enable the bladder to be replaced in the normal position, but this is unusual. Once farrowed, euthanasia may be an appropriate economic option if the condition cannot be resolved.

Alternatively, under general anaesthesia the bladder can be repositioned through a caudal midline laparotomy. Cystopexy to the caudal abdominal floor should prevent recurrence. A course of antibiotics and analgesics is advisable.

Caesarean section

The sow with dystocia is likely to be exhausted and possible toxaemic. In a survey, sow mortality following caesarean section was reported to range from 10 to 30%. Some or all of the piglets were dead. Sow mortality and piglet mortality increased with the duration of time the sow had been in labour. Delay in performing a caesarean section will reduce the chances of survival of the sow and piglets.

General anaesthesia is recommended to ensure an immobile patient and to facilitate an uninterrupted procedure. Intravenous fluids (via the ear vein), betamethasone, and flunixin may all be useful to support the sow prior to surgery. Dextrose may be added to the intravenous fluid if required.

The supramammary ventrolateral approach is the surgical site of choice. This site avoids the mammary blood vessels and carries less risk of wound contamination and piglet interference when compared with a midline approach. The site also gives good access to both uterine horns. The operation is performed with the sow in lateral recumbency, usually on the floor. The upper limb obstructs the operational site. The leg must be elevated and moved backwards into extension before being secured in this position.

A 15- to 20-cm skin incision is made parallel and dorsal to the mammary gland, starting in the inguinal region and extending cranially just below the flank fold. The abdominal muscles and the peritoneum are incised using scissors and forceps. The nearest uterine horn to the incision is partially exteriorized and a 6- to 8-cm incision made in the long axis of the uterine horn. The site for this incision should be as close to the bifurcation of the uterine horns as possible to facilitate the removal of piglets from both horns. The piglets in the exteriorized horn should be squeezed out of the horn through the incision. Attempts should be made to extract the piglets from the other horn through the same uterine incision. If this is not possible, the uterine wound should be closed using inverting sutures of absorbable material and the horn replaced into the peritoneal cavity. The other horn should be manipulated up to the abdominal wound. An incision into the second horn should be made in the long axis of the horn to enable the remaining piglets to be removed. Always examine the uterine body and vagina caudally to ensure that no piglets are missed. Warm saline may be used to lavage the uterus to reduce the risk of postoperative adhesions. Four lines of sutures are then used to close the abdomen, these are:

- the peritoneum and the transversus abdominis muscle,
- the internal and external oblique muscles,
- the subcutis and fat, and
- the skin.

Intraoperative and postoperative antibiosis and analgesia are recommended. Oxytocin can be given once surgery is complete to facilitate uterine involution. The skin sutures should be removed in 14 days. The potential for future reproduction is only slightly reduced following surgery provided that there are no complications.

Castration: baby pigs

This is no longer done routinely in commercial herds in the UK. Research indicates that castrating piglets at 2 weeks of age minimizes the stress of castration and maximizes the performance to weaning. The use of local anaesthetic has been shown to reduce postoperative discomfort. The use of NSAID analgesics may also reduce the discomfort. Prophylactic treatment with tetanus antitoxin may be a wise precaution depending on the risk assessment.

The piglet is suspended by the hind legs. The surgical site is prepared aseptically. Local anaesthetic (2% lignocaine or 5% procaine hydrochloride with adrenaline [epinephrine]) is injected subcutaneously down the length of each testicle at the incision site and around each spermatic cord at the inguinal ring. At each of the four sites, approximately 0.5 mL of anaesthetic is used. An open castration is performed. An incision is made over each testicle using a scalpel blade that incises the skin and the tunica vaginalis. The incision should be long enough to enable the testicle to be exteriorized through the wound. The gubernaculum, which attaches the base of the testicle to the scrotal sac, is broken using a hooked finger. Each testicle is 'shelled out' of the tunica vaginalis and exteriorized. The spermatic cord is broken by traction, enabling the removal of the testicle. Haemorrhage is rarely if ever a problem in this age of piglet. Topical antibiotic spray is often used to reduce the risk of infection. Systemic antibiotics are rarely used. Replacing the piglet in the creep area under a heat lamp will be beneficial. Inguinal hernias and undescended testicles may be found at the time of surgery. These are discussed below.

Castration: pet pigs

Castration is a common procedure in male pet pigs to avoid unwanted mating, aggression, and unpleasant odours following puberty. Before starting the surgical procedure, it is important to check for inguinal hernias, which are relatively common in miniature breeds. It is advisable to castrate the pig between 2 weeks and 3 months of age. After this time, the animal may become more difficult to handle and restrain. Younger pigs require greater aftercare following surgery, as they are more prone to hypothermia and hypoglycaemia.

Deep sedation and local anaesthesia or general anaesthesia is most commonly used when castrating pet pigs. Postoperative inguinal herniation is an important consideration in miniature breeds of pig, and closed castration is recommended to avoid this problem. The surgical procedure is usually performed in dorsal recumbency. Following appropriate anaesthesia, a skin incision is made over the testicle and is extended over the inguinal ring. The integrity of the tunica vaginalis is maintained. The gubernaculum, which attaches the base of the testicle to the scrotal sac, is broken using a hooked finger. A ligature of absorbable suture material is placed around the spermatic cord, which is still ensheathed in the tunica vaginalis. The spermatic cord is then cut below the ligature and the testicle removed. The stump will then retract into the peritoneal cavity via the inguinal canal. It is advisable to place a horizontal mattress suture through the inguinal ring as an additional precautionary measure to prevent a postoperative inguinal hernia. Monofilament nylon has been used for this procedure. Subcutaneous tissue is then apposed using absorbable suture material, and the skin is closed with non-absorbable sutures. These are removed in 10–14 days. Antibiotics given both topically and systemically are advisable. Analgesia in the form of NSAIDs can be given. Prophylactic treatment with tetanus antitoxin may be a wise precaution depending on the risk assessment.

During the procedure, the pig's core temperature should be monitored for hypothermia or hyperthermia. The ambient temperature of the operating room should be comfortably warm. The animal should be kept warm until recovery is complete. Adequate shelter and bedding should be provided for the 24 h following the operation.

Castration: adult or larger pigs

Owners may wish to have older boars that are no longer used for breeding castrated. Pet pig owners may find that a male animal is too aggressive as it reaches maturity.

The testicles in mature boars are large, and haemostasis at surgery is an important consideration. Inguinal hernias can occur following castration, and so a closed technique that minimizes this risk may be preferable, particularly in pet pigs in which the risk is higher than in miniature pigs.

Large pigs are not easy to restrain and can in certain circumstances be aggressive. Excessive movement during the operation makes the procedure difficult and increases the risk of wound infection due to inadvertent contamination. Deep sedation with local anaesthesia or more preferably general anaesthesia is recommended.

If local anaesthesia is required, a subcutaneous line block over the surgical site is performed. Local infiltration around the spermatic cord and intratesticular injection of 5–10 mL of 2% lignocaine will ensure effective anaesthesia. Several minutes may be required following the injection of the local anaesthetic to achieve a satisfactory level of anaesthesia. With the boar in lateral recumbency, a

skin incision large enough to remove the testicle is made over the distal part of the testicle towards the ventral scrotum (this site will facilitate postoperative drainage). The gubernaculum attaching the testicle to the ventral scrotal sac is broken using the fingers. The testicle still within the tunica vaginalis should be exteriorized and the spermatic cord stripped of inguinal fat and adherent tissue. The testicle is then twisted to coil the spermatic cord into a tight rope-like structure. Two crushing forceps can then be placed across the spermatic cord. Two transfixion ligatures are placed proximal to the forceps using 4–5 metric absorbable suture material. The spermatic cord is then cut between the two pairs of forceps, and the testicle is removed. A pair of Allis tissue forceps are applied to the ligated spermatic cord and the crushing forceps removed to check for haemostasis. The ligated spermatic cord is then released. As an additional precaution, particularly in the pet pig, the inguinal ring can be closed using one or two monofilament nylon mattress sutures. The procedure is repeated to remove the remaining testicle.

In most commercial pigs, the scrotal skin incision is left open to facilitate drainage. Alternatively, particularly with pet pigs, the wound can be closed. Absorbable suture material can be used to close the subcutis and minimize the dead space. The skin is closed with monofilament nylon. Skin sutures should be removed in 14 days. Sedation or good restraint will be required.

A course of antibiotics for 3–5 days is recommended, and topical antibiotics may also be used. Analgesia in the form of NSAIDs will reduce postoperative discomfort. The pig should be kept confined in a small pen with clean straw for 7–10 days. Boar taint associated with the meat of mature boars will dissipate over 6–8 weeks following castration.

Entropion

Entropion occurs most frequently in pot-bellied pigs. The large amount of periocular subcutaneous fat, in combination with enophthalmos, contributes to the development of entropion in this type of pig. Clinical signs include epiphora, blepharospasm, rubbing of the affected area, corneal ulceration and vascularization (chronic cases), conjunctivitis with purulent discharge, and rolling in of the eyelid.

In most cases the problem is confined to the lower eyelid, but it may also involve the upper eyelid. The condition may be unilateral or bilateral. Surgical correction is usually needed. General anaesthesia is required. The length and width of skin to be resected can be estimated by pinching the skin below the lower lid margin. A pair of forceps is placed 3–5 mm below and parallel to the lower lid margin. A test length and width of skin are held between the jaws of the forceps and the position of the lower eyelid margin assessed. The position of the forceps should be adjusted until the entropion is resolved without creating an ectropion. The ridge of skin marked by the jaws of the forceps is then removed using a scalpel. The skin incision is then closed using interrupted PDS sutures to avoid the need for removal. Complications may include under-correction. If there is involvement of the upper eyelid, the process can be repeated at this site.

Exploratory laparotomy

In order to investigate suspected cases of perforated gastric ulceration, gastric torsion, intestinal obstruction, and intestinal torsion, a midline approach under general anaesthesia is recommended.

Fractures

Porcine fractures are rarely cost-effective to repair, and euthanasia is often the best option. Fracture repair in expensive boars or pet pigs may be possible. Mature commercial boars may weigh 250–300 kg, with heavy musculature, making repair of femoral or humeral fractures extremely difficult. External fixation and internal fixation of bones of the lower limbs have been used with success in pigs when used appropriately. The procedures used in small animal orthopaedics are applicable to pigs.

Hernia: inguinal

In commercial pigs, inguinal hernias are most commonly found on the left side. The contents of the inguinal hernia, which are usually omentum and intestines, can be examined using palpation and ultrasonography. If the contents are reducible once the testicle is exteriorized, the spermatic cord can be massaged and twisted, starting at the testicle, which will force the contents back into the peritoneal cavity. The twisted spermatic cord and tunica vaginalis can then be ligated and the testicle removed. A horizontal mattress suture can then be placed across the inguinal canal to close it. If the contents are irreducible, the tunica vaginalis should be opened and adhesions broken down. Sometimes, it is difficult to replace the herniated contents back into the abdomen. Carefully incising the tunica vaginalis bordering the inguinal canal, and increasing the size of the inguinal canal by cutting the cranial border of the ring with a pair of scissors, may allow reduction of the contents. A paramedian abdominal laparotomy can also be used to facilitate the reduction of the hernia. The contents of the hernia can then be pulled back into the abdomen. The spermatic cord can then be ligated and the testicle removed. Whichever of these techniques is used, the inguinal canal should then be closed with mattress sutures.

Fig 15.2 – Umbilical hernia in a weaner.

Hernia: umbilical

An umbilical hernia is a defect in the abdominal wall at the umbilicus (Fig. 15.2). They are common and affect about 1% of pigs. Males and females are affected. Umbilical hernias may be congenital or follow neonatal omphalitis. There may be multiple abscesses with extensive chronic granulation tissue. Intestinal incarceration and strangulation can occur, and bowel resection may be needed in these cases. Some umbilical hernias are hereditary. In commercial pigs, it is likely that the cost of repair will exceed the value of the animal. General anaesthesia is recommended.

The anaesthetized animal is placed in dorsal recumbency, ideally in a V-shaped trough. In the male the prepuce, prepucial diverticulum, and penis should be reflected in order to access the hernia. The hernial sac can be grasped with forceps and the skin at the base incised. The perimeter of the hernia can be identified by palpation, and this can be felt as a clearly defined ring. If the hernia is reducible, it may be possible to preserve the peritoneal lining of the hernial sac. This can then be pushed back into the peritoneum and the edges of the hernial ring apposed using interrupted monofilament nylon. More usually, there is a need to explore the hernial sac for adhesions, which may need to be broken down, and to check the intestines for ischaemic damage. The edges of the hernial ring can be resected or scarified to facilitate healing. The hernia is closed by apposing the abdominal muscle and peritoneum with absorbable suture material. It may be impossible to facilitate the closure of a large hernia using simple apposition. In such cases, the hernia may be repaired or covered using an inert polypropylene mesh, which can be sutured internally to the peritoneum. No attempt is made to close the hernial deficit. The mesh becomes filled with fibrous tissue. The mesh is sutured in place with monofilament nylon. The subcutis can sometimes be closed over the mesh. The skin is then sutured with non-absorbable sutures.

Hoof trimming

A major predisposition to overgrown hooves is the lack of exercise. If not corrected, the lateral hoof wall may roll under the sole, causing lameness and discomfort with reluctance to exercise. In addition, overgrown hooves often become cracked. The hoof-trimming procedure will need sedation unless the pig is very cooperative. In pigs with normal hooves, there is a straight line from the carpometacarpal joint to the point of the toe and complete sole contact with the ground. Deviation from this usually indicates that hoof trimming and reshaping are required. A hoof rasp, cattle hoof knife, and hoof clippers are required for corrective trimming. In some miniature pigs, the hoof may be severely distorted, and care is needed to avoid over-trimming and injury to the sensitive laminae. In these cases, regular corrective trimming with gradual attempts at improving the hoof conformation will be required. The dew claws sometimes become overgrown and misshapen. These can be trimmed back using the hoof trimmers.

Joint lavage

Joint lavage can be used in the treatment of joint infections. Under general anaesthesia, the skin of the affected joint is aseptically prepared. Two 16 needles are placed at different sites in the affected joint capsule. Warm Hartmann's solution is run into the joint by attaching a giving set tube to the bag and one needle. The fluid exits through the other needle. The fluid pressure can be increased manually by squeezing the bag containing the Hartmann's fluid. Alternatively, the fluid can be flushed through the joint using syringes. The target is to lavage the joint with 0.5–1.0 L of fluid. Ceftiofur powder mixed with sterile water to form an aqueous solution can be placed into the joint capsule at the end of the procedure before the needles are removed. This procedure should ideally be repeated on two further occasions at 48-h intervals, although a single flushing in some cases has been successful. The procedure is expensive.

Mastectomy

Single mammary glands can become chronically infected. They may become grossly enlarged and interfere with piglet suckling and sow comfort. Surgical removal is done under general anaesthesia during the dry period and not during lactation. The early gestation period (up to 2 weeks) and the last 4 weeks of gestation should be avoided to minimize the impact on embryonic or fetal losses. The affected tissue is excised, ensuring that there is sufficient loose remaining skin to effect closure over the

area. Ligation of veins during the operation will minimize haemorrhage. Closure should attempt to eliminate dead space.

Ovariohysterectomy

This procedure may be desirable in pet pigs to reduce or abolish the changes in behaviour associated with oestrus. It is recommended that the operation be performed between 4 and 8 months of age, although it can be performed in older sows. The technique is similar to that used in the bitch. The procedure is performed under general anaesthesia with the animal in dorsal recumbency. An incision is made through the ventral midline of the abdomen. The ovaries are identified and gently elevated to the wound site. Care is needed not to rupture the ovarian pedicle before ligation of the ovarian artery and vein, as the ovarian ligament is relatively weak compared with in the dog. Each pedicle, which includes the ligament, ovarian artery, and ovarian vein, is ligated twice. Before releasing the ligated pedicle, the ovarian stump is held with a pair of Allis tissue forceps to check for signs of haemorrhage. The uterus is elevated towards the wound. A large pair of forceps is used to crush the distal body of the uterus just proximal to the cervix. Each middle uterine artery is ligated. The uterus is ligated distal to the crushing forceps using an anchored ligature. The proximal uterus is then transected and removed. The remaining uterine stump is held with a pair of Allis tissue forceps, and the crushing forceps are removed to check for haemorrhage. The uterine stump is then released. The midline abdominal incision is closed in three layers: the peritoneum–abdominal muscles–linea alba, the subcutis, and the skin. Perioperative or postoperative antibiotics and analgesics are essential.

Persistent frenulum

It is usual for the congenital attachment of the penis to the prepuce (the frenulum) to separate before the onset of sexual maturity at 7–8 months of age (or 3–5 months in miniature pigs). Failure of this separation will prevent intromission when mating is attempted. Under general anaesthesia, the persistent frenulum can be identified as a band of tissue connecting the penis and the prepuce. To correct the problem, the frenulum is cut with scissors. Haemostasis is usually not a problem.

Prepucial diverticulum (removal)

The reproductive performance of the boar may be compromised by discomfort caused by ulceration of the prepucial diverticulum and diversion of the penis into the diverticulum at mating. Removal of the lining of the diverticulum may restore breeding soundness. The procedure is performed under general anaesthesia.

The prepucial diverticulum is a bilobed structure. A pair of forceps is passed through the prepucial orifice into one of the lobes. The diverticulum can be supported externally with the other hand, which makes manipulation easier. The lining of the lobe is grasped using the forceps and withdrawn, everting the lobe through the prepucial orifice. This is repeated on the second lobe using a second pair of forceps. The two lobes are then excised by cutting across the base of the everted lobes with the base retained in position by the application of Allis tissue forceps proximal to the incision. The incision created by this procedure should be sutured using non-absorbable suture material. The remaining everted tissue is released. The tissue recoils through the prepucial orifice.

Prolapse: penis

Prolapse of the penis in the boar is uncommon. It may follow damage to the penis sustained during mating activities, and is reported in some boars following overdosing with azaperone. The prolapsed penis may be traumatized once exposed outside the prepuce, and it is advisable to replace the penis to avoid this risk. General anaesthesia is recommended. The penis should be cleaned with an antiseptic solution and swabs. If the penis is extensively damaged, then surgical repair may be necessary before replacement. Lubrication of the penis and massaging of the prepuce will assist in returning the penis to its normal position within the prepucial sheath. A purse string suture using 5 metric monofilament nylon is placed around the prepucial opening and drawn tight around a finger placed in the prepucial opening. This will allow urine to escape but should retain the penis within the sheath. The suture should be removed in 10 days' time. Resumption of sexual activity should be delayed for a further 14 days if the penis was not traumatized. This period should be extended depending on the severity of the penile trauma and the extent of the repair required. In traumatized cases, a course of antibiotics with additional prepucial lavaging is advisable.

Prolapse: prepuce

Prolapse of the prepuce results in eversion of the prepucial mucosa. In neglected cases, the everted tissue may become traumatized and granulomatous. The prolapse may get worse because of the increasing weight of the abnormality. In mild cases, the prolapse can be replaced and retained in position with a purse string suture around the prepucial orifice. In severe cases, amputation (circumcision) of the prolapsed mucosa is the treatment of choice. General anaesthesia is required. Care is required to identify the prepucial diverticulum. It must either be preserved or the diverticulum removed as described above. The prepucial prolapse is extended so that vital prepucial mucosa is visible below the prepucial orifice. The prolapsed tissue

is anchored in position by horizontal sutures around the base of the prolapse in the vital mucosa. The prolapse is amputated using a scalpel blade. The insertion of a plastic tube into the lumen of the prolapse is useful to cut down on to during the amputation. The internal and external sides of the remaining stump are apposed using interrupted absorbable sutures in a clock face pattern. The wound is inverted into the prepuce and a purse string suture placed around the prepucial orifice. This is removed 10 days later. Sexual rest for 30 days should be imposed. A course of antibiotics is recommended.

Prolapse: rectal

Rectal prolapse is a relatively common condition in pigs and has been associated with many factors, including acute diarrhoea, short tail (amputated too short at tail docking), and chilling (causing excessive huddling and piling). The condition is characterized by intermittent or persistent tenesmus. The rectal mucosa becomes everted through the anus. The prolapse may be mild, with only a small amount of mucosa protruding through the anus (sometimes intermittently), or severe, with extensive amounts of mucosa being permanently protruded (Fig. 15.3). The prolapsed tissue may become oedematous, damaged, and devitalized. Other pigs in the group may traumatize the rectal prolapse, and isolation is essential on recognition of the condition until the problem is resolved.

Rarely, bladder retroversion may occur, with the organ becoming entrapped within the prolapse. In this case, there is a large swelling in the prolapse. Catheterization of the bladder can be used to deflate the bladder. Once deflated, the bladder may return spontaneously into the pelvic cavity or can be manipulated to do so by external palpation of the prolapse.

In mild cases with minimal damage, reduction can often be achieved by lubrication and gentle massage of the prolapse. Using local or regional anaesthesia, perianal monofilament nylon sphincter purse string suture, leaving a one- to two-finger anal orifice on tightening, is then used to prevent recurrence. The suture should be left in place for 7–10 days.

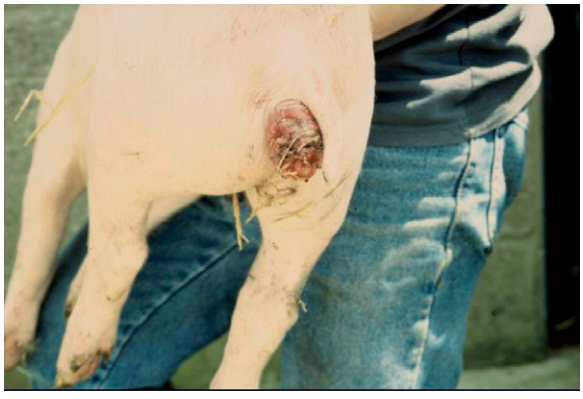

Fig 15.3 – Rectal prolapse. (Courtesy of D. Chennells.)

In severe cases with a large prolapse and traumatized or desiccated mucosae, amputation is required. This procedure is performed under general anaesthesia or sedation and lumbosacral anaesthesia or local perianal infiltration. The simplest method is to use a plastic rectal tube. The plastic tube has a groove around the middle. The tube is positioned within the rectum with the groove adjacent to the anal ring. A tight rubber ring is then applied around the tissue to be amputated with the ring seated in the groove in the rectal ring tube. A lamb or calf elastrator and rubber castration ring can be used for this purpose. Alternatively, a very tight-fitting thick rubber band can be used. Faeces can pass through the tube to allow defecation. The rubber ring cuts off the blood supply to the prolapsed tissue and the tissue undergoes ischaemic necrosis. The necrotic tissue, ring, and tube fall off after 3–7 days. If epidural anaesthesia is used, the inclusion of xylazine will reduce tenesmus for a period of up to 24 h.

Rectal stricture may be a complication following the replacement or amputation of a rectal prolapse. The rectal stricture can be identified by digital examination of the rectum as a tight band with narrowing of the rectal diameter. There is no effective treatment for this condition. In mild cases, the condition will go unrecognized. In severe cases, the animal may have abdominal distension due to physical difficulty is passing faeces. Tenesmus is not usually a feature.

Prolapse: vaginal

This condition most commonly occurs prepartum in late gestation but can occur in the early postpartum period. The vaginal prolapse may be secondary to other causes of tenesmus, such as a rectal prolapse, retroversion of the bladder, and cystitis (Fig. 15.4). In the prepartum animal, it may also arise as a result of the prepartum relaxation of pelvic tissues and increased intra-abdominal pressure. The prolapsed vagina protrudes through the vaginal lips with the external cervix visible. The primary cause should be treated in addition to the repositioning of the vagina back into the pelvic cavity. Sedation and epidural anaesthesia using lignocaine and xylazine are recommended. The prolapse is cleaned and lubricated before gently pushing the prolapse back through the vulval lips. A Buhner's purse string suture (umbilical tape or 5 metric monofilament nylon is used) is placed subcutaneously around the vulva and tightened to reduce the orifice to the width of three fingers. In the prepartum animal, this must be removed once farrowing is imminent. In the postpartum animal, it is removed 7–10 days following the procedure. Treatment with NSAIDs may be useful in reducing inflam-

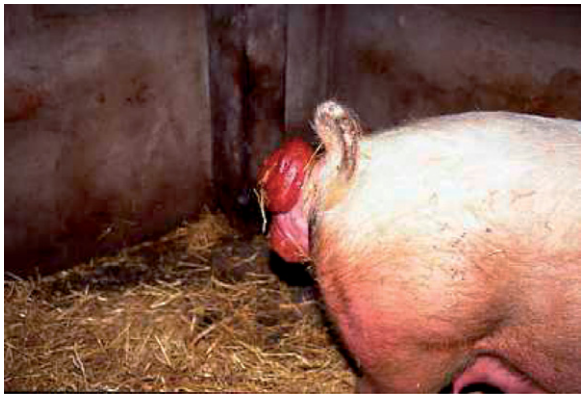

Fig 15.4 – Vaginal prolapse with secondary rectal prolapse.

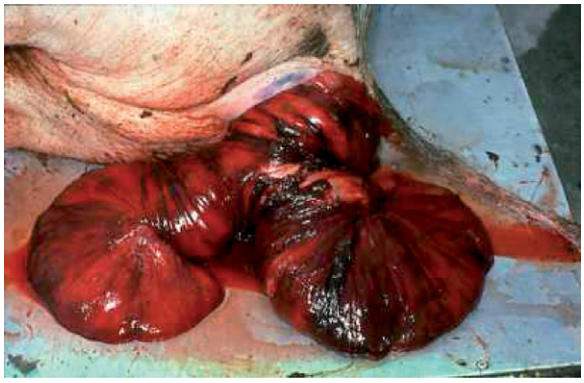

Fig 15.5 – Uterine prolapse in a sow.

mation and providing analgesia. A course of antibiotics may reduce the risk of infection.

Prolapse: uterus

This condition occurs during parturition or within several days of parturition (Fig. 15.5). It is quite common. In many cases, the sow is found dead as a result of haemorrhage from the ovarian or uterine arteries. One or both horns may be involved. If one horn remains in the abdomen, it may contain piglets. The sow may be in shock or may appear to be undisturbed by the condition in recent cases and may continue feeding her piglets. In severely shocked animals, euthanasia on welfare and economic grounds may be indicated.

The treatment is to return the uterus to the normal position. This is complicated in the sow because of the length of the uterine horns. General anaesthesia or deep sedation with lumbosacral epidural anaesthesia is required. Fluid therapy can be given using the ear vein. Sternal recumbency with the hindquarters elevated is the recommended position to facilitate the replacement of the uterus through the vulval lips. Any uterine lacerations should be repaired before replacement. Oedema of the uterus may be reduced by the application of sugar granules or oral electrolyte powder to the uterus to create a hypertonic surface layer to draw out fluid by osmosis. This process takes 10–15 min following the application. The application of lubricants is not recommended, as it is difficult to manipulate the uterus.

The body of the uterus should be progressively returned to the pelvic cavity, starting at the vulval lips. Then each uterine horn should be inverted back into the pelvic cavity. Once this has been achieved, 3 L of sterile saline can then be instilled into the uterine lumen to facilitate inversion of the uterine horns. It may be impossible to replace the uterus. In this situation, the uterus can be pulled back into the abdomen through a left flank laparotomy by grasping one of the uterine horns. An assistant can assist this process by applying gentle external pressure to the uterine horn – the 'pull and push' approach.

Following replacement, oxytocin should be given to facilitate uterine involution. A course of antibiotics and analgesics should be given. Some clinicians place a subcutaneous purse string suture round the vulva to 'prevent recurrence'. It is unproven that this has a value.

In some sows, the prolapsed uterus may be severely and extensively damaged or impossible to replace. In these cases, if the sow is to be kept for the duration of the lactation, amputation of the uterus is indicated. See under *Amputation: uterus*.

Tail docking

Tail docking can be performed in the UK only where there is a recognized problem of tail biting, and where a risk assessment indicates that tail docking will reduce the prevalence of the condition. The procedure is usually carried out during the first week of life, and hygiene of the tail and surgical instruments is important to avoid postoperative infections. Emasculators may be used to crush the tail, and then the tail is removed using a scalpel blade. Alternatively, the tail is amputated using a sharp pair of pliers. The tail should be amputated 1–2 cm from its base to avoid an increased risk of rectal prolapse in later life. Haemostasis of the stump can be facilitated by thermocautery using a gas or electric calf disbudding iron.

Tooth clipping

At birth, the top and bottom corner incisor teeth and canine teeth are usually present. The four canine teeth are very sharp and sometimes cause injury to sows and the other piglets. The sow's teats and mammary gland may be bitten or lacerated during rooting and suckling. This causes pain, and the sow may be reluctant to allow the piglets to feed. If other piglets are bitten or scratched the

wounds can become infected, causing abscessation, joint infections, and predisposition to skin infections. Teeth clipping is allowed in the UK only where there is a recognized problem. The procedure should not be performed before 18 h of age to avoid compromise of colostrum intake. Sharp and clean wire cutters should be used. The teeth should be cut as close as possible to the gum line. Care must be taken to avoid damaging the gum or fracturing the tooth root. If this occurs, there is a risk of tooth root infection and/or bacteraemia, which may result in disseminated infections such as joint ill.

Tusk trimming

These teeth can be hazardous to other animals and people. Complete removal of canine teeth (tusks) is difficult because of the deeply embedded roots, therefore they are usually only trimmed. Restraint by a snare or deep sedation–general anaesthesia is required. A length of embryotomy (obstetrical) wire with a handle on each end is used to saw through the tooth. Clippers should not be used as they can fracture the tooth root, causing pain and a portal of entry for infection. The tooth should be cut just above the gum margin that is above the pulp cavity. Care should be taken to avoid cutting the gums. The edges of the tooth should be rasped to remove sharp edges. Depending on the growth rate of the tusks, they may have to be cut every 6–12 months to ensure that they are not a risk.

Undescended testicles (cryptorchid)

An undescended testicle may be identified at the time of castration. In commercial pig herds the problem is ignored, as further procedures cannot be financially justified. In pet pigs, it is advisable to locate and remove the undescended testicle to reduce undesirable behaviours.

The testicle(s) is usually located within the inguinal canal or within the abdomen at the entrance to the inguinal canal. The scrotal sac should be opened in the manner described for a normal castration. A spay hook can be used to explore the inguinal canal. If the testicle is identified, the hook can be used to pull it through the inguinal canal into the descended position. The testicle is then removed as described earlier. If it is not found following this examination, a laparotomy is required to explore other potential locations between the kidney and the inguinal ring. The penis must be identified before a paramedian incision is made. Once the undescended testicle is located, the spermatic cord should be ligated and the testicle removed.

Vasectomy

Vasectomy may be requested to produce a teaser boar for oestrus detection when using artificial insemination. The procedure is performed under general anaesthesia.

The boar is placed in dorsal recumbency. A 5-cm skin incision is made over each spermatic cord. The vas deferens is identified as a thin white tubular strand within the spermatic cord. The small stab incision is made through the tunica vaginalis, which surrounds the spermatic cord over the vas deferens. Care is needed not to damage the spermatic artery. Using forceps or a cat spay hook, the loop of vas deferens is exteriorized through the incision in the tunica vaginalis. A 3-cm section of the exteriorized vas deferens is isolated between two artery forceps. The section between the two forceps is excised, and each end of the vas deferens is ligated before removal of the forceps. Closure of the tunica vaginalis incision is optional. Some clinicians place a transfixion ligature through each end of the vas deferens and the subcutis to further reduce the possibility of reanastomosis. The skin incision is ideally closed with non-absorbable suture material, although some clinicians use absorbable material because of the extreme difficulty of removing the sutures without further sedation. The procedure is repeated on the other spermatic cord. A postoperative course of antibiotics is advisable. Intraoperative analgesia may reduce postoperative discomfort. Preservation of the removed sections of spermatic cord in 10% formal-saline with appropriate labelling is recommended as evidence that the procedure was performed.

FURTHER READING

Hodgkinson O 2007 Practical sedation and anaesthesia in pigs. In Practice 29:34–39

16

Sampling and post-mortem examination of the pig

Sampling

Selection of animals

It is important to inspect, examine, and sample sufficient animals to identify a problem of significance in a herd. Submission of individual pigs, or samples from individual pigs, are not ideal. Where possible, sample untreated animals. Take samples for attempted bacterial culture from acute cases and from untreated pigs.

The post-mortem examination of representative animals at the regional laboratory is often more cost-beneficial than submitting tissue samples from dead animals. Where possible, and subject to welfare considerations, submission of live animals is preferable to that of dead ones. In the UK, a movement licence would be required. Where possible, animals should be submitted at the acute stage of the disease process.

Packing and sending

Great care is needed when packaging samples, and the packaging must conform to the postal regulations for packaging of pathological material.

Blood samples

Selection of sample tubes

Ensure that the sample taken is in the correct type of tube for the analysis required. This may vary between laboratories. The type of tube and the colour coding used in the UK for commonly requested tests are shown below.

- Green (heparin): copper, glutathione peroxidase, methaemoglobin, haemoglobin, packed cell volume, lead, plasma inorganic iodine.
- Purple (ethylene diamine tetra-acetic acid, EDTA): haematology.
- Grey (oxalate/fluoride): phosphorus and glucose.
- Blue (citrate): clotting times, fibrinogen.
- Red – none (serum): most other blood biochemical parameters and serology.
- All plastic containers without rubber or glass: zinc assays.

The tube should be filled and gently inverted to allow for mixing (serum tubes being the exception). Blood should be kept cool before dispatch, but clotted samples should not be refrigerated until the clot has formed. If only serum is required, it is better to remove serum unless blood can be dispatched promptly, thus avoiding any problems with haemolysis. Enzymes are broken down in the body at varying rates, and most need to be assayed soon after sampling.

Paired serology

A rise in the titre using paired serum samples, with a duration of at least 2 weeks between the first and second sample, provides strong evidence of recent infection in animals over 24 h of age. It is important that the first sample is taken before seroconversion is complete. It is wise to sample several animals in a group to demonstrate a consistent response. Individual identification is important if paired serology is to be used.

Diseases that have serological tests

- *Actinobacillus pleuropneumoniae* (type-specific)
- Aujeszky's disease
- *Brucella* (*abortus*, *melitensis*, *suis*)
- Classical swine fever (CSF)
- Encephalomyocarditis virus
- *Erysipelothrix*
- *Haemophilus parasuis*
- *Lawsonia intracellularis*
- *Leptospira* (range of serovars)
- Listeria
- *Mycoplasma hyopneumoniae*
- *Mycoplasma hyosynoviae*
- Porcine circovirus 2
- Porcine epidemic diarrhoea
- Porcine parvovirus
- Porcine reproductive and respiratory syndrome (PRRS)
- Swine influenza (various strain types)
- Talfan's disease
- Transmissible gastroenteritis
- Porcine respiratory coronavirus

Blood smears

For eperythrozoon investigations, 2 mL of whole blood in an EDTA tube or two thinly spread films fixed in methanol are required.

Tissues

Tissue samples for histopathology

Tissue samples for histopathology should not be more than 1 cm thick. A representative sample that contains the normal tissue and the junction between the gross abnormal lesions and the normal tissue should be included. The sample should be placed in 10–20 times their volume of fixative and should be sent in a suitably sized container with a wide top to facilitate removal. The recommended fixative for most cases is 10% formol saline. The brain should be fixed whole to allow appropriate samples to be selected at the laboratory. Samples of intestine should be collected as soon as possible following death. The inclusion of samples from several sites of small and large intestine is recommended. Lengths of 1–2 cm are suitable. Crushing with forceps should be avoided, otherwise the quality of the histopathology may be affected. The sample should be shaken gently in the fixative to help displace food material. Fixation of most small samples will be completed in 24–48 h. Cases can be sent immediately if the container is filled with fixative, so that primary fixation occurs in transit. If non-urgent, tissue can be initially fixed for 48 h then sent in a reduced volume of fixative.

Tissue samples for biochemical or toxicological analysis

For most biochemical assays, 10 g of liver is the tissue of choice. When sampling cases of suspected poisoning, submit 10 g of liver, 10 g of kidney, stomach contents, fat, and muscle.

Sampling for aerobic bacteriology

In the practice laboratory, bacterial growth and antimicrobial sensitivities can be achieved in 6–8 h. In other laboratories, growth may require 24 h of incubation and antibiotic sensitivity testing a further 24 h. Some organisms require more prolonged culture times, for example *Salmonella* (3 days minimum), *Campylobacter* (up to 5 days), *Mycoplasma* (10 days to 4 weeks), and dermatophytes (3 weeks). Faecal samples in addition to swabs may be useful if further testing is required. The best technique when swabbing is to sear the surface of organs with a flame or heated scalpel blade prior to swabbing. Alternatively, samples can be obtained from sections of tissue cut with a sterile scalpel blade. The swab samples should be submitted in a suitable transport medium such as charcoal transport medium (this should not be used when fluorescent antibody test examination is required).

Sampling for anaerobic bacteriology

When using swabs, it is important to use anaerobe transport medium; when sending fluid samples, it is important to ensure that the container is filled to exclude air. When clostridial enterotoxaemia is suspected, send a minimum of 2 mL of small intestinal contents collected from several sites in the ileum. If *Clostridium novyi* infection in pigs is suspected, make several impression smears from the cut surface of affected muscle or liver, air dry, and send in a slide box for a fluorescent antibody test, or submit the tissue specimen in a sealed airtight container.

Cytology

A sample of aspirated fluid should be placed in an EDTA container. Several smears should be made in the same way as blood smears then air dried and fixed in methanol for a minimum of 5 min.

Faeces and skin

Faeces

Faecal samples should contain at least 10 g of faeces in a wide-mouthed, screw-capped container.

Skin

Submit deep scrapings or (preferably) earwax for sarcoptic mange, hair for ringworm, and undamaged specimens of lice in screw-capped containers.

Diseases and samples

Tables 16.1–16.7 provide examples of samples requested by a laboratory by disease. Always check with the diagnostic laboratory to be used which samples are required.

Euthanasia of pigs on farm

It is advisable for any method using shooting that a snare is used to restrain the pig, with the person holding the rope standing in front of the pig and behind the operator.

The method to be used depends on the age of the pig and the material required for post-mortem. A lethal dose of barbiturates is a desirable method. Intravenous injection of a lethal overdose, using appropriate restraint or chemical sedation, can be used in any age of pig. However, physical restraint can be difficult in larger pigs. The intracardiac and intraperitoneal routes are particularly useful in small pigs.

Captive bolt pistols can be used to stun pigs. Following stunning, the animal should be pithed or bled out to kill the pig. The Cash Special Captive Bolt using a 0.22 3 g Green cartridge is suitable for all sizes of pig. The site of shooting is in the midline of the forehead, one finger-width above the eye level. The muzzle should be placed against the head and directed towards the brain. Bleeding

Table 16.1 Neonates and sucklers: enteric disorders

Condition or infection	Specimen	Comments
Diarrhoea or death in all age groups	Carcasses or live piglets (preferably)	Submit acutely affected piglets, particularly if transmissible gastroenteritis or porcine epidemic diarrhoea is suspected.
Enterotoxigenic *Escherichia coli*	Rectal swabs, faeces, carcasses	Culture, serotype, and antimicrobial susceptibility.
Non-enterotoxigenic *E. coli* coliforms (e.g. attaching and effacing *E. coli*, enterohaemorrhagic *E. coli*)	Live pigs	Culture, serotype, antimicrobial susceptibility, histopathology.
Clostridial necrotic enteritis	Carcasses or necrotic bowel with contents	Gross pathology, Gram smear, culture, ELISA for toxins.
Salmonella	Faeces (preferably >15 g); swabs are less sensitive	Culture. Infection is often subclinical but of public health significance.
Coccidiosis	Live untreated acutely scouring piglets	Histology of intestine essential. Faeces usually negative for piglet oocysts during early clinical period. Mucosal smears are of limited diagnostic value.
Cryptosporidium spp.	Faeces or large intestinal contents, live piglets	Often subclinical.
Rotavirus	Faeces or intestinal contents	Polyacrylamide gel electrophoresis test.
Transmissible gastroenteritis	Live pigs; faeces or intestinal contents	Antigen ELISA fluorescent antibody test or serology.
Porcine epidemic diarrhoea	Live pigs; faeces or intestinal contents	Antigen ELISA, serology.

ELISA, enzyme-linked immunosorbent assay.

Table 16.2 Weaners and growers: enteric disorders

Condition or infection	Specimen	Comments
Escherichia coli, *Salmonella*, rotavirus, transmissible gastroenteritis, and porcine epidemic diarrhoea as with neonates and sucklers	Live pigs, carcasses, fresh faeces	–
Swine dysentery (*Brachyspira hyodysenteriae*, spirochaetal and non-specific colitis)	Fresh faeces, carcasses, colon or caecum, large intestinal contents	PCR, gross pathology, histology, fluorescent antibody test or anaerobic culture.
Proliferative enteropathy or adenomatosis (*Lawsonia intracellularis*)	Carcasses or live pigs, faeces, clotted blood	Gross pathology, histology, silver stains, PCR, modified Ziehl–Neelsen smears, serology.
Bowel oedema	Carcasses	Gross pathology, culture, serotyping verocytotoxic *E. coli*.
Parasitic gastroenteritis	Faeces	Worm egg count.
Gastric ulceration	Carcasses	Gross pathology.

PCR, polymerase chain reaction.

Table 16.3 Adults: enteric disorders

Condition or infection	Specimen	Comments
Swine dysentery, parasitic gastroenteritis, *Salmonella*, proliferative enteropathy or adenomatosis	Faeces, carcasses	Polymerase chain reaction, gross pathology, histology, fluorescent antibody test or anaerobic culture.
Iron deficiency anaemia	Blood (EDTA or heparin)	Haematology or iron biochemistry.
Mastitis	Milk (sampled aseptically)	Culture.

EDTA, ethylene diamine tetra-acetic acid.

Table 16.4 Heart diseases

Condition or infection	Specimen	Comments
Endocarditis	Carcasses, swab from vegetative lesion	Culture.
Mulberry heart disease	Carcasses, fixed heart, blood (clotted or heparin)	If vitamin E or selenium deficiency is suspected, consider blood sampling a representative group.

Table 16.5 Respiratory diseases

Condition or infection	Specimen	Comments
Swine influenza, porcine reproductive and respiratory syndrome, porcine respiratory coronavirus	Blood: paired at 2- to 3-week interval or group sampling carcasses	Gross examination and histopathology and immunohistocytochemistry.
Progressive atrophic rhinitis (toxigenic *Pasteurella multocida*)	Nasal or tonsil swabs, snouts collected at slaughter, monitoring of chronic damage to turbinates and nasal septum	Culture: note that it is essential to sample before obvious clinical signs. (Submit swabs from at least 20 pigs where the level of overt disease is low.)
Actinobacillus pleuropneumoniae, *Pasteurella multocida*, *Haemophilus parasuis*	Lungs	Culture and sensitivity.
Enzootic pneumonia (*Mycoplasma hyopneumoniae*)	Lungs	For monitoring of gross lesions, examine at least 20 lungs. Culture.

Table 16.6 Other conditions

Condition or infection	Specimen	Comments
Postweaning multisystemic wasting syndrome or PDNS	Four to six acutely affected pigs (usually 8–12 weeks old) or formalin-fixed lymph nodes (inguinal, mesenteric, and renal) and kidney from at least three acutely affected pigs	Gross pathology, histopathology and immunohistocytochemistry if appropriate. Serology for porcine circovirus 2.
Sudden death	Carcasses (anthrax test)	Peripheral blood smears and peritoneal fluid smears.
Locomotor disorders	Blood (clotted, paired samples), joint fluid aspirate	Serum agglutination test for *Erysipelothrix rhusiopathiae*, culture for *Mycoplasma hyosynoviae* and *Erysipelothrix* spp.
Erysipelas and *Mycoplasma*	Carcasses	
Osteochondritis dissecans, spontaneous fractures	Carcasses	
Nervous disorders (e.g. streptococcal meningitis, salt poisoning, oedema disease)	Carcasses, meningeal swabs for bacteriology, fixed brain	Culture, histopathology.
Skin diseases: exudative epidermitis, PDNS, mange, ringworm, swine pox	Carcasses, skin scrapings, swabs	Culture, histopathology.
Urogenital disease; cystitis or pyelonephritis	Carcasses, urine sample	Gross pathology, histopathology, culture.
Polyserositis and Glasser's disease	Carcasses, serosal surface swabs	Culture.
Eperythrozoonosis	Blood (heparin) or fresh smears	Preferred sample is a blood smear prepared from a live pig, air dried and methanol-fixed on the farm.

PDNS, porcine dermatitis and nephropathy syndrome.

Table 16.7 Abortion, stillbirths, and infertility

Condition or infection	Specimen	Comments
Most bacterial infections	Fresh fetal stomach contents, liver and cervical–vaginal swabs	Culture.
Parvovirus	Lung and liver	Fetuses less than ~17 cm crown–rump length or mummies: thoracic fluid ELISA for antigen. Fetuses greater than ~17 cm: haemagglutination inhibition test for single maternal bloods antibody (sows will have seroconverted).
Erysipelas, swine influenza, leptospirosis, and PRRS	Maternal bloods	Single or paired serology.
Cause uncertain	Whole litter of fetuses with fetal membranes and maternal bloods (clotted, heparin)	–
Infertility, other reproductive disorders (e.g. vulval discharge)	Vaginal–cervical swabs taken via speculum to avoid contamination	Culture: misleading results may occur because of urinary disorders.
Returns to service	Group blood samples, paired serology	Serology for evidence of active PRRS, swine influenza, leptospirosis, erysipelas, and parvovirus. Most regular returns to service are physiological or managemental.

ELISA, enzyme-linked immunosorbent assay; PRRS, porcine reproductive and respiratory syndrome.

out is achieved by severing the major blood vessels (carotid arteries and jugular veins) by a deep cut with a sharp knife across the neck. Pithing requires the insertion of a plastic or metal rod into the entry hole made by the captive bolt in the skull, followed by destruction of the brain–spinal cord by movement backwards and forwards of the pithing rod.

A free bullet humane killer will kill all ages of pig by the release of a single bullet. The site of shooting is the same as that described for the captive bolt. It is not recommended for use in buildings, and the possible exit of the bullet and ricochet should be carefully considered when a site is chosen. Everyone present should stay behind the operator.

A 12-bore shotgun can be used to kill larger pigs by the release of shotgun pellets. The muzzle of the gun should be held between 5 and 25 cm away from the head. The site of the shooting can be either the same as the captive bolt or through the eye or from behind an ear, pointing in the direction of the brain. It is not recommended for use in buildings, and the possible exit of the shotgun pellets and ricochet – although unusual – should be carefully considered when a site is chosen.

Fetuses

In abortion or stillbirth investigations, three or four entire fetuses and placentas should be sent to the laboratory to optimize the isolation and identification of causal infectious agents. If on farm, post-mortems are the only realistic option to obtain fresh samples, and then sterile scissors and forceps should be used when preparing the samples. The following samples should be submitted.

- Bacteriology: liver and lung.
- Virology: lung, kidney, spleen, and brain.
- Histopathology: lung, heart, liver, kidney, brain, and placenta.
- Serology: serum from fetuses greater than 20 cm and a representative number of affected sows.

Stillbirths

Stillborn pigs are usually still covered in fetal membrane. Their hooves have a white covering, indicating that they have never walked. The lungs have not inflated and are plum-coloured. They sink in water. Meconium is sometimes found in the respiratory tract, indicating peripartu-rient fetal stress. In newly born pigs, the tissues will be quite fresh if the animal died during farrowing. Soft mushy tissue indicates death a few days before birth, and mummified fetuses indicate an earlier death.

Neonatal piglets

Piglets weighing less than 800 g usually do not survive. A white covering of the hooves may indicate that the piglet was too weak to walk. Absence of milk in the stomach is a sign that the animal has not suckled; there was no milk available or access was denied. Sunken eyes are a sign of dehydration. Swollen joints indicate an infectious arthritis. Animals that have died but are otherwise in good

condition may have been overlayed. Bite wounds on the body may have been the result of savaging by the sow or gilt. Anaemia presents as pallor of the skin and mucous membranes. Animals may be in poor condition as a result of starvation or more chronic disease.

Post-mortem technique and diagnostic gross pathology for the suckling, weaner, grower, and adult pig

A fresh carcass or ideally euthanasia of one or more representative animals should be used. In cases of sudden death, blood and peritoneal fluid smears followed by staining with polychromatic methylene blue may be appropriate to eliminate anthrax as a cause. The age and identification of the animal should be carefully noted. A sharp knife or scalpel, scissors, and forceps are required to perform the post-mortem. A wheelbarrow is very useful for transporting the carcass following the post-mortem. For sample collection, labelled bottles of 10% formol saline, sterile plastic bags, glass slides, sterile swabs with transport medium, markers, and labels will be needed. The post-mortem should be carried out in a sheltered area of the farm on a concrete floor that can easily be cleaned and disinfected.

If euthanasia is being performed, blood samples for haematology, serology, and blood biochemistry should be collected before euthanasia in case these are required later.

External and internal lymph nodes should be examined for abnormalities during the post-mortem.

The post-mortem technique is illustrated in Figures 16.1–16.10.

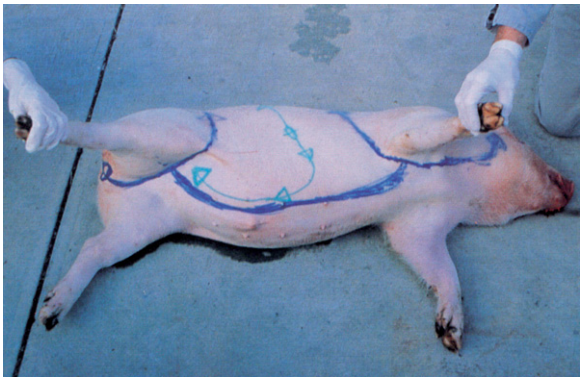

Fig 16.1 – Pig showing cut lines: blue lines for skin cuts, green lines for opening up the peritoneum. (Courtesy of the Pig Research and Development Corporation.)

Examination of the external carcass

The external carcass should be carefully examined for abnormalities. This should include the skin, orifices, eyes, ears, feet, and general body condition.

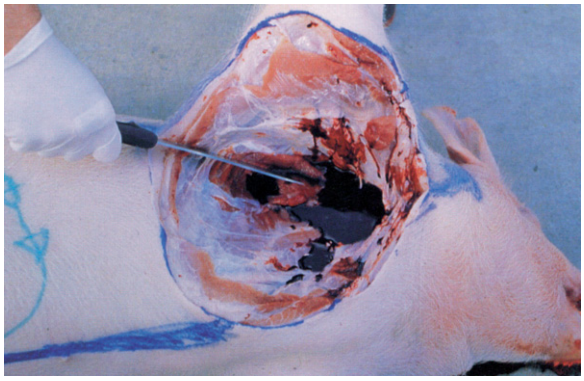

Fig 16.2 – A cut is made between the sternum and foreleg. The leg is reflected laterally. (Courtesy of the Pig Research and Development Corporation.)

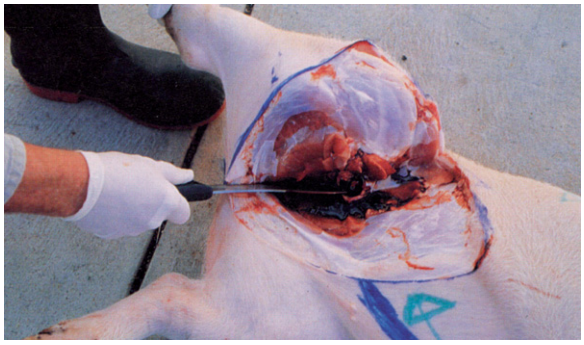

Fig 16.3 – A cut is made down to the hip joint. (Courtesy of the Pig Research and Development Corporation.)

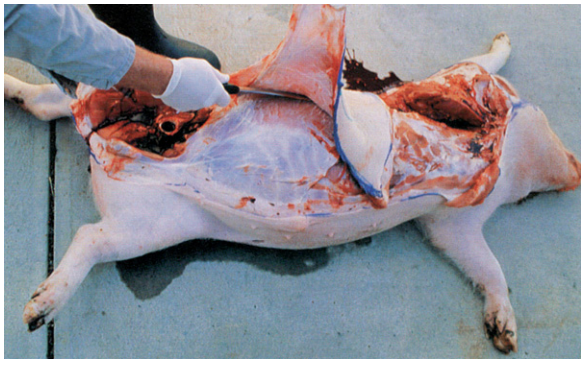

Fig 16.4 – The skin is dissected off the abdominal musculature. (Courtesy of the Pig Research and Development Corporation.)

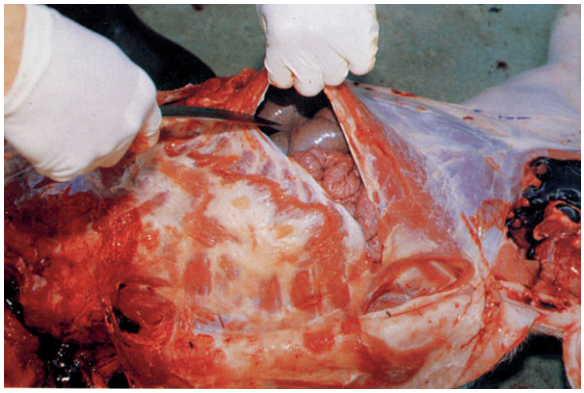

Fig 16.5 – Entry into the peritoneal cavity: the abdominal musculature is cut below the rib and elevated with a finger. (Courtesy of the Pig Research and Development Corporation.)

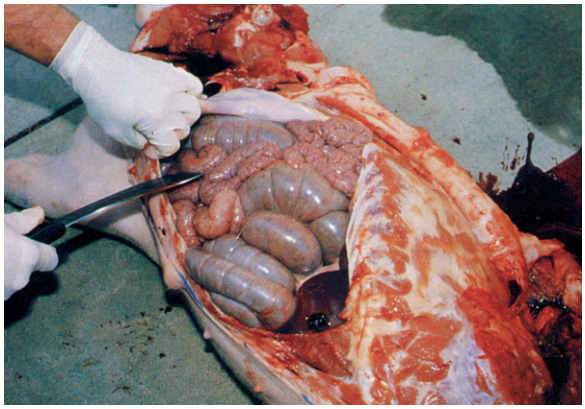

Fig 16.6 – Exposure of the peritoneal contents: the abdominal musculature is removed from the ventral abdomen. (Courtesy of the Pig Research and Development Corporation.)

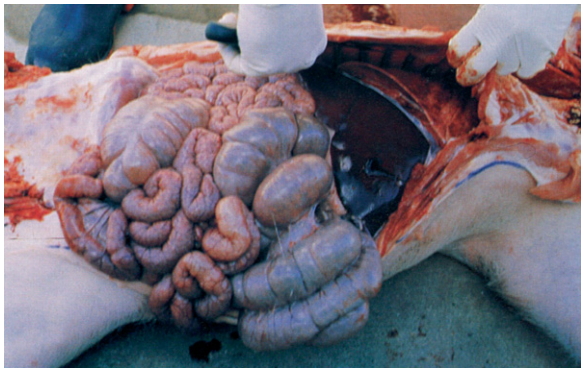

Fig 16.7 – The diaphragm is cut. (Courtesy of the Pig Research and Development Corporation.)

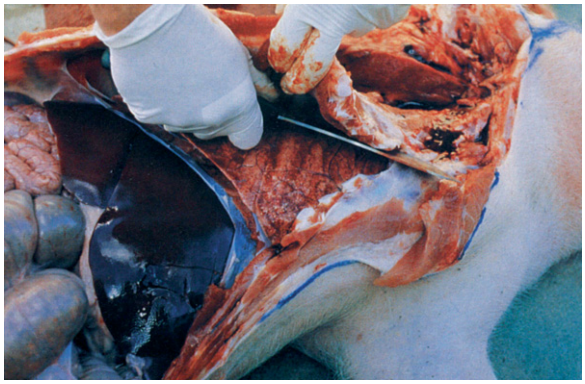

Fig 16.8 – Exposure of the thoracic contents: the ribs are cut at the costochondral junction. (Courtesy of the Pig Research and Development Corporation.)

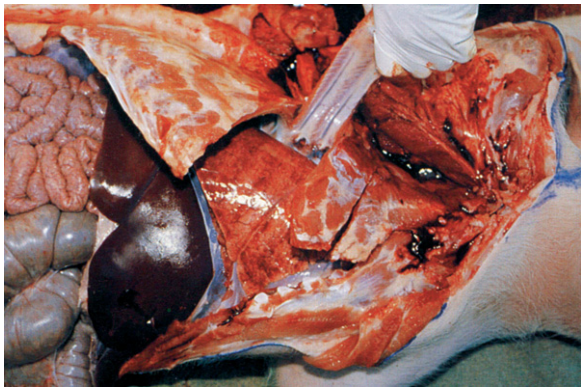

Fig 16.9 – Exposure of the thoracic contents: the intercostal muscles are cut between the ribs and the ribs disarticulated. (Courtesy of the Pig Research and Development Corporation.)

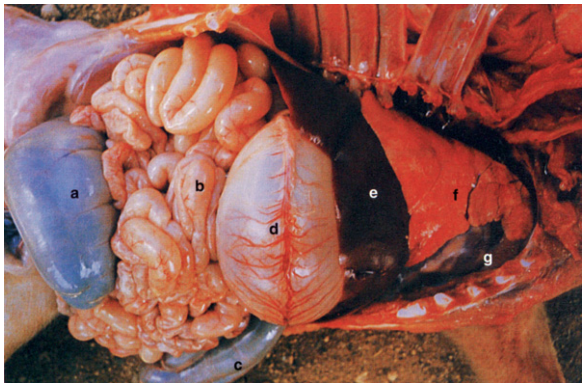

Fig 16.10 – Exposed contents of the peritoneal and thoracic cavities: a, caecum; b, small intestines; c, large intestines; d, stomach; e, liver; f, lungs; g, heart. (Courtesy of the Pig Research and Development Corporation.)

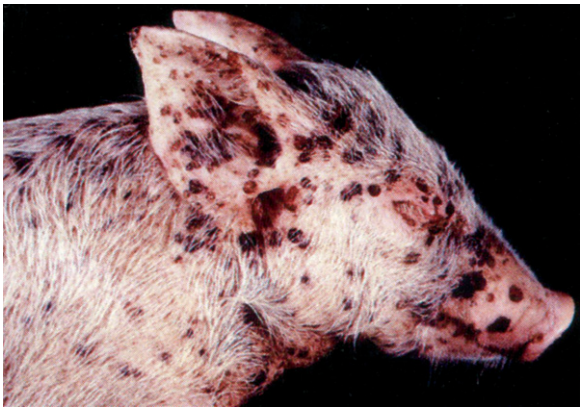

Fig 16.11 – Chronic exudative epidermitis. (Courtesy of the Pig Research and Development Corporation.)

Suckling piglet

Abnormalities of the skin may include necrosis of the ears, tail, or teats; generalized greasy brownish discoloration of exudative epidermitis (greasy pig disease; Fig. 16.11), and purple discoloration in septicaemic conditions. Pallor may be seen with anaemia. Jaundice may be seen in isoimmune haemolytic anaemia. Pityriasis rosea lesions with characteristic ring-shaped areas that coalesce into a mosaic pattern may be present. Pustular dermatitis (streptococcus) is characterized by pustular lesions and circular scabs. Swine pox presents as generalized multiple white macules. There may be erosion and bruising on the feet, and skin abrasions over the carpal joints. Enlarged joints indicative of joint infections may be self-evident.

Weaner, grower, and adult

Swelling of the eyelids may be present in oedema disease. In chronic cases, the carcass may be emaciated (e.g. in postweaning multisystemic wasting syndrome, PMWS). Localized erythema (red), purple-black discoloration, or cyanosis (blue) of the skin may be seen in systemic infections such as erysipelas, salmonellosis, and streptococcal infections. Erythema and haemorrhages of the skin may be seen in swine fever, African swine fever (ASF), and porcine dermatitis and nephropathy syndrome (PDNS). In PDNS, cutaneous haemorrhages (red circular or oval skin lesions) are most obvious on the hind legs, perineum, and flanks. Jaundice may be seen with *Leptospira interohaemorrhagiae*, *Eperythrozoon suis*, PMWS, and *Ascaris suum* (causing bile duct obstruction) infestations. Pallor will be seen with anaemia, as seen with PMWS.

Skin lesions such as erythematous macules or papules and thickened cracked and scaly skin may indicate sarcoptic mange. Lice (*Haematopinus suis*) or lice eggs may be observed. In vesicular diseases – which include foot and mouth disease, swine vesicular disease, vesicular exanthema, and vesicular stomatitis – primary vesicles may be seen on the snout, oral mucosa, coronary band, interdigital space, and teats. Ruptured vesicles appear as raw, ulcerated areas.

Subcutaneous abscesses may be seen as swellings anywhere on the body. Decubital lesions may be observed over the spine of the scapula, the hip joints, and the caudolateral area of the hocks. Lesions caused by ringworm, pityriasis rosea, and parakeratosis (zinc deficiency) may be present. Ringworm produces erythematous patches that may be quite large. In the chronic form of ringworm, the patches become brown or grey with a dry crust. Parakeratotic skin as seen in zinc deficiency is grey-brown, dry, and roughened, with a scaly crust. In outdoor pigs, sunburn and photosensitivity may be seen as erythematous, oedematous areas with blistering followed by necrosis, scaling, and peeling of the skin.

Foot lesions may include swelling (bacterial infections), sloughing of the horn (trauma to accessory digits, foot and mouth disease), cracks (origin: coronary band – true sand crack), white line (false sand crack), biotin deficiency, overgrown hooves, bruising and thickening of heel (trauma), vesicles and erosions (vesicular diseases), solar ulceration, and overgrown hooves.

Examination of the mammary gland and external genitalia

The mammary gland of the sow and the external genitalia of the male can be examined before opening the carcass.

Mammary gland

In the sow, the mammary glands should be examined for signs of inflammation and infection. In acute mastitis, the skin over the affected gland(s) is often hyperaemic. The gland may be enlarged or indurated and misshapen. Cutting into the gland may indicate an acute inflammatory response or chronic fibrosis.

External male genitalia

In the male, the external genitalia should be examined for abnormalities. Cutting into the prepucial diverticulum and the testis will reveal any gross abnormalities. Dissection of the penis by incising the prepuce will allow a more detailed examination. Abnormalities may be seen in the prepuce (prolapse), prepucial diverticulum (diverticulitis, necrosis, ulceration), penis (persistent frenulum), scrotum (adhesions), swellings (hernia, haematoma), testes (hypoplasia, degeneration, enlargement, orchitis), or epididymis (epididymitis, spermatocele). Once the abdominal organs have been removed, it is possible to examine the vesicular glands, prostate, and the bulbouretheral glands for abnormalities.

Positioning the carcass for post-mortem examination and removal of the skin

The preferred position for the post-mortem is in lateral recumbency. The body is stabilized in this position by adducting the upper limbs. This is achieved by cutting through the skin down into the axilla of the upper foreleg and through the hip joint and muscles of the upper hind leg. The upper limbs are reflected laterally. The skin is incised along the ventral midline, and the skin is removed to reveal the ribs and abdominal musculature. The superficial lymph nodes (precrural, prescapular, inguinal, and axillary) should be identified and examined.

Examination of lymph nodes

When examining lymph nodes, relate any abnormal findings to their drainage area. Consider if the response is localized or generalized. Their relative size should be assessed. The exterior should be examined for abnormalities. The lymph nodes should be cut longitudinally to examine the cortex and medulla for congestion, haemorrhage, necrosis, or abscessation. Enlargement is usually due to oedema, inflammation, haemorrhage, emphysema, benign lymphoid hyperplasia, or neoplasia. Generalized enlargement is associated with septicaemias (e.g. erysipelas and salmonellosis) or systemic viral infections (e.g. PRRS, CSF, ASF, PDNS, and PMWS). In PMWS, the inguinal lymph nodes are enlarged and easily palpable, although the lymph nodes are not haemorrhagic in this condition. Oedema of the lymph nodes is seen in oedema disease (mesenteric lymph nodes) and mulberry heart disease (mesenteric, inguinal, and axillary lymph nodes). Haemorrhagic lymph nodes occur in salmonellosis, anthrax, CSF, ASF, erysipelas, clostridial infections, PDNS, thrombocytopenia purpura, and most septicaemias. *Mycobacterium avium* and *bovis* (and tuberculosis) granuloma may be found in the draining lymph nodes at the portal of entry to the body (mesenteric, bronchial, and retropharyngeal). Enlargement of the thymus may indicate a lymphoma.

In the suckling piglet, septicaemias may cause congestion and darkening of the nodes. Iron injections will cause darkening of the subcutis and musculature around the site of an iron injection. The regional lymph node also becomes darkened. Pigs with PMWS present with a lymphadenopathy with noticeable enlargement of the inguinal lymph nodes.

Opening the peritoneal cavity

The peritoneal cavity is opened by making a small incision through the abdominal musculature parallel to the last rib. The incision is then extended using scissors so that the abdominal muscles are removed from the upper part of the abdomen down to the midline along the entire length of the flank.

Examination of the peritoneum and the peritoneal cavity

In the normal animal, the peritoneum will contain a small amount of amber-coloured transparent peritoneal fluid. Abnormal colour, position, and size of the abdominal organs, and the presence of fibrin, adhesions, and abnormal quantities and type of peritoneal fluid, should be noted.

Gut rupture will result in gastrointestinal contents being present in the peritoneal cavity. Fibrinous tags on the peritoneum and adhesions between organs reflect a long-standing peritonitis or inflammation of an underlying viscus. Umbilical and congenital structures (urachus, umbilical arteries, and umbilical vein) may be infected and may become abscessated or contain pus.

Excessive quantities of fluid in the peritoneum may be transudates (from hepatic disease and other conditions causing hypoproteinaemia), modified transudates (from haemoperitoneum, uroperitoneum [ruptured bladder], or heart failure), or exudates (e.g. Glasser's disease presents with a serofibrinous or fibrinopurulent peritonitis).

Opening the thoracic cavity and the pericardium

To open the thoracic cavity, the diaphragm is identified under the last rib and the attachments to the rib cage are cut. Using a knife, the cartilage between the costochondral junction and the sternebrae is cut from the xiphisternum to the thoracic inlet. In young animals, the rib cage can be reflected manually, which results in fracture of the proximal ribs. In adult pigs, the proximal part of the rib has to be cut using bone cutters (hedge secateurs are an effective substitute). This can be achieved by cutting down the intercostal muscles between adjacent ribs and cutting each rib just below the articulation with the thoracic vertebrae. The pericardium surrounding the heart is cut open in situ. The pleura should be examined.

Examination of the pericardium

The pericardial sac is normally thin and transparent. It contains a small amount of fluid. There are no adhesions with the epicardium. Pericarditis may be recognized by pericardiomyocardial adhesions, increased pericardial fluid (often serosanguineous), and a thickened pericardium. Glasser's disease presents with a serofibrinous or fibrinopurulent pericarditis. In mulberry heart disease, the pericardium is distended with serous pericardial fluid containing fibrin strands. In PDNS and ASF, there can be distension of the pericardium resulting from accumulations of serous fluid in the pericardial sac.

Examination of the pleura

Adhesions indicating pleurisy may be observed. In the normal animal, there are only small quantities of pleural fluid. Abnormalities such as an increase in quantity or changes in colour (bloody) or clarity (turbid, flocculent) should be noted.

Pleuritis may be seen in association with enzootic pneumonia. *Haemophilus parasuis* (Glasser's disease), *Mycoplasma hyorhinis*, *Actinobacillus pleuropneumoniae*, and ASF present with a serofibrinous or fibrin purulent pleuritis. Serous fluid can be present in PDNS and ASF infections. Multiple subpleural petechial and ecchymotic haemorrhages may be present in CSF, ASF, and septicaemias.

Removal of the heart and lungs from the thoracic cavity

Removal of the lungs and heart from the thoracic cavity can be achieved by cutting transversally through the trachea, the oesophagus, and the major blood vessels in the upper cervical area. The heart and lung are pulled up using the trachea, oesophagus, and blood vessels for traction, and the attachments ventral and dorsal to the thoracic cavity are severed. The 'pluck' is elevated out of the thoracic cavity, and the oesophagus is cut just proximal to the stomach. Ligation of the oesophagus before cutting is recommended to prevent contamination by refluxing material. The thoracic organs can then be removed from the thoracic cavity.

Examination of the lungs

In the healthy animal, the upper lung surfaces are pink and smooth with a soft rubbery texture on palpation. The lower lungs are normally darker due to hypostatic congestion. Oedematous lungs are heavy and wet with a rubbery consistency, and copious amounts of clear to serosanguineous fluid are present at cut surfaces. Multiple infections are common. The presence of adhesions between the lung lobes indicates pleuritis. Oedematous lungs occur in pneumonic, systemic, and cardiovascular conditions. Firm dark brown or grey areas indicate pneumonia. Abscesses may be a sequel to chronic bronchopneumonia (*Arcanobacter pyogenes* and *Pasteurella multocida* are common causes). Cutting down the trachea and into the major bronchi will reveal any abnormal contents. Squeezing the cut surface of a lung lobe may reveal purulent material in the bronchioles.

Neonatal and suckling piglets

Meconium in the trachea or major bronchi may be present in piglets that have died from fetal anoxia and inhalation of meconium as a result of fetal stress. Complete atelectasis of the lungs indicates that the lungs have not expanded and the piglet has been stillborn. The collapsed lung is usually red-blue to purple. Pneumonia in piglets may be caused by *Bordetella bronchiseptica* (scattered red patches), *Pasteurella multocida*, and *Streptococcus suis* (grey ventral consolidation). *Escherichia coli* septicaemia can produce a fibrinous pleural and peritoneal exudate. In PRRS, gross lesions are usually absent.

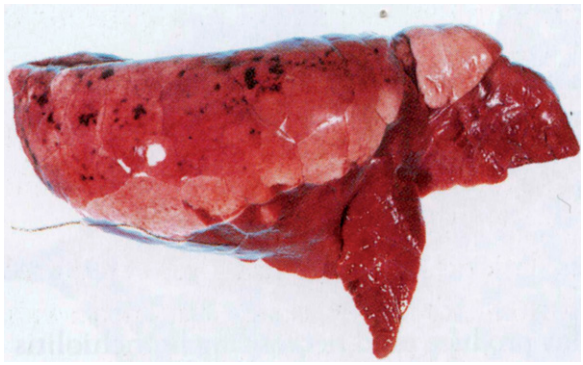

Fig 16.12 – Mycoplasma pneumonia: cranioventral bronchopneumonia of the lung lobes. (Courtesy of the Pig Research and Development Corporation.)

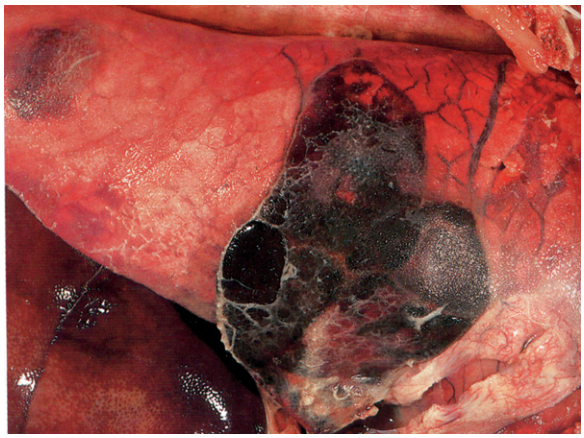

Fig 16.13 – *Actinobacillus pleuropneumoniae*: the right middle lobe is dark red due to extensive haemorrhage, and the lung is coated in a sheet of fibrin. (Courtesy of the Pig Research and Development Corporation.)

Weaners, growers, and adults

Streptococcus suis or *Pasteurella multocida* pneumonia may cause a purulent to fibrinous bronchopneumonia with patchy lobular consolidation with fluid or fibrin in the pleural cavity. Mycoplasma pneumonia is characterized by darker sunken areas at the ventral edges of the lung lobes (Fig. 16.12). Pleuropneumonia is associated with haemorrhages (Fig. 16.13), which may be more common in the caudal lung lobes; cutting into the trachea

and extending the cuts into each major bronchus will reveal blood-stained froth. Haemorrhages on the serosal surface of the lung are also seen in CSF and ASF. In PRRS, gross lesions are usually absent, although the interstitial pneumonia may give the lungs a mottled appearance. Swine influenza causes a bronchointerstitial pneumonia with cranioventral lung consolidation. There is a copious thick mucoid exudate that may be blood-stained in the trachea and bronchi. In chronic CSF, there is a fibrinous pleuritis and pneumonia. In some cases of PDNS, PMWS, and ASF, the lungs are firm and rubbery. Pleurisy may be present. Concurrent conditions may be present, particularly with PMWS. Flotation tests in water will indicate lung segments that are consolidated. Consolidated and pneumonic lung tissue sinks.

Exploration of the bronchi by cutting and squeezing the lungs may reveal thread-like lungworm (metastrongyles). There may also be multiple grey nodules along the ventrocaudal aspects of the diaphragmatic lobes in metastrongyle infestations.

Examination of the heart

The size and colour of the heart should be noted. The ventricles and auricles of the right and left sides of the heart are cut to expose the heart valves.

Congestive heart failure is characterized by hydropericardium, hydrothorax, pulmonary congestion and oedema, ascites and hepatic congestion, and centrilobular necrosis. The heart may be enlarged (congestive heart failure). The valves may be roughened and have fulminating attachments indicating valvular vegetative endocarditis (usually caused by erysipelas or streptococci infections). Haemorrhages on the external surface of the heart are sometimes present in vitamin E or selenium deficiency (myocardium may be pale), CSF, ASF, or septicaemia. In mulberry heart disease and ASF, there is a hydrothorax with effusion of fibrin, pulmonary oedema, and epicardial petechial haemorrhages (Fig. 16.14). Diffuse and focal pallor is seen in conditions causing myocardial necrosis (mulberry heart disease, foot and mouth disease, and encephalomyocarditis viruses). Pericardial adhesions indicate a pericarditis.

Neonatal and suckling piglets

An enlarged pale heart may be seen in iron deficiency. Pale stripes – 'tiger heart' – may be seen in piglets in foot and mouth and encephalomyocarditis viral infections.

Examination of the liver

The liver should be observed in situ. The size, colour, texture, and position should be evaluated. The liver can be removed by cutting the attachments to the diaphragm and other organs. The porcine liver has a reticulated pattern on the surface, which is sometimes described as being similar in appearance to Moroccan leather.

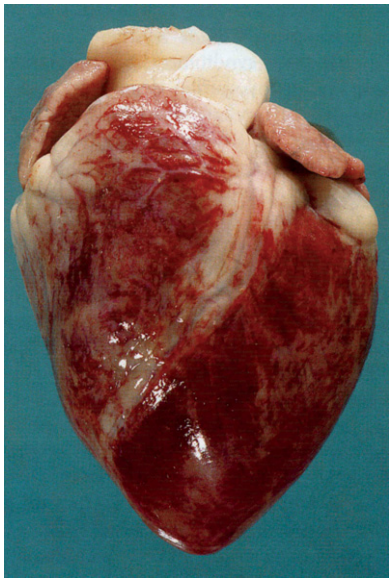

Fig 16.14 – Mulberry heat disease: petechial epicardial haemorrhage with pallor of the right ventricular myocardium. (Courtesy of the Pig Research and Development Corporation.)

Hepatic torsion of the left lateral lobe that rotates through 180–360° should easily be identified. The liver surface is usually smooth, evenly coloured, and firm on palpation, with sharp edges. Congested livers are dark purple with rounded edges. Pallor may result from anaemia, ischaemia, or fibrosis. Increased friability is present in fatty change, necrosis, and autolysis. Increased firmness indicates fibrosis. White spots that extend below the surface indicate damage by ascarids (*Ascaris suum*). *Mycobacterium avium* and *Mycobacterium bovis* can produce granulomas, 'white spots', which may be distributed throughout the hepatic parenchyma. Focal hepatic necrosis and haemorrhage can be produced by a number of diseases, including salmonellosis. Cystic structures may indicate *Cysticercus tenuicollis* (intermediate stage *Taenia hydatigena*) or *Echinococcus granulosus* in outdoor pigs. Adhesions and fibrin indicate an inflammatory process. Centrilobular pallor may indicate ischaemic necrosis caused by cardiac failure (e.g. mulberry heart disease, endocarditis). In hepatosis dietetica, the liver is grossly mottled in appearance, with yellow areas of degenerate hepatic tissue and red areas of haemorrhage. Hepatic abscesses are occasionally found. Acute aflatoxicosis causes severe hepatic necrosis.

Examination of the peritoneal cavity

The peritoneum should be examined. The position and appearance of the intestines and the stomach should be

noted (bloated, twisted, or torsion). The position of the spleen should be appraised in situ to check for splenic torsion.

Peritoneum

Check for adhesions, particulate matter from the gut, fibrin tags, and excessive amounts of peritoneal fluid. Oedema may be present in PDNS.

Removal of the gastrointestinal tract

The intestine should be ligated just below the pylorus and at the rectum before removal to prevent contamination by gut content. The intestines, stomach, and spleen are then removed from the abdominal cavity by severing the attachments.

Examination of the stomach

The stomach is isolated from the intestines by placing a ligature at the pylorus. The stomach is opened along the greater curvature to check the contents and the mucosa.

Weaners, growers, and adults

The stomach may be twisted, bloated, and sometimes ruptured if there is a gastric volvulus. The mucosa of the pars oesophagea may be ulcerated, and the stomach may contain clotted blood from a bleeding ulcer. Foreign objects (stones) may be present. *Hyostrongylus rubidus* may be present. These are small reddish worms. In oedema disease, there may be marked submucosal oedema, which can be detected by making shallow cuts across the serosal surface of the stomach (Fig. 16.15).

Suckling piglets

In normal piglets, the stomach should be full of milk. Absence of milk indicates anorexia or agalactia in the sow.

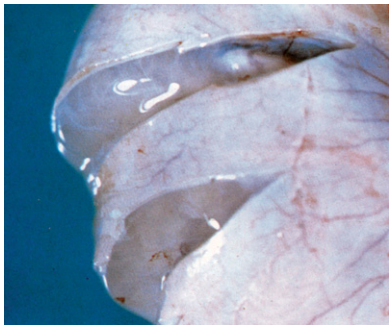

Fig 16.15 – Oedema disease: submucosal oedema of the stomach. (Courtesy of the Pig Research and Development Corporation.)

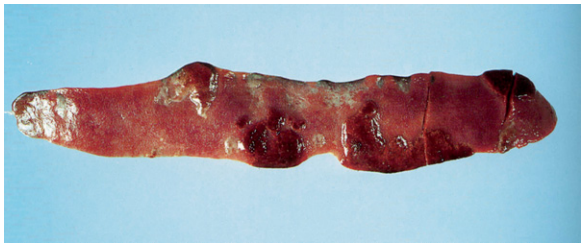

Fig 16.16 – Splenic infarcts. (Courtesy of the Pig Research and Development Corporation.)

Examination of the spleen

The spleen can be removed prior to the removal of the gastrointestinal tract by dissecting the attachments, or removed with the gastrointestinal tract.

Enlargement and congestion of the spleen will follow gastric or splenic torsion. Torsion of the spleen may occur independently to gastric torsion and rupture may follow. External trauma can result in rupture of the spleen with haemorrhaging. Splenic infarcts are seen in pigs with CSF, ASF, and PDNS, although it is often incorrectly stated that it is pathognomic for CSF (Fig. 16.16). Moderate splenomegaly may be found following a systemic infection (e.g. *Eperythrozoon suis*, erysipelas, and septicaemic salmonellosis). Abscesses are uncommon in the spleen. Nodular lymphomas in the spleen have been reported.

Examination of the intestines

The intestine should be appraised for the presence of haemorrhages, thickening, and adhesions. Torsion of the greater mesentery may be obvious by the twists in the root of the mesentery. A cut should be made into the intestines to examine the contents (watery, bloody, solid, or pasty). *Ascaris suum* worms may be present.

Examination of the small intestine

Weaners, growers, and adults

In proliferative enteritis, thickening of the terminal 30 cm of the small intestine is common, with haemorrhagic and necrotic lesions. There is segmental hyperplasia and congestion of the ileal mucosa in porcine intestinal adenomatosis and proliferative haemorrhagic enteropathy. Proliferative haemorrhagic enteropathy is commonly associated with massive intraluminal haemorrhage. In salmonellosis, the luminal contents may be watery, yellow to grey, and turbid, and they may contain fibrinonecrotic material. In some cases, blood is also present and the contents are described as dysenteric. Obstruction of the intestine may be caused by ascarid worms (Fig. 16.17), an intussusception, or an indigestible foreign material (e.g. sawdust). Scrotal hernias and umbilical

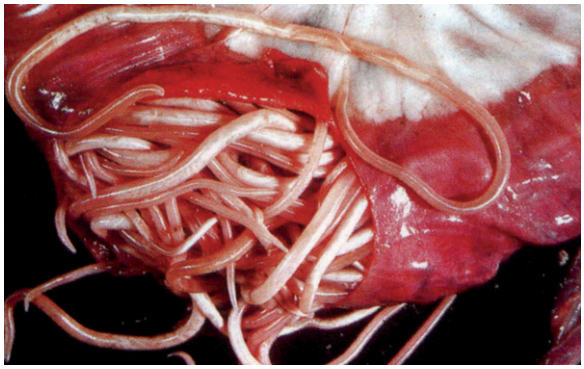

Fig 16.17 – *Ascaris suum* in the intestines. (Courtesy of the Pig Research and Development Corporation.)

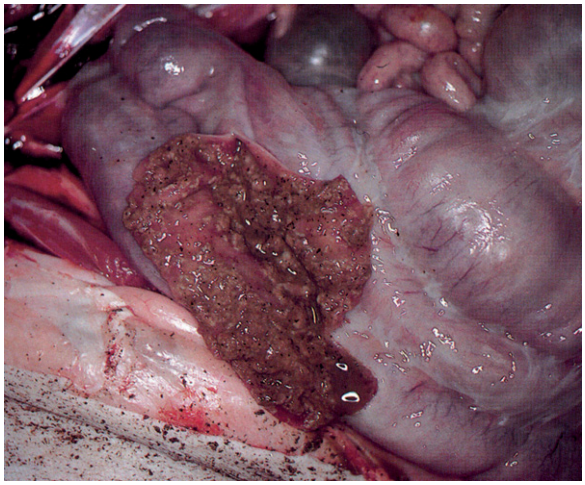

Fig 16.18 – Swine dysentery: thickened colon with haemorrhage. (Courtesy of the Pig Research and Development Corporation.)

hernias may contain incarcerated and infarcted small intestines. Rupture of the small intestines may follow ischaemic necrosis or severe external trauma. CSF and ASF can cause petechial and ecchymotic haemorrhage on the serosa and mucosa.

Suckling piglets

Transmissible gastroenteritis coronavirus and rotavirus cause villous atrophy in the jejunum and ileum. Appraisal of villous atrophy can be performed in euthanized animals by placing inverted small sections of intestine into a test tube of water for visual appraisal, or by fixing small sections of the intestine in formol saline for subsequent histopathological examination. Autolysis occurs quickly following death, and interpretation then becomes impossible if these procedures are delayed. Intensely dark red to black haemorrhagic intestines are seen in mesenteric torsion and intestinal haemorrhage syndrome. In *Clostridium perfringens* type C infection, the upper small intestines are usually affected and are thickened. The necrotic membrane produced by coccidiosis is usually found in the lower half of the small intestine in the jejunum and ileum. Impression smears and histopathology can be used to confirm coccidiosis. Enterotoxigenic colibacillosis results in flaccid distension of the small intestine, with voluminous, watery, or creamy intestinal contents; in some cases, these appear haemorrhagic. *Campylobacter coli* is associated with a pale and thickened terminal ileum with a hyperaemic mucosa. In this condition, the intestinal contents may be watery or creamy and may contain blood. Occlusion causing obstruction of the small intestine may be present in atresia ilei.

Examination of the large intestine

Weaners, growers, and adults

Proximal colonic bloat and rupture may be seen and are usually diet-related. A peritonitis may be present depending on the survival time following the rupture. Rupture due to external trauma may be seen. The contents of the large intestine should be washed out. The intestine should be checked for thickening and ulceration. Nodules containing *Oesophagostomum* larvae may be present. Mucosal inflammation and mucohaemorrhagic faeces will be present with swine dysentery. Rectal stricture will be identified as a fibrotic ring constriction at the ecto-endoderm junction of the rectum and may be associated with salmonellosis or rectal prolapse.

Deep erosions or ulcerations are seen in salmonellosis (usually *Salmonella typhimurium*), yersiniosis (*Yersinia pseudotuberculosis*), ASF, CSF, PDNS, and PMWS. These are sometimes called 'button ulcers' because of their raised edges. In salmonellosis, there is a fibrinonecrotic membrane attached to the mucosa of the large intestine. Ulceration is usually most severe in the large intestine. In oedema disease, the large intestine serosa may appear gelatinous as a result of the accumulation of oedema. This may also be seen in mulberry heart disease, ASF, and swine dysentery. Serosal haemorrhage is seen in CSF and ASF. Haemorrhagic contents are seen sometimes with bleeding gastric ulceration, proliferative haemorrhagic enteropathy, and torsions of the intestines. Blood from bleeding gastric ulcers will appear tar-like black (melaenic) in the large intestine due to partial digestion of the blood. Large intestinal haemorrhage is seen in swine dysentery, salmonellosis, CSF, and ASF. In swine dysentery, the mucosal surface is often covered with flecks of fresh blood and fibrinous necrotic material (Fig. 16.18).

Firm contents are seen in diseases causing luminal obstruction (rectal stricture, atresia ani, and megacolon). Impaction from ingesting sawdust may occur. The small *Trichuris suis* nematode may be present and associated

with diarrhoea; the large intestine is sometimes thickened, with focal haemorrhages on the mucosal surface.

Examination of the kidney and bladder

The zoonotic risk from leptospirosis should be considered when examining the urinary tract. The bladder can be cut open and the mucosa and urine examined. The kidney can be removed and cut longitudinally to examine the cortex and medulla.

The bladder wall may be thickened and inflamed, indicating cystitis. Red urine may indicate haemolytic anaemia or haematuria. CSF and ASF present with petechial and ecchymotic haemorrhages on the bladder mucosa. Pus may be present in the urine as well as the medulla of the bladder in pyelonephritis. *Stephanus dentatus* may be found in the kidneys and ureters. In PDNS, the kidneys are often swollen with pale foci and/or haemorrhages on the surface of the cortex. In PMWS, the kidneys may be pale but not haemorrhagic, with subcapsular white foci. CSF and ASF have petechial and ecchymotic haemorrhages in the renal parenchyma.

Examination of the uterus, vagina, and ovaries

These structures can be removed from the peritoneal cavity by severing the attachments.

Check the uterus for dead piglets, endometritis (Fig. 16.19), and metritis. The ovaries can be cut to investigate the status of the follicles and corpora lutea.

Examination of the musculoskeletal system

Bones, joints, and muscles

Several joints should be cut open to check for signs of infection. Samples of joint fluid and bacterial swabs can be taken. Articular cartilage should be carefully examined. Muscles can be cut at various sites to check for abnormalities. Dissection of suspected traumatized sites may provide the diagnosis.

Several joints should be cut open to check for signs of infection. Articular cartilage should be carefully examined for signs of osteochondrosis, particularly the lateral aspect of the medial femoral condyle and the lateral head of the humerus (Fig. 16.20). Suspected traumatic injuries such as humeral or femoral fractures, torn adductor muscles, and savaging should be examined in detail to obtain a diagnosis. Muscles can be cut at various sites if porcine stress syndrome is suspected to check for pale, soft musculature, although this is often difficult to interpret. Similar changes may be seen with vitamin E deficiency, particularly in the psoas muscles.

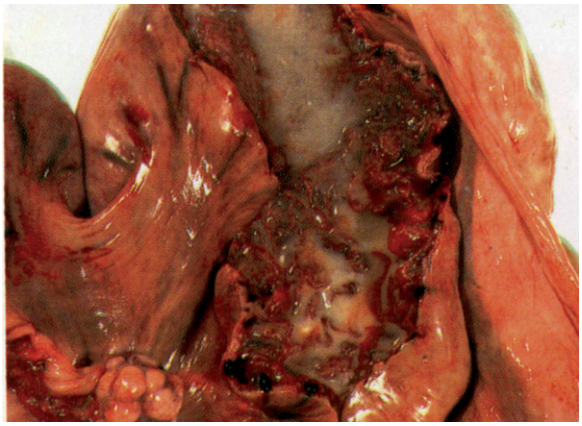

Fig 16.19 – Acute endometritis: the uterine mucosa is hyperaemic, with suppurative exudate in the lumen. (Courtesy of the Pig Research and Development Corporation.)

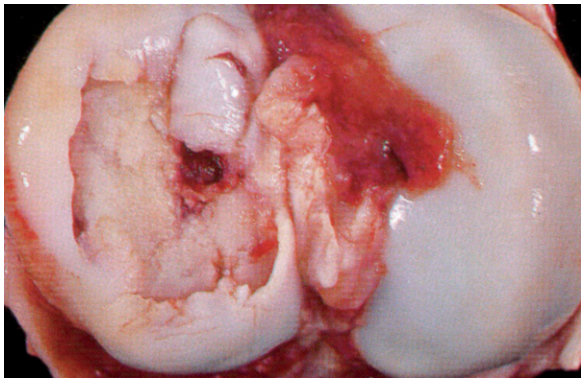

Fig 16.20 – Osteochondritis dissecans of the medial condyle of the distal femur: there is a large defect in the articular surface. (Courtesy of the Pig Research and Development Corporation.)

Examination of the skull

Removal of the brain and spinal cord in adults is best performed at a regional laboratory with appropriate facilities. The nasal turbinates can be exposed with a cross-sectional cut. In animals older than 5 months, the cut is between premolars one and two, using a hacksaw.

Suckling pigs

The head is split longitudinally using a knife. Pus on the surface of the meninges indicates a purulent meningitis. Half the brain can be fixed in 10–20 times its volume of formol saline. The other half can be submitted fresh in a sterile plastic bag. Take a swab of the meninges in suspected cases of meningitis. The nasal turbinates can be exposed with a cross-sectional cut using a hacksaw. In

swine influenza, there is inflammation of the turbinate mucosa. Atrophic rhinitis causes distortion of the turbinates, which may or may not be evident at this age. *Bordetella bronchiseptica* and *Pasteurella multocida* may be isolated if bacterial swabs are taken. Diphtheritic necrosis of the turbinates is sometimes seen with inclusion body rhinitis. A sample of turbinate scroll can be placed in formol saline. Histopathological examination will be able to identify the inclusion bodies or characterize the abnormalities.

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Haematology and blood biochemistry in the pig

Introduction

Haematology and blood biochemistry (Fig. 17.1) are under-utilized in the investigation of pig diseases. The perceived difficulty in obtaining blood samples, the cost, and the time delay in obtaining results have minimized their adoption in the past. However, the rapid growth in in-house and handheld 'animal side' analysers and the reduction in the cost of tests should stimulate increased interest in sampling selected clinical cases before resorting to post-mortem examinations.

Haematology

Diseases affecting the blood

The following diseases affect the blood in pigs.
In unweaned pigs:
- isoimmune thrombocytopenia,
- isoimmune haemolytic anaemia,
- eperythrozoonosis,
- iron deficiency anaemia, and
- umbilical haemorrhage.

In weaned pigs:
- coagulopathies (including warfarin),
- hereditary lymphosarcoma,
- African swine fever,
- classical swine fever,
- eperythrozoonosis,
- von Willebrand's disease, and
- porcine dermatitis and nephropathy syndrome.

Relative polycythaemia may occur in pigs with dehydration. Anaemia is a common finding. Isoimmune thrombocytopenia and haemolytic anaemia are rare. Haemorrhagic and haemolytic anaemias show a regenerative response and elevated bilirubin levels. Iron deficiency produces a non-regenerative, normocytic–microcytic, normochromic to microchromic anaemia.

Diseases causing anaemia

The following diseases can cause anaemia in pigs.
Haemorrhagic:
- gastric ulceration,
- proliferative haemorrhagic enteropathy,
- trauma with blood loss,
- umbilical haemorrhage,
- coagulopathies,
- *Hyostrongylus rubidus*, and
- trichuriasis.

Haemolytic anaemia:
- eperythrozoonosis and
- isoimmune haemolytic anaemia.

Anaemia due to defective erythropoiesis:
- iron deficiency and
- lymphosarcoma.

Leucocytosis and leucopenia

Leucocytosis (an increase in the total number of leucocytes) occurs in inflammatory disease, stress (e.g. transport, parturition, and handling), and lymphoid or myeloid leukaemia.

In stress leucograms, there is a mature neutrophilia, an absolute lymphopenia, and a reduction in the number of eosinophils.

Inflammation is characterized by an absolute neutrophilia with some immature neutrophils (left shift), with a relative reduction in the number of lymphocytes. Toxic changes may be seen in neutrophils. Dramatic left shifts are most often seen in suppurative conditions. Viral infections – such as African swine fever, classical swine fever, and porcine reproductive and respiratory syndrome virus – and severe overwhelming bacterial infections produce leucopenias and neutropenias.

Platelets

Large reductions in the numbers of platelets occur in classical swine fever and African swine fever infections. Mild reduction occurs in porcine reproductive and respiratory syndrome virus infections. Isoimmune thrombocytopenia may occur in piglets born to sows sensitized to the platelets of their offspring following the ingestion of colostrum. Disseminated intravascular

Fig 17.1 – Haematology and serum biochemistry may indicate the type and severity of the pathophysiological process in porcine disease.

coagulation associated with septicaemia will depress the platelet count. Increases in platelet numbers are found in stress. Reduction in the platelet counts will increase the risk of haemorrhage. (See *Coagulopathies* section below.)

Plasma proteins

Hypoproteinaemia may occur in exudative epidermitis, pars oesophageal ulceration, protein-losing enteropathies, and glomerular disease. Inflammatory responses or dehydration will result in an increase in plasma protein levels. Fibrinogen will be elevated in acute inflammatory conditions.

Coagulopathies

Clotting defects are characterized by multiple haemorrhages. These may include haemorrhages in the subcutis, gastrointestinal tract, lymph nodes, and aural haematomata. Thrombocytopenia, von Willebrand's disease anticoagulant rodenticides, some mycotoxins, and severe liver disease can lead to a coagulopathy.

Clotting and toxicological tests may be helpful in differentiating some of these conditions.

Haematology and blood biochemistry by system

Stomach

Blood loss anaemia (regenerative) and hypoproteinaemia may occur due to ulceration of the pars oesophagea.

Intestines

Severe diarrhoea may result in a metabolic acidosis, hypochloraemia, hyponatraemia, and hyperkalaemia. Hypoglycaemia may occur in neonates with diarrhoea. Loss of plasma proteins leading to hypoproteinaemia may occur when there is damage to the intestinal mucosa. Dehydration may cause a relative polycythaemia and an increase in plasma proteins. Acute necrotizing intestinal lesions will produce a severe leucopenia.

Liver

With hepatocyte damage, the enzymes aspartate aminotransferase (AST), sorbitol dehydrogenase, and glutamate dehydrogenase will be elevated.

Disease of the biliary system (rare in pigs) will result in elevated alkaline phosphatase and γ-glutamyl transpeptidase. In chronic hepatic disease, there is a reduction in albumin synthesis. Increased bilirubin levels may indicate an obstruction of the biliary outflow or an increased rate of haemolysis. In severe hepatic disease, there may be a reduction in the production of clotting factors, with coagulopathy and haemorrhage. A transient eosinophilia may accompany ascarid larval migrations in the liver. The liver is used to assay copper and selenium, and is used in toxicological investigations.

Cardiovascular system

Myocardial necrosis causes increases in creatinine kinase, AST, and lactate dehydrogenase (LDH). Skeletal muscle damage also causes a rise in creatinine kinase, AST, LDH, and creatinine. Liver damage also causes a rise in AST levels. Isoenzyme analysis of LDH to target the heart is rarely undertaken. Measurement of plasma and liver selenium and vitamin E levels may assist in investigations of deficiency. Plasma glutathione peroxidase activity in erythrocytes can also be used to assess historical (4–6 weeks prior to sampling) selenium levels. Vegetative endocarditis may be accompanied by haematological changes indicating inflammation.

The lungs

Severe pulmonary disease will result in a respiratory acidosis. Eosinophilia may accompany infections with *Metastrongylus*. Fulminating pneumonia will produce a leucopenia, but more chronic low-grade infections result in a leucocytosis with a relative neutrophilia (left shift).

Bones

Serum alkaline phosphatase will be elevated in rickets and fibrous osteodystrophy. However, serum alkaline phosphatase is high is young rapidly growing animals, so interpretation in immature animals needs caution.

Skeletal muscle

Creatinine kinase is found in skeletal muscle cells and is elevated when they are damaged. It is also found in the myocardial cells. This enzyme is elevated in nutritional myopathy and porcine stress syndrome. AST and LDH are also found in myocardial and skeletal muscle. Creatinine kinase is usually used to screen for skeletal muscle damage. Eosinophilia may be present in trichinosis. Stress leucograms occur in porcine stress syndrome.

Skin

Zinc levels can be determined in serum if parakeratosis is suspected. In non-specific coagulopathies with cutaneous haemorrhages, coagulation assays may be able to characterize the problem in more detail.

Urinary tract

Increases in serum urea and creatinine concentrations indicate prerenal (e.g. cardiovascular disease), renal (e.g. severe pyelonephritis), or urinary outflow (obstruction) dysfunction. Aqueous humour urea levels correlate relatively well with serum levels up to 2 h after death. Hypoproteinaemia may result from a nephropathy with elevated protein levels in the urine. Leucocytosis occurs in pyelonephritis, and non-regenerative anaemia may accompany severe chronic renal disease. Free-flow urine samples can sometimes be obtained by making the animal stand. A red urine may indicate a haematuria (e.g. cystitis) or a haemoglobinuria (haemolytic anaemia). The erythrocytes from a haematuria will sediment on standing or centrifugation. Red-brown rusty-coloured urine is found in myoglobinuria. Turbid flocculent urine indicates a pyuria (pyelonephritis). If a stable foam is produced following shaking, this may indicate a proteinuria, which will produce an elevation in specific gravity (mean, 1.010; range, 1.001–1.030). Examination of the sediment following standing under the microscope at ×10 may identify a crystalluria (usually phosphate). The kidney is one of the organs of choice for toxicological analysis (e.g. copper, selenium, zinc, arsenic, cadmium, and lead.)

Tables of normal values

The haematological and serum biochemical values for the pig are shown in Tables 17.1–17.4. Reference ranges supplied by individual laboratories should be consulted when interpreting results from these laboratories.

Table 17.1 Haemotological values for pigs: conventional units

Parameter	Value
Haemoglobin (g/dL)	10.0–16.0
Haematocrit (packed cell volume) (%)	32.0–50.0
Red blood cells ($\times 10^6/\mu L$)	5.0–8.0
Mean corpuscular volume (fL)	50.0–68.0
Mean corpuscular haemoglobin (pg)	17.0–21.0
Mean corpuscular haemoglobin concentration (g/dL)	30.0–34.0
Thrombocytes ($\times 10^3/\mu L$)	320–520
White blood cells (per μL)	11 000–22 000
Neutrophils (mature) (per μL)	3080–10 450
Neutrophils (band cells) (per μL)	0–880
Lymphocytes (per μL)	4290–13 640
Monocytes (per μL)	200–2200
Eosinophils(per μL)	55–2420
Fibrinogen(mg/μL)	–

(From Radostits OM, Gay CC, Blood DC et al. 2000 Veterinary medicine, 9th edn. Saunders, London, pp 1819–1822.)

Table 17.2 Haematological values for pigs: Système International units

Parameter	Value
Haemoglobin (g/L)	100–160
Haematocrit (packed cell volume)	0.32–0.50
Red blood cells ($\times 10^{12}$/L)	6.8–12.9
Mean corpuscular volume (fL)	50–68
Mean corpuscular haemoglobin (pg)	17.0–21.0
Mean corpuscular haemoglobin concentration (g/L)	303–340
Thrombocytes ($\times 10^9$/L)	320–520
White blood cells ($\times 10^9$/L)	11.0–22.0
Neutrophils (mature) ($\times 10^9$/L)	3.1–10.5
Neutrophils (band cells) ($\times 10^9$/L)	0–0.1
Lymphocytes ($\times 10^9$/L)	4.3–13.0
Monocytes ($\times 10^9$/L)	0.2–2.2
Eosinophils ($\times 10^9$/L)	0.05–2.4
Fibrinogen (g/L)	–

(From Radostits OM, Gay CC, Blood DC et al. 2000 Veterinary medicine, 9th edn. Saunders, London, pp 1819–1822.)

Table 17.3 Blood biochemical values for pigs: conventional units

Parameter	Value
Electrolytes	
Sodium (mEq/L)	140–150
Potassium (mEq/L)	4.7–7.1
Chloride (mEq/L)	95–103
Acid : base status	
pH (venous)	–
Pco_2 (venous) (mmHg)	–
Bicarbonate (mEq/L)	18–27
Total carbon dioxide (mEq/L)	17–26
Anion gap (mEq/L)	10–25
Minerals	
Calcium, total (mg/dL)	7.1–11.6
Calcium, ionized (mg/dL)	3.5–5.8
Phosphorus (mg/dL)	5.3–9.6
Magnesium (mg/dL)	1.1–1.5
Iron (μg/dL)	73–140
Iron-binding capacity (μg/dL)	270–557
Renal function	
Urea nitrogen (mg/dL)	10–30
Creatinine (mg/dL)	1.0–2.7
Liver function	
Total bilirubin (mg/dL)	0–10
Direct (conjugated) bilirubin (mg/dL)	0–0.3
Bile acids (mg/mL)	–
Metabolites	
Ammonia (μg/dL)	–
Cholesterol (mg/dL)	28–48
Free fatty acids (mg/L)	–
Glucose (mg/dL)	85–150
Ketones	
Acetoacetate (mg/dL)	–
Acetone (mg/dL)	–
3-Hydroxybutyrate (mg/dL)	–
Lactate (mg/dL)	–
Triglyceride (mg/dL)	–
Hormones	
Cortisol (μg/dL)	2.6–3.3
Thyroxine (μg/dL)	–
Enzymes	
Alanine aminotransferase (units/L)	31–58
Alkaline phosphatase (units/L)	120–400
Amylase (units/L)	–
Aspartate aminotransferase (units/L)	32–84
Creatine kinase (units/L)	0–500
Glutamic oxaloacetic transaminase (see aspartate aminotransferase)	–
Glutamic pyruvate transaminase (see alanine aminotransferase)	–
γ-Glutamyltransferase (units/L)	10–52
Isocitrate dehydrogenase (units/L)	–
Lactate dehydrogenase (units/L)	380–630
Sorbitol dehydrogenase (units/L)	1–5.8
Protein	
Total protein (g/dl)	3.5–6
Albumin (g/dL)	1.9–2.4

(From Radostits OM, Gay CC, Blood DC et al. 2000 Veterinary medicine, 9th edn. Saunders, London, pp 1819–1822.)

Table 17.4 Serum biochemical values for the pig: Système International units

Parameter	Value
Electrolytes	
Sodium (mmol/L)	140–150
Potassium (mmol/L)	4.7–7.1
Chloride (mmol/L)	94–103
Osmolality (mmol/kg)	–
Acid : base status	
pH (venous)	–
Pco_2 (venous) (mmHg)	–
Bicarbonate (mmol/L)	18–27
Total carbon dioxide (mmol/L)	17–26
Minerals	
Calcium, total (mmol/L)	1.78–2.90
Calcium, ionized (mmol/L)	0.9–1.4
Phosphorus (mmol/L)	1.30–3.55
Magnesium (mmol/L)	0.78–1.60
Iron (μmol/L)	–
Iron-binding capacity (μmol/L)	48–100
Renal function	
Urea nitrogen (mmol/L)	3–8.5
Creatinine (μmol/L)	90–240
Liver function	
Total bilirubin (μmol/L)	0–17.1
Direct (conjugated) bilirubin (μmol/L)	0–5.1
Bile acids (μmol/L)	–
Metabolites	
Ammonia (μmol/L)	7.6–63.4
Cholesterol (mmol/L)	3.05–3.10
Glucose (mmol/L)	3.6–5.3
Ketones	
Acetoacetate (mmol/L)	–
Acetone (mmol/L)	–
3-Hydroxybutyrate (mmol/L)	–
Lactate (mmol/L)	–
Triglyceride (mmol/L)	–
Hormones	
Cortisol (nmol/L)	75–88
Thyroxine (nmol/L)	–
Triiodothyronine (nmol/L)	–
Enzymes	
Alanine aminotransferase (units/L)	31–58
Alkaline phosphatase (units/L)	120–400
Amylase (units/L)	–
Aspartate aminotransferase (units/L)	32–84
Creatine kinase (units/L)	0–500
Glutamic oxaloacetic transaminase (see aspartate aminotransferase)	–
Glutamic pyruvate transaminase (see alanine aminotransferase)	–
γ-Glutamyl transferase (units/L)	10–60
Isocitrate dehydrogenase (units/L)	
Lactate dehydrogenase (units/L)	380–630
Sorbitol dehydrogenase (units/L)	1–5.8
Protein	
Total protein (g/L)	19–24
Albumin (g/L)	19–24

(From Radostits OM, Gay CC, Blood DC et al. 2000 Veterinary medicine, 9th edn. Saunders, London, pp 1819–1822.)

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18

Differential diagnosis

Introduction

The process of diagnosis is an important but often complex function. This chapter is intended as a guide to differential diagnosis. Detail regarding the conditions mentioned can be found in the other chapters of this book. A knowledge and understanding of the conditions that can affect pigs is vital to avoid misinterpretation or over-interpretation of clinical information.

Outcomes of the clinical examination

The identification of clinical abnormalities and disease-associated risk factors are outcomes of the clinical examination. From these findings, the following may be derived:

- the most likely cause,
- the organs or systems involved,
- the location of the lesion,
- the type of lesion present,
- the pathophysiological processes occurring,
- the severity of the disease,
- the morbidity and mortality of the disease, and
- the epidemiology of the outbreak.

Using this information, the clinical abnormalities, and the relative prevalences of disease, an appropriate list of differential diagnoses can be generated.

Prevalence

Consideration of the relative prevalence of the conditions on the differential diagnosis list will improve the selection of further investigations. Figures 18.1–18.11 show the diagnoses of the samples submitted to the Veterinary Laboratories Agency in the period 1996–2003.

Mortality rates and intervention levels

Mortality rates can be used as a benchmark for further investigation and intervention (Table 18.1).

Web sites

There are three web sites that may assist the reader in differential diagnosis. These can be found by entering the name of the web site into an Internet search engine or by using the web site addresses given in the references at the end of the chapter.

Consultant

Consultant is a veterinary diagnostic support system used to suggest possible causes for clinical signs and to provide a brief synopsis of the cause, including:

- a general description of the disease,
- species affected,
- the signs observed reported in the published literature, and
- a list of recent literature references.

Dogs, cats, cattle, sheep, pigs, goats, birds, and horses are the species represented.

Consultant can be used by searching for information by diagnosis, and by searching of disease by sign(s). In the search by diagnosis, a search can be made for a particular condition using a keyword. Matches to the keyword are then displayed. Information regarding the nominated condition can then be obtained.

In the search by sign(s), a selection can be made of one or more signs in a nominated species and the search will then return all the diagnoses that contain all the signs (set of signs) that were entered. This represents a differential diagnosis list for the signs entered. The differential diagnoses are not ranked. It is important that the signs that are entered are accurate. An example is shown in Box 18.1.

Case Diagnosis: Swine Dysentery

Signs entered:

- diarrhoea
- melaena or occult blood in faeces, stools
- bloody stools, faeces, haematochezia
- underweight, poor condition, thin, emaciated, unthriftiness, ill thrift
- weight loss

Possible diagnoses that have all the signs entered (9):

- African swine fever
- anticoagulant, rodenticide, toxicity
- arsenic toxicity
- hog cholera, classical swine fever
- liver, hepatic disease
- proliferative enteropathy, enteritis, ileitis, *Lawsonia intracellularis* in pigs
- stachybotryotoxicosis
- swine dysentery, *Brachyspira hyodysenteriae*
- systemic candidiasis

Box 18.1 – Consultant: possible diagnosis for a set of signs consistent with swine dysentery.

The Pigsite

This site provides diagnostic algorithms for pig diseases and reproductive disorders.

An example of the diseases algorithm is shown in Box 18.2. The clinical data are entered by selecting clinical signs and age-related information on a menu. Possible differential diagnoses are listed, with an agreement rating between the information entered and the disease. There are links to further information for each disease.

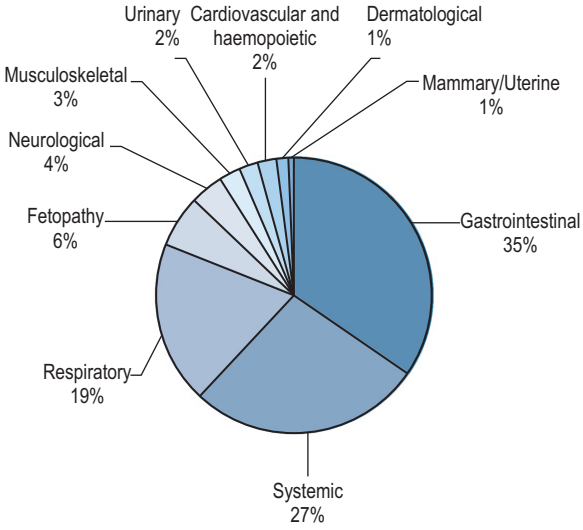

Fig 18.1 – Samples submitted to the Veterinary Laboratories Agency (1996–2003) that belonged to defined disease categories (n=18 255).

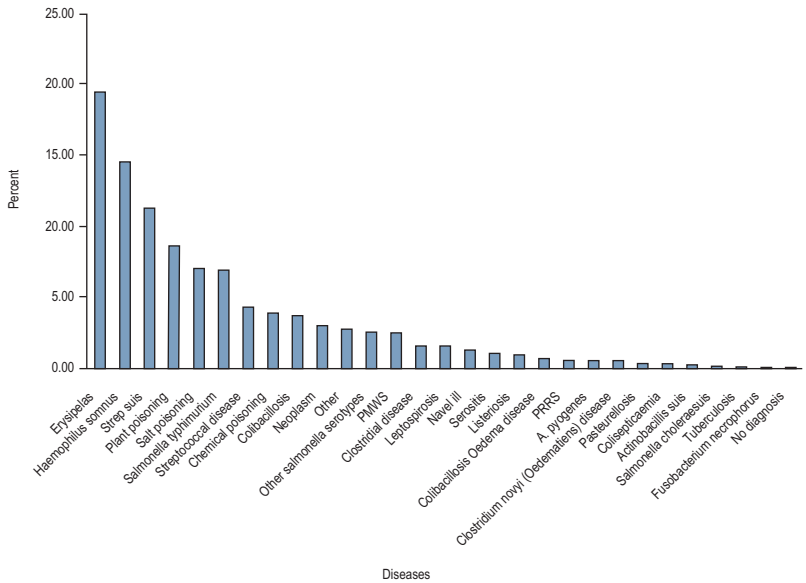

Fig 18.2 – Systemic diseases: each disease as a percentage of the total number of diagnoses in this category (Veterinary Laboratories Agency, 1996–2003). PMWS, postweaning multisystemic wasting syndrome; PRRS, porcine reproductive and respiratory syndrome.

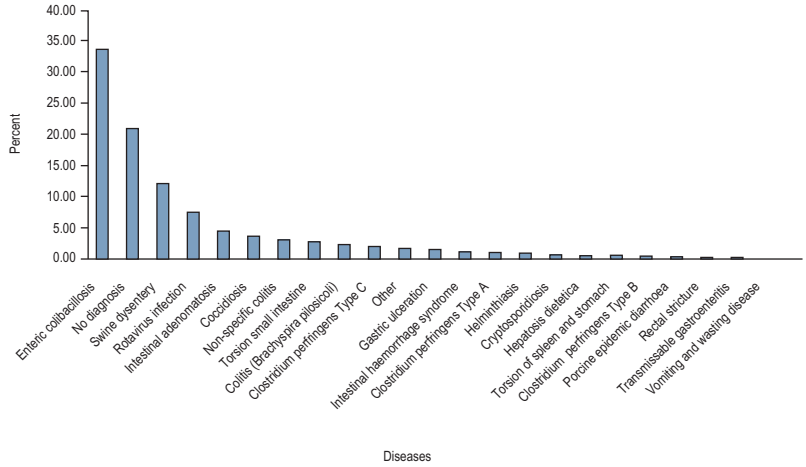

Fig 18.3 – Gastrointestinal diseases: each disease as a percentage of the total number of diagnoses in this category (Veterinary Laboratories Agency, 1996–2003).

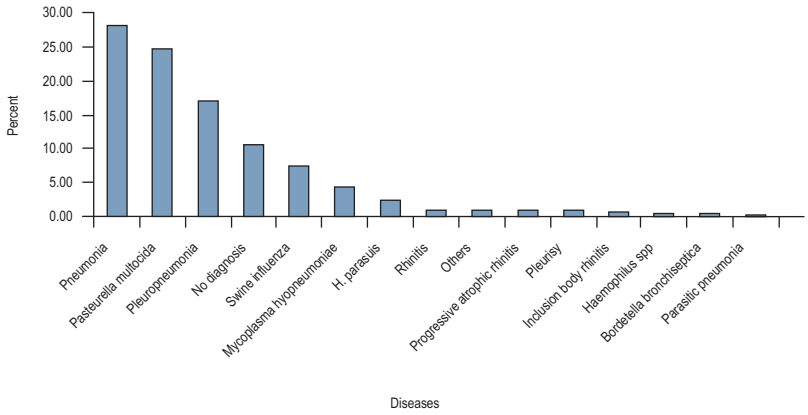

Fig 18.4 – Respiratory diseases: each disease as a percentage of the total number of diagnoses in this category (Veterinary Laboratories Agency, 1996–2003).

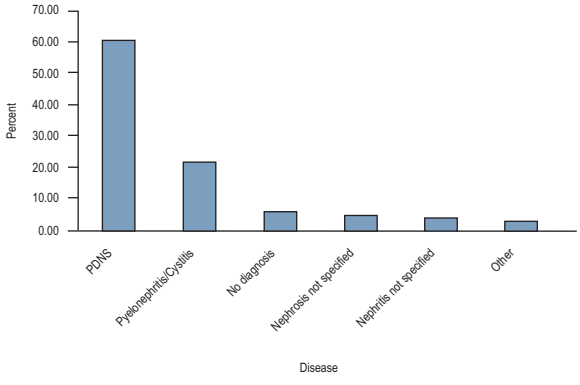

Fig 18.5 – Urinary system: each disease as a percentage of the total number of diagnoses in this category (Veterinary Laboratories Agency, 1996–2003). PDNS, porcine dermatitis and nephropathy syndrome.

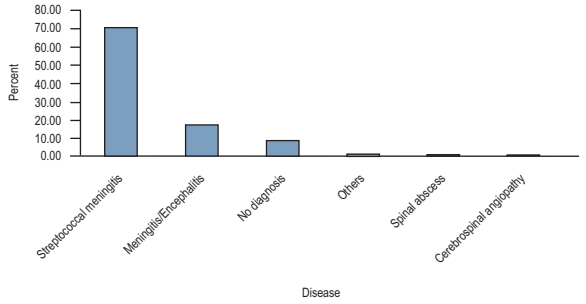

Fig 18.6 – Neurological system, including the special senses: each disease as a percentage of the total number of diagnoses in this category (Veterinary Laboratories Agency, 1996–2003).

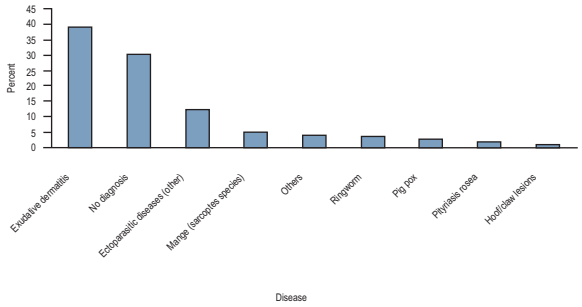

Fig 18.7 – Dermatological diseases: each disease as a percentage of the total number of diagnoses in this category (Veterinary Laboratories Agency, 1996–2003).

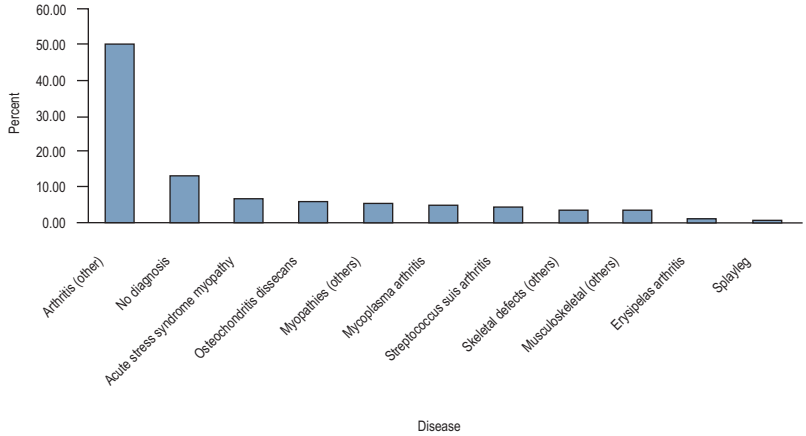

Fig 18.8 – Musculoskeletal system: each disease as a percentage of the total number of diagnoses in this category (Veterinary Laboratories Agency, 1996–2003).

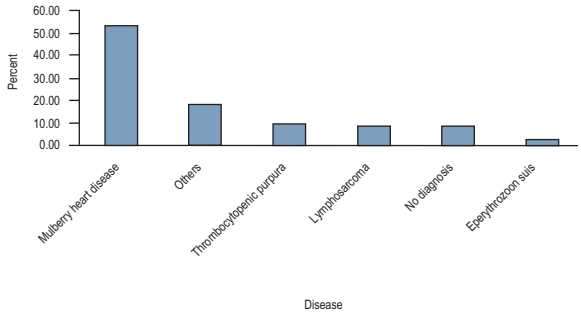

Fig 18.9 – Cardiovascular and haemopoietic system: each disease as a percentage of the total number of diagnoses in this category (Veterinary Laboratories Agency, 1996–2003).

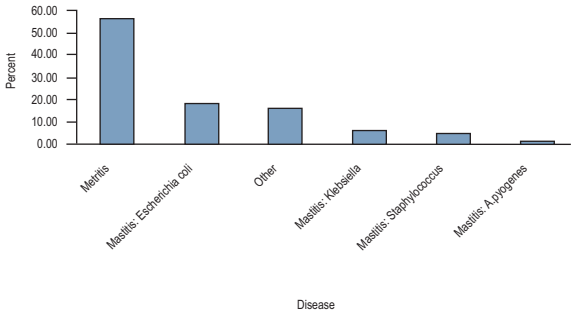

Fig 18.11 – Mammary gland and uterus: percentage of all the diseases in this category (Veterinary Laboratories Agency, 1996–2003).

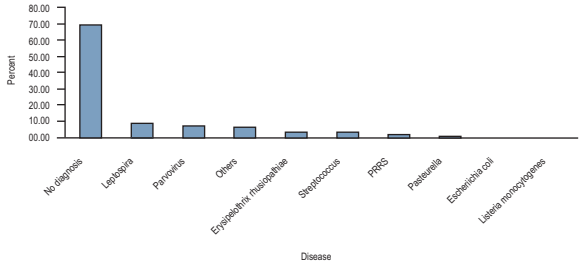

Fig 18.10 – Fetopathy: each disease as a percentage of the total number of diagnoses in this category (Veterinary Laboratories Agency, 1996–2003). PRRS, porcine reproductive and respiratory syndrome.

Table 18.1 Mortality rates and intervention levels

Age group	Intervention (%)	Target (%)
Newborn	>8	Stillbirths <6 Mummified <1
Suckling piglets	>14	<10
Weaners	>3	<2.0
Growers and finishers	>3	<1.5
Breeders		
>200 sows	>5	<5
<200 sows	>3	<3

Case Diagnosis: Porcine Dermatitis and Nephropathy Syndrome (PDNS)

Signs:

- pigs affected >10%
- skin lesions (black, brown, red)
- haemorrhage
- inappetance
- lymph nodes enlarged
- mortality >2% increase
- poor growth
- weaners – weaned 15–56 days

For data entry, the signs are entered using the menus below.

1. *Select which ages of pig are affected*
   Select all ages (alternatively, select individual ages below)
   - Sows/gilts/boars
   - Piglets: age 0–7 days
   - Piglets: age 8 days to weaning
   - Weaners: weaned to 14 days
   - Weaners: weaned 15–56 days
   - Finishers/growers
2. *Number of pigs affected (select one)*
   - <10% affected
   - >10% affected
3. *Do pigs have a high temperature/fever?*
   Yes/no
4. *Are the pigs off their food?*
   Yes/no
5. *Consider your mortality rate*
   - Increase <2%
   - Increase >2%

   Are some pigs dying suddenly? (Good pigs found dead.)
6. *Select the symptoms you see from the list below:*
   Locomotion/nervous signs
   - Fits/convulsions
   - Head on one side
   - Incoordination
   - Lameness/stiffness/arthritis
   - Paralysis/'dog sitting' position
   - Poor viable/hypothermia/splay legs
   - Trembling/shaking

   Excretion/discharge signs
   - Blood in (normal) faeces
   - Blood in semen
   - Blood/pus in urine
   - Conjunctivitis (discharges from eye)
   - Constipation
   - Diarrhoea: general
   - Diarrhoea: watery
   - Diarrhoea: with blood/mucus
   - Mucus/pus or blood from nose
   - Mucus/pus in urine
   - Mucus/pus or blood from vulva
   - Salivation
   - Urine chalky/abnormal colour
   - Vomiting

   Skin/bodily signs
   - Abscesses
   - Abdomen bloated or distended
   - Anaemia/pale pigs
   - Blisters/vesicles
   - Blood on/in skin, vulva, rectum
   - Dehydration
   - Gangrene/necrosis (rotting skin)
   - Haemorrhage
   - Hairy pigs
   - Lymph nodes enlarged
   - Poor growth/wasting/starvation
   - Skin: black, brown, or red lesions
   - Skin: raised patches
   - Skin: blue
   - Skin: greasy brown
   - Skin: jaundice (yellow)
   - Skin: ulcerated/inflamed/dermatitis
   - Scratching/rubbing/irritation of the skin
   - Shivering
   - Swellings/oedema
   - Thin sows

   Reproductive signs
   - Abnormal oestrus
   - Abortion/not in pig
   - Anoestrus (lack of heat)
   - Farrowing slow
   - Loss/lack of libido
   - Low birth weight
   - Mastitis
   - Mummified pigs/fetal death
   - No milk or colostrum
   - Not sucking
   - Premature farrowing
   - Reproductive failure (general)
   - Small litter sizes
   - Stillbirths
   - Swollen testicles

   Respiratory and other signs
   - Blindness
   - Coughing
   - Fractures
   - Meningitis/jerky eye movements
   - Nose deformed
   - Pain
   - Pneumonia/rapid breathing
   - Sneezing
   - Bottom of form

Results

A score of less than 70% is considered a poor match. The diseases that gave a score above 70% are shown below.

- PDNS (100% match)
- Postweaning multisystemic wasting syndrome (PMWS) (71% match)
- Salmonellosis (71% match)

Box 18.2 – Pigsite: signs entered, sign menus and possible diagnoses for a case of porcine dermatitis and nephropathy syndrome.

The Iowa State University Swine Site

This site provides lists and pictures of diseases by age, system, and signs.

Conditions by age and system

Conditions by age

Many diseases in pigs are age-related. A knowledge of the relationship between the age of an animal and the diseases to which it is susceptible is essential for differential diagnosis. Table 18.2 and Figure 18.12 show conditions in relation to age.

Conditions by system

The conditions by system are described in the following chapters.

- *Diseases of the musculoskeletal system* (Ch. 3)
- *Diseases of the respiratory system* (Ch. 4)
- *Diseases of the gastrointestinal system* (Ch. 5)
- *Diseases of the skin* (Ch. 6)
- *Diseases of the nervous system* (Ch. 7)
- *Diseases of the cardiovascular, haemopoietic, and lymphatic systems* (Ch. 8)
- *Diseases of the urogenital system and the udder* (Ch. 9)
- *Obstetrics and reproduction in pigs* (Ch. 10)
- *Polysystemic diseases* (Ch. 11)

Key information for all investigations

Attempts should be made to obtain the following information.

- Age of animal affected
- Prevalence, morbidity, and mortality
- Groups affected
- Origin of affected pigs (homebred or purchased?)
- Date of purchase
- Health status of unit of origin
- When the disease was first detected
- The signs that have been observed
- The progression of the disease
- Severity of disease
- The treatments that have been tried and the outcome
- The current preventive measures
- Identification of clinical abnormalities and disease-associated risk factors
- Post-mortem results, abattoir feedback reports, and test results

Investigative approaches by syndrome and signs

This section provides investigative approaches for the following syndromes.

- Delayed puberty
- Delayed weaning to mating interval
- High returns to service
- Reduced farrowing rates
- Failure to maintain pregnancy
- Low litter size
- Boar infertility
- Poor milk production
- High replacement rate
- Preweaning scours
- Preweaning deaths
- Weaner scours
- Weaner deaths (up to 10 weeks of age)
- Grower and finisher scours
- Poor growth rates in growers and finishers
- Grower and finisher sudden death
- Coughing pigs
- Nervous signs
- Skin diseases
- Lameness
- Sudden death in adults

Delayed puberty

- Is (are) the affected animal(s) too young or too light?
  Onset of puberty is usually 23–29 weeks, with pigs weighing 120 kg plus.
- Is the diet adequate to achieve the target weight?
- Is it summertime with hot weather?
  Seasonal infertility may reduce feed intake.
- Is it a breed that is late maturing?
  Durocs are an example.
- Have they had sufficient exposure to an appropriate boar?
  The boar should be 12 months plus with 30 min of exposure/day.
- Is the group size optimal?
  Groups of 6–8 gilts are better than larger groups.

Delayed weaning to mating interval

Lactation inhibits reproductive cycling. This parameter involves the weaning to oestrus interval and the ability to detect oestrus. Animals with a weaning to oestrus interval of 6 or 10 days have 2.33 and 2.27 litters/year, respectively. The target for commercial herds is to have 90% of sows mated within 7 days of weaning. This delay will result in an increase in the parameter *non-productive days*, which excludes gestation, lactation, and a 6-day weaning interval.

Table 18.2 Disorders of the pig by age

Condition	Age (weeks)													Adults		
	2	4	6	8	10	12	14	16	18	20	22	24	26	Gilt	Sow	Boar
Actinobacillus pleuropneumoniae																
Abrasions																
Abscesses																
Actinobacillus suis																
Ascariasis																
Atrophic rhinitis: sneezing																
Atrophic rhinitis: twisted																
Bacterial arthritis																
Bordetella bronchiseptica																
Borrelia granuloma																
Bursitis																
Carpal abrasion																
Swine fever																
Clostridium difficile																
Clostridium perfringens																
Coccidiosis																
Colitis																
Congenital tremor																
Cystitis																
Dermatosis vegetans																
Escherichia coli																
Epidemic diarrhoea																
Epiphysiolysis																
Epitheliogenesis imperfecta																
Erysipelas: skin																
Erysipelas: arthritis																
Facial necrosis																
Flaky skin																
Foot and mouth																
Gastric ulceration																
Glasser's disease																
Greasy pig disease																
Haemorrhagic bowel																
Hernia																
Ileitis																
Insect bites																
Joint ill																
Leptospirosis																
Meningitis																
Mulberry heart																
Mycoplasma hyosynoviae																
Mycoplasma pneumonia																
Overgrown feet																
Parakeratosis																
Parvovirus																

(continued)

Table 18.2 Disorders of the pig by age—cont'd

Condition	Age (weeks)													Adults		
	2	4	6	8	10	12	14	16	18	20	22	24	26	Gilt	Sow	Boar
Pasteurellosis						■	■	■	■	■						
Porcine dermatitis and nephropathy syndrome					■	■	■	■								
Pityriasis rosea				■	■											
Postweaning multisystemic wasting syndrome				■	■	■										
Prolapse							■	■	■	■	■	■	■	■	■	
Porcine reproductive and respiratory syndrome virus	■	■	■	■	■	■	■	■	■	■	■	■	■	■	■	■
Pseudorabies		■	■	■	■	■	■	■								
Pyelonephritis															■	
Ringworm							■	■	■	■	■	■	■	■	■	■
Rotavirus	■															
Salmonellosis			■	■	■	■	■	■	■	■	■					
Sarcoptic mange			■	■	■	■	■	■	■	■	■	■	■	■	■	■
Shoulder sores															■	
Spirochaetal colitis						■	■	■	■							
Splay leg	■															
Streptococcus arthritis		■														
Sunburn																
Swine dysentery						■	■	■	■							
Swine influenza				■	■	■	■	■	■	■	■	■	■	■	■	■
Swine pox		■	■	■												
Tail biting vices			■					■	■	■	■	■	■			
Transmissible gastroenteritis	■	■	■	■												
Thrombocytopenia	■															
Trauma	■	■	■	■	■	■	■	■	■	■	■	■	■	■	■	■
Trichuriasis			■	■	■	■	■	■	■	■	■	■	■			

Key: white, rare; light shading, possible; dark shading, common.
(Courtesy of Dr John Carr, Iowa State University, Ames.)

- Is the herd composed of a large number of young animals? (High culling rate?)
  First-litter sows have longer weaning to oestrus intervals.
- Does the herd have an early weaning policy?
  Early weaning (3 weeks) increases the weaning to mating interval by up to 18 days.
- Are the sows in a low condition score at weaning?
  A low condition score will increase the weaning to mating interval. This may result from either sows being over-fat at the start of lactation, with a depressed appetite or underfeeding, or providing a low-quality diet. High temperatures can also reduce intake.
- Are the staff adequately trained and diligent in detecting oestrus?
- Is there optimal boar contact after weaning?

High returns to service

A high rate of return to service at 18–24 days after mating means a low conception rate. The parameter *non-productive days*, which excludes gestation, lactation, and a 6-day weaning interval, will be increased.

- Is management at fault?
  Ensure that animals are checked twice a day for signs of oestrus. Serve gilts at first detection of oestrus; mate sows 12 h after this. Double mate.
- Is the problem confined to a certain age group?
  A good performance in one age group may mask a poor performance by another group. Young sows in poor condition are often a problem.
- Is the mating supervised and are conditions suitable?
  The boar may be aggressive or the conditions for mounting may be slippery.

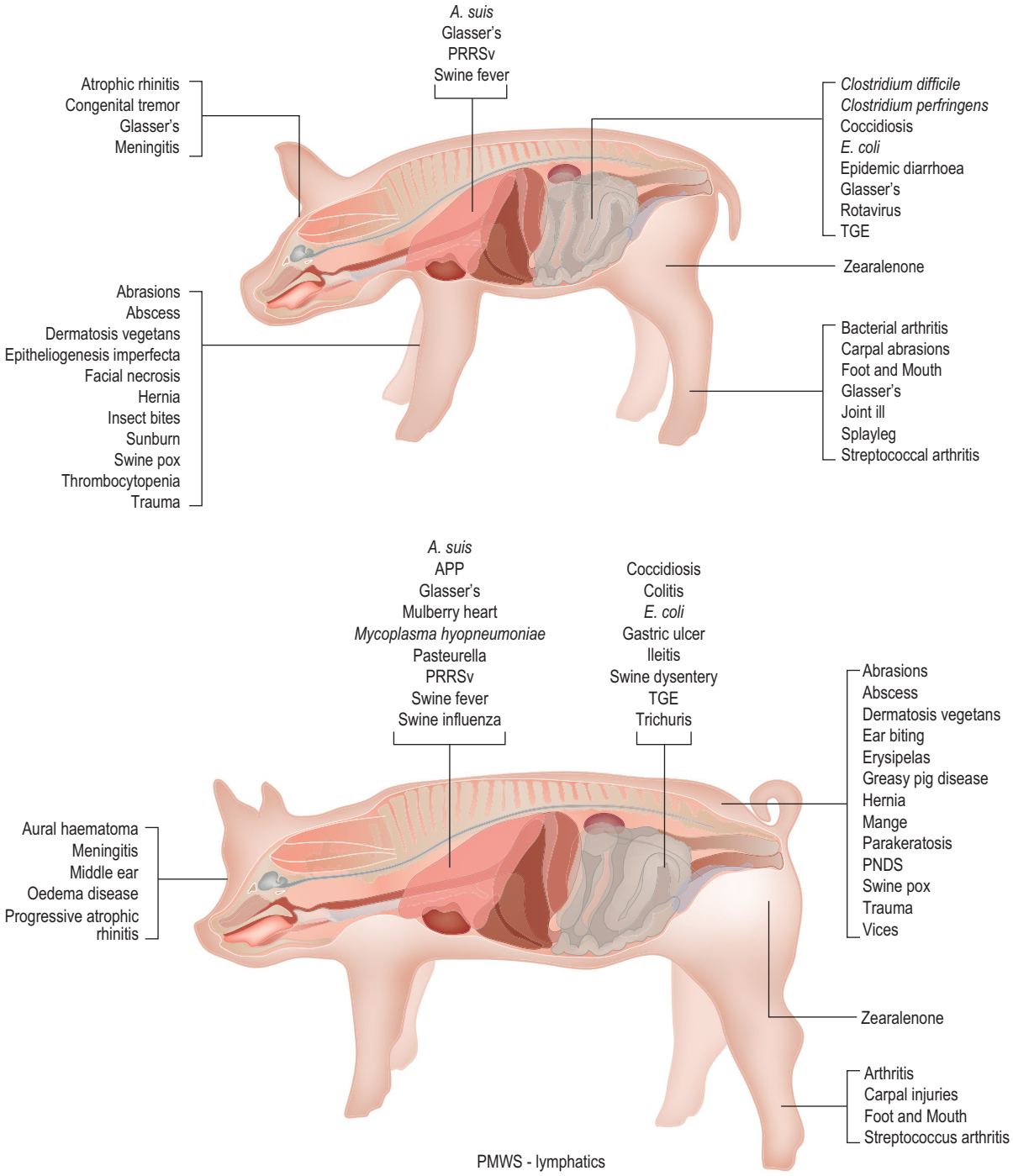

Fig 18.12 – Major disorders of the pig by age and group. PDNS, porcine dermatitis and nephropathy syndrome; PIA, porcine intestinal adenopathy; PMWS, postweaning multisystemic wasting syndrome; PRRS, porcine reproductive and respiratory syndrome; TGE, transmissible gastroenteritis. (Photographs courtesy of Dr John Carr, Iowa State University, Ames.)

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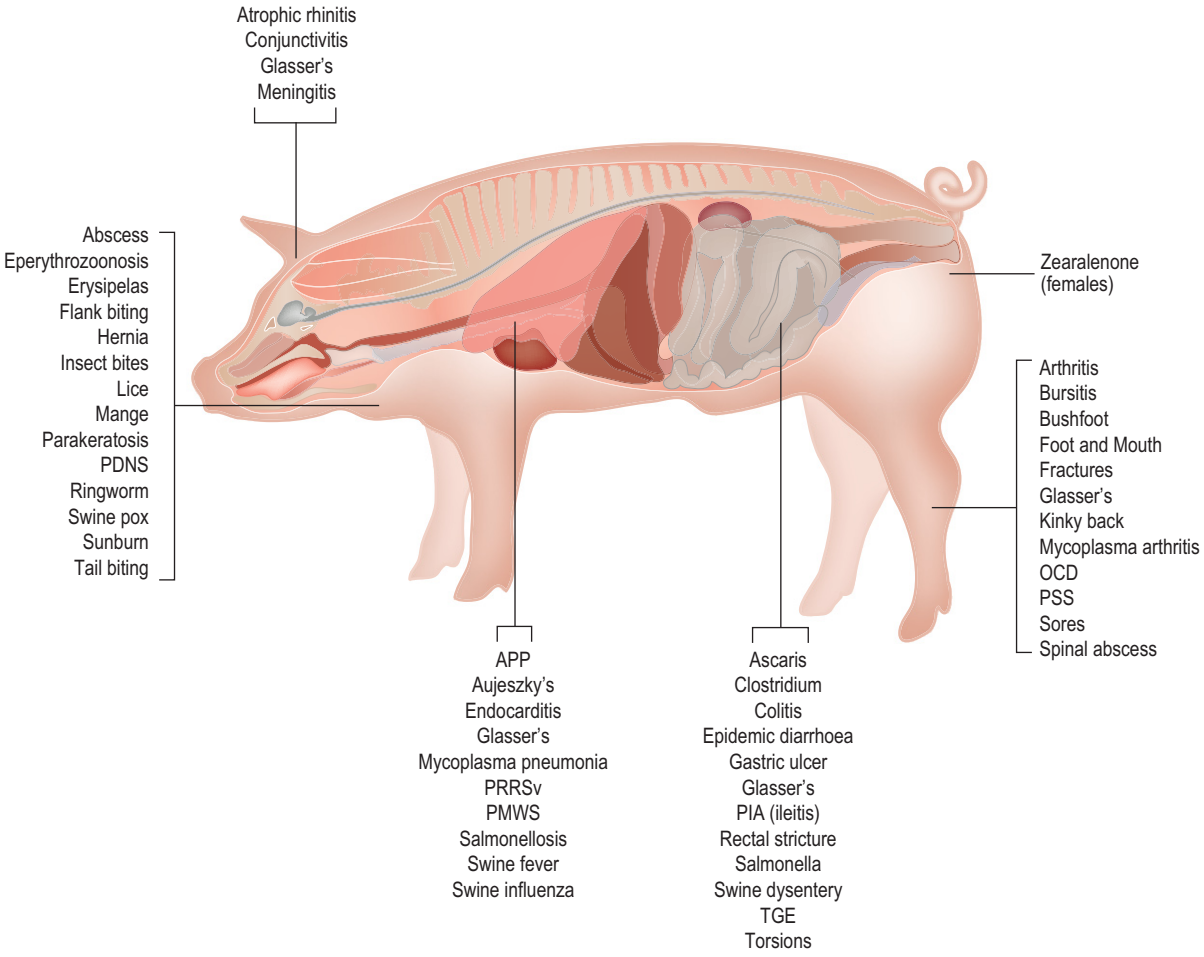

Fig 18.12 – cont'd

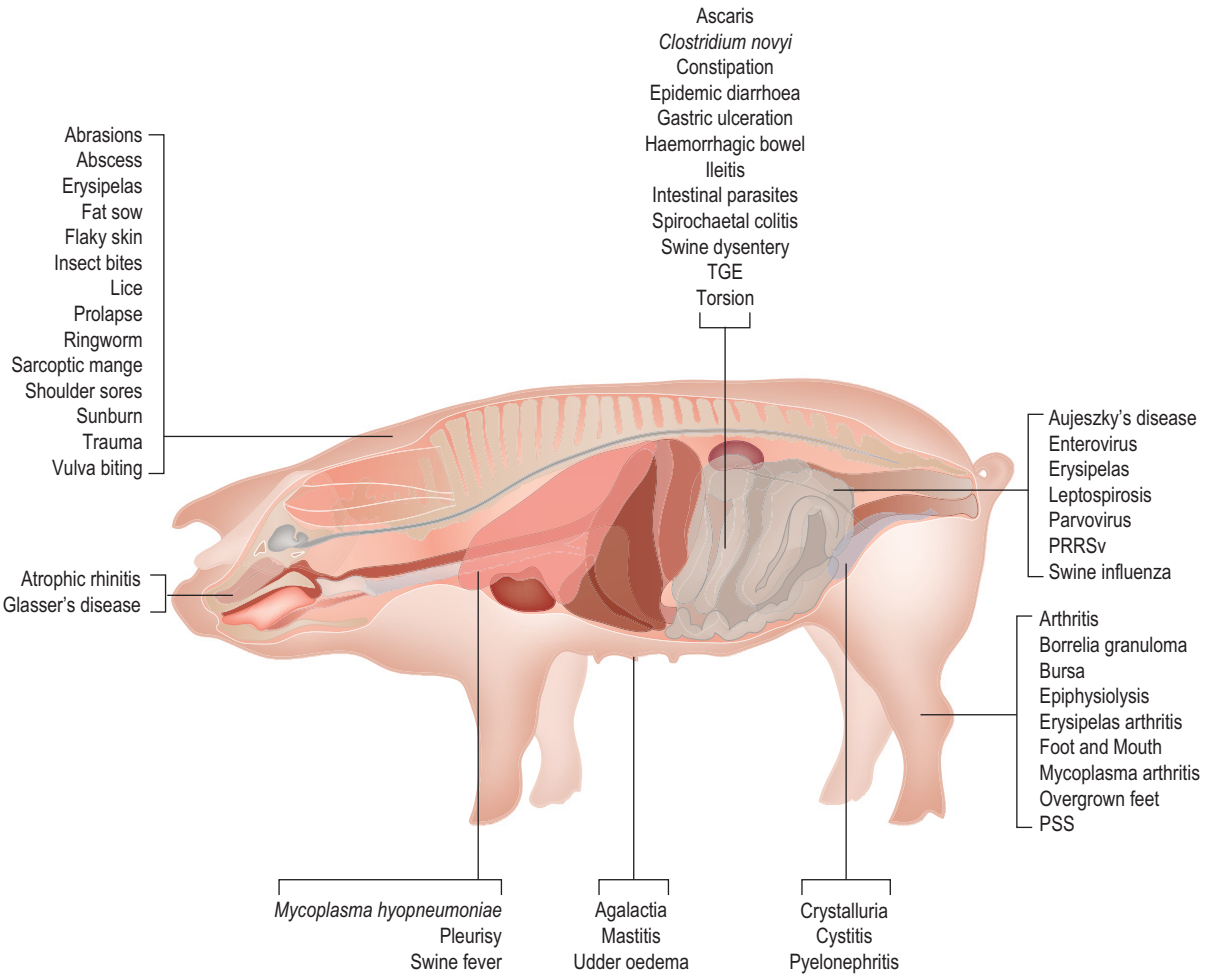

Fig 18.12 – cont'd

- Is there a difference between staff on duty?
- Is the boar working and is he fertile?
- Is artificial insemination being used?
  Technique or semen quality may be the problem.
- Are the number of sows returning to service with a vaginal discharge greater than 2%?
  Low-grade endometritis may reduce conception rates.
- Are the animals thin?
  Underfeeding will reduce conception rates, although too much feeding after mating may reduce litter sizes.
- Is the food mouldy and do the sows have an extended oestrus period?
  Mouldy food containing zearalenone may be responsible.
- Is there a short lactation period extending the weaning to service interval?
  Animals mated 8–12 days after weaning have reduced conception rates when compared with animals mated 3–7 days after weaning.
- Has the weather been exceptionally warm?
  Very high temperatures can reduce conception rates. Boars are affected only at 40°C plus.

Reduced farrowing rates

The farrowing rates are dependent on the conception rates, late returns to service, abortion rates, the proportion of mated sows that are culled, and the proportion of pregnant sows that die. It is an important parameter that has a direct impact on pig flow in the unit. The investigation should include the questions for high returns to service, pregnancy rates, abortion rate, culling rates, death rates, and boar fertility.

Failure to maintain pregnancy

When embryos die before day 12, the sows will return to service 18–24 days after mating. When fetuses die between days 12 and 30, they are resorbed by the sow's uterus. Most sows return to service more than 25 days after mating, but some may not recycle. Others with total litter loss may have a pseudopregnancy. When fetuses die between days 30 and 70, mummification occurs. After this, abortion is likely.

The causes of abortions, late returns to service (25 days plus after mating), and sows found not in pig should be investigated. Abortion may be caused by infectious pathogens (e.g. leptospirosis) or non-infectious agents (e.g. sunburn). Late returns to service are seen in cases of seasonal infertility, parvovirus, fungal toxins, and stress. The causes of sows identified as not in pig may include the conditions resulting in abortion and late returns to service, as well as inadequate oestrus and pregnancy checks. Attempts should be made to identify the extent and pattern of the problem.

Low litter size

The economic consequences of low litter sizes are often overlooked. The reduction of just one live piglet in a litter is equivalent to decreasing the farrowing rate from 85% to 75%. The low litter size may be due to low conception rates, early embryonic losses, mummifications, abortions, stillbirths, and boar infertility. Defining the problem by record analysis using appropriate targets and interference levels is important. Investigation of the possible causes must be systematic. There are many factors that impact on litter size.

- Are the records accurate?
  Difficult to assess but vital to address.
- Is there a problem with management at mating?
  Find out if oestrus detection, mating protocol, and boar usage are satisfactory.
- Could seasonal infertility or extremely hot weather be responsible?
- What is the age structure of the herd and which age groups are most affected?
  The herd may be young, with first-litter sows producing small litters, or may be composed of old sows with waning fertility.
- Is there a problem with farrowing or farrowing crate design?
  Inexperienced assistants, inappropriate induction protocols, or poor crate design can increase piglet mortality around farrowing.
- Is the lactation period short and the weaning to service period long?
  Every day below 28 days in the length of the lactation results in a reduction of 0.1 pigs born/litter.
- Is the feeding regimen from weaning to mating and in the early postweaning period appropriate? What is the body condition score of sows entering and leaving the farrowing crate?
  Inadequate feeding before mating and overfeeding immediately after mating can reduce the litter size. Thin sows may have smaller litters.
- Is the problem characterized by either a high number of late returns to service or by mummified fetuses or abortions or a high level of stillbirths?
  Consider infectious causes.
- Is the problem confined to high numbers of stillbirths?
  Consider deaths before farrowing (late infections), during farrowing (management and environmental factors), and early misdiagnosed deaths in live-born piglets.
- Is there a problem with the boar?
  Is he too young, subfertile, overused, or functional?
- Is artificial insemination being used?
  Are the technique and semen quality OK?

Boar infertility

Production records may indicate a reduction in farrowing rate, total number born, with an increase in the frequency of returns to service at 21 days ±3 days. Individual boar records are rarely available with indoor systems and not at all with the use of boar groups, so infertile individuals can be difficult to detect. Consider whether the problem may be over-usage or immaturity. Observations of mating behaviour (libido), mounting behaviour, and intromission in the presence of a sow in oestrus, and examinations of the scrotum, epididymis, testes, penis, and semen, may all be necessary to identify the type of problem.

Failure to mate effectively

- Poor libido
- Failure to mount
- Locomotory problems
- Failure to gain intromission
- Congenitally short penis
- Persistent frenulum
- Impotence
- Dilation or inflammation of prepucial diverticulum
- Prolapse of prepuce
- Injuries to penis

Mating and intromission normal

- Sperm abnormalities
- Low sperm numbers
- Infections of testes, epididymis, or accessory sex glands
- Systemic infections

See Glossop (1995) and Potter (1995) for further information.

Poor milk production

The condition is recognized by piglets in poor conditions that are persistently vocalizing and that do not settle on the teats. There is an increase in interpiglet aggression. Sows with painful udders will remain in sternal recumbency when the piglets are rooting.

- Are there enough teats? Are some inverted? Is there trauma to the teats?
- Is the sow very thin?
- Are any quarters affected by mastitis?
- Does the sow have mastitis, metritis, and agalactia?
- Is there enough food and water for the lactating sow?
- Could stress around farrowing be a factor?
  Moving the animal around or during farrowing or changing the diet can result in reduced milk production.

High replacement rate

This parameter includes animals that have been culled and animals that have died.

This figure needs to be considered in relation to the policy for optimizing performance and the genetic improvement on the farm. In a mature herd, 90% of gilts should progress to their second litter, with 85% of second-litter sows farrowing a third litter.

Unplanned culling may account for 60% of all culls. Major causes of unplanned culls include lameness, failure to conceive, and not in pig. Minor causes include lack of milk, anoestrus, abortion, and disease. Recording the reasons for culling will assist in identifying the problem. Prevalence studies within the herd are also helpful in identifying the cause(s) of the problem. Main causes of death on a farm include urogenital infections, intestinal accidents, lameness, and trauma.

Preweaning scours

Preweaning scours are an important cause of preweaning deaths and reduced growth rate. The age of pig affected and the severity may suggest a specific infectious agent (*Escherichia coli*, coccidia, rotavirus, and clostridia). Confirmation, if required, is by taking appropriate faecal and post-mortem samples.

Escherichia coli infection usually starts within the first 24–48 h (up to 5 days) and is more prevalent in piglets from gilts or piglets that have failed to suck, with failure of passive immunoglobulin transfer. It may also occur at about 2 weeks in association with coccidial infections. Mortality can be high. Scours caused by coccidiosis are usually seen at 10–14 days. Mortality is low. Rotavirus usually occurs from 4 to 12 days, with low mortality. *Clostridium perfringens* type C is uncommon but causes a severe bloody diarrhoea between birth and 14 days, with a high mortality.

Preweaning deaths

Most deaths occur in the first week of life. Consider if the problem is with the sow or the piglets and if it relates to infections, environment, or nutrition. Infections of the piglets include those causing scours (see *Preweaning scours*), septicaemia, greasy pig disease, and joint infections. Environmental factors include chilling, overlays, trauma, and suffocation. Nutritional factors include starvation, iron deficiency, vitamin E deficiency, and lack of colostrum. In addition, piglets that are weak, have splay legs, are anaemic (navel bleeding), are immature, or have low birth weights will have reduced viability.

Weaner scours

This is a major problem causing reduced growth rate and food conversion efficiency. *E. coli* and rotavirus scours occur within 10 days of weaning. *E. coli* is the most severe and common. Scouring can also occur in oedema disease. Swine dysentery can cause scours from 3 to 4 weeks after weaning but is more common in older pigs. Salmonellosis and proliferative enteritis occasionally cause scours 3–4 weeks after weaning. *Trichuris* may cause scours in extensively reared piglets.

Weaner deaths (up to 10 weeks of age)

Sudden deaths may occur without observed clinical signs (e.g. with vitamin E deficiency), and post-mortems are important when trying to establish the cause. The mortality rate and the morbidity rate of the surviving members of the group should be established.

- Was scouring observed?
  Scouring may indicate postweaning E. coli *infection.*
- Were nervous signs observed that may indicate meningitis?
  Salt poisoning, streptococcal meningitis, and bowel oedema should be considered.
- Are skin lesions present?
  Erysipelas, PDNS, and greasy pig disease have characteristic skin lesions.
- Were there signs of coughing and rapid breathing?
  Pleuropneumonia should be considered.
- Is the animal emaciated and jaundiced?
  If so, PMWS should be included in the differential diagnosis.

Grower and finisher scours

Swine dysentery is characterized by early soft pale yellow faeces, which later contain mucus and flecks of blood. The colour at this stage may vary from grey to reddish brown. Faeces may dribble from the anus, with the consistency of thin porridge.

Growth rates are poor. Pigs with proliferative enteritis are often thin with poor growth rates. Scouring may be

mild or severe, with normal or diarrhoeic faeces that may be blood-stained. Colitis is milder, with no blood in the faeces. Salmonellosis may present with blood-stained yellow diarrhoeic faeces. Gastric ulcers may present with anaemia and melaena. Endoparasites are usually a problem only in extensive pigs and present with loose green faeces.

Poor growth rates in growers and finishers

This problem may be recognized only following the analysis of the records. Growth rate for age should be analysed. Housing, management, nutrition, and infection may be responsible.

- Are there any signs of gastrointestinal infections (swine dysentery, proliferative enteritis, colitis, endoparasitism)?
- Are there signs of mange or lameness?
- Are there any signs of respiratory problems?
  Coughing (Mycoplasma hyopneumoniae, *with or without* Pasteurella multocida, *pleuropneumonia*), *sneezing, nosebleeds, twisted snouts* (*atrophic rhinitis*).
- Is the nutrition satisfactory?
  Energy, vitamin, mineral, and amino acid (e.g. lysine) deficiencies result in poor growth rates. Mycotoxins in mouldy feed can be a problem. Water availability should be checked.
- Is the housing and management satisfactory?
  Check the stocking density, trough space, air quality, and temperature.

Grower and finisher sudden death

Examine the rest of the group for clinical signs to decide if this is a sporadic condition or one affecting other animals in the group as well. The aetiology may be infectious, environmental, nutritional, or traumatic. Pigs may die suddenly from infections such as erysipelas, pleuropneumonia, *Pasteurella* pneumonia, proliferative enteritis, swine dysentery, oedema disease, PMWS and PDNS, anthrax, salmonellosis, and endocarditis. Nutritional causes include mulberry heart disease, hepatosis dietetica, gastric ulceration, and mycotoxins. Environmental factors are electrocution, heat stress, severe traumatic injuries, and porcine stress syndrome.

Coughing pigs

This is a major problem of intensive pig units. Mycoplasma pneumonia is the most common and may be complicated by secondary infections or other concurrent infections. Porcine reproductive and respiratory syndrome is now widespread and in the endemic form presents as a respiratory disease. Pleuropneumonia is severe, although less common than mycoplasma pneumonia.

- Is it a respiratory disease problem and what is the economic impact?
  Signs include coughing, uneven growth, reduced food intake, reduced food conversion efficiency, reduced weight gain, pigs reluctant to exercise, some pigs being panters, and death. Check the records for food conversion efficiency, mortality rates, medication used, and cost per kg/live weight gain.
- What is the cough index for each group?
  Monitor 50 pigs for 3 min after they have been disturbed. Interpret as follows.
  – *Less than two coughing episodes/3 min: normal*
  – *Three to five coughing episodes/3 min: suspicious*
  – *More than five coughing episodes/3 min: intervene or investigate*
  – *Over 10 coughing episodes/3 min: problem*
- Investigations should include post-mortems and slaughter inspection reports of lung lesions.

Nervous signs

- Is the pig preweaning or postweaning?
  Preweaning conditions include hypoglycaemia, central meningitis, and (uncommonly) congenital tremor and tetanus. Postweaning conditions include streptococcus meningitis, water deprivation/salt poisoning, oedema disease, Glasser's disease, spinal cord trauma or abscessation, and middle ear infection.
- Are the signs central (generalized) or localized?
  Local signs are present in spinal cord trauma or abscessation and middle ear infection.
- Are the generalized signs excitatory or is there depression?
  Meningitis and water deprivation/salt poisoning cause excitation. Hypoglycaemia, oedema disease, and Glasser's disease result in depression.
- Is a single animal affected or several?
  Spinal cord trauma or abscessation and middle ear infection are sporadic. Water deprivation/salt poisoning usually affects a group of animals.

Skin diseases

Establish the age of the pigs, the distribution, the types of lesion present, and if the condition is pruritic. Possible differential diagnoses include mange (pruritic); lice (pruritic); greasy pig disease (oily, crusty, brown); swine pox (small brown crusty lesions); ringworm (light brown patches); traumatic wounds; pityriasis rosea (raised red rings on belly and flank); parakeratosis (thick scabs on legs and belly); sunburn (painful); tail and flank sucking or biting; teat, tail, or ear necrosis (piglets); erysipelas (raised patches); and salmonellosis and other septicaemias (blue ears, limbs, and belly).

Lameness

Lameness may result from damage to the joints, the tendons, the ligaments, the muscles, the bones, the periph-

eral nerves, the spinal cord, or central brain lesions. A useful first step is to try to decide which structures are affected. Joint infections are characterized by distension of the joint capsule, pain on manipulation, and heat. More than one joint may be affected. Joint fluid can be obtained by joint taps using a needle and syringe following antiseptic preparation. Gross examination may confirm a septic arthritis. Cytology may confirm an inflammatory response and the presence of bacteria. Samples can be sent to a local diagnostic laboratory for culture. Serology may be useful in the case of suspected mycoplasma infection. Fractures and ruptured tendons and muscles usually cause severe lameness with dysfunction and deformity. Fractures may be palpable, with the presence of crepitus. Humeral and femoral fractures in larger animals may be difficult to confirm because of the size and bulk of the upper leg musculature. Fractures of the head of the femur may be detected following manipulation and palpation. Ruptured muscles are swollen and can be confirmed by ultrasound if required. Major tendon ruptures are palpable. Osteochondrosis resulting in degenerative changes to the articular cartilage can be confirmed at post-mortem or by radiology. Spinal abscesses or traumatic fractures of the lumbar vertebrae cause hind leg weakness or paralysis. Severe cases present in a 'dog sitting' position.

- *Preweaning*: splay leg, sole abrasions, and septic arthritis are easily characterized.
- *Growers and finishers*: joint infections (Glasser's disease, erysipelas, *Streptococcus suis*, *Mycoplasma hyosynoviae*), fractures, spinal abscesses, and osteochondrosis.
- *Adults*: fractures, muscle ruptures, foot infections, and overgrown claws.

Sudden death in adults

- Was a single animal or more than one animal found dead?
- If more than one animal were found dead, were they in the same group?
- Were the dead animals lying close together?
  Beware the possibility of electrocution. Turn the power off before investigating!
- If it was a single animal, has there been a series of sporadic deaths in the recent past?
  Spreading infection?
- Are there any other animals in the group showing clinical signs?
- Are there any obvious superficial abnormalities on the bodies?
- Is it possible to do a post-mortem?

Conditions to consider are septicaemias, endocarditis, torsion of the intestines, gastric torsion, fighting injuries, porcine stress syndrome, gastric ulceration and peritonitis, *Clostridium novyi*, heatstroke, cystitis or pyelonephritis, metritis, electrocution, and asphyxiation.

Troubleshooting algorithms and component analysis

By examining key parameters and the factors that influence those parameters, it is possible to isolate the abnormal factors that are having a major impact on the output of the unit. Parameters and factors can be organized in dendrograms or trees. By working down the tree, the investigation will move from a key parameter to component parameters and then to a group of causes or specific causes. The parameter *non-productive days* (this excludes gestation, lactation, and a 6-day weaning interval) is a compound parameter that can be analysed using component analysis. Another key output parameter that can be analysed in this way is the *number of weaned piglets produced per week* on the unit. The factors influencing the output are shown in Figure 18.13.

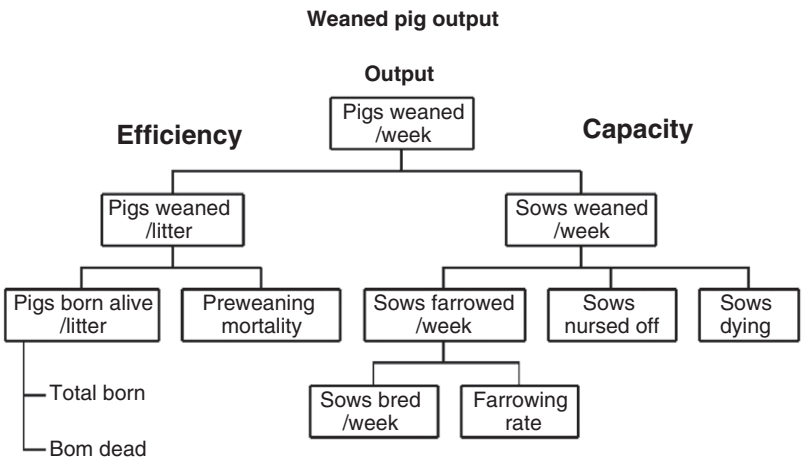

Fig 18.13 – Component tree for pigs weaned/week. (From PigChamp. Online. Available: http://www.pigchampinc.com. Courtesy of PigChamp and University of Minnesota.)

By using the component trees to troubleshoot weaning numbers, you can see all variables that contribute to weaned pig output. If it is concluded that the efficiency variables are the problem, in this case pigs weaned per week, then the variables pigs born alive and preweaning mortality can be focused on. Further examples are given in Figures 18.14–18.20.

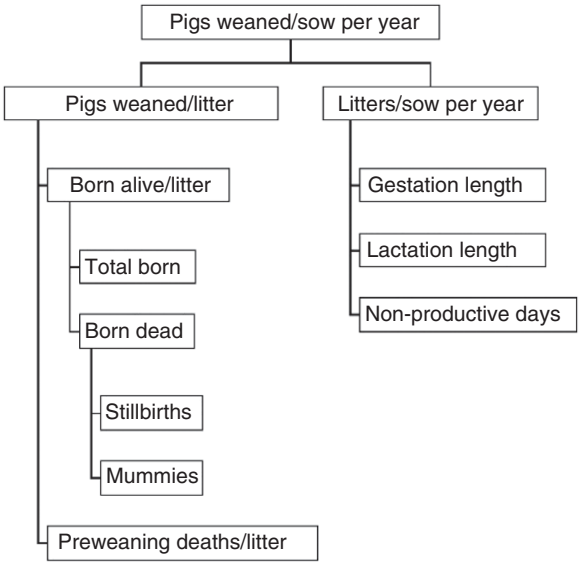

Fig 18.14 – Component analysis for pigs weaned/sow per year. (From PigChamp. Online. Available: http://www.pigchampinc.com. Courtesy of PigChamp and University of Minnesota.)

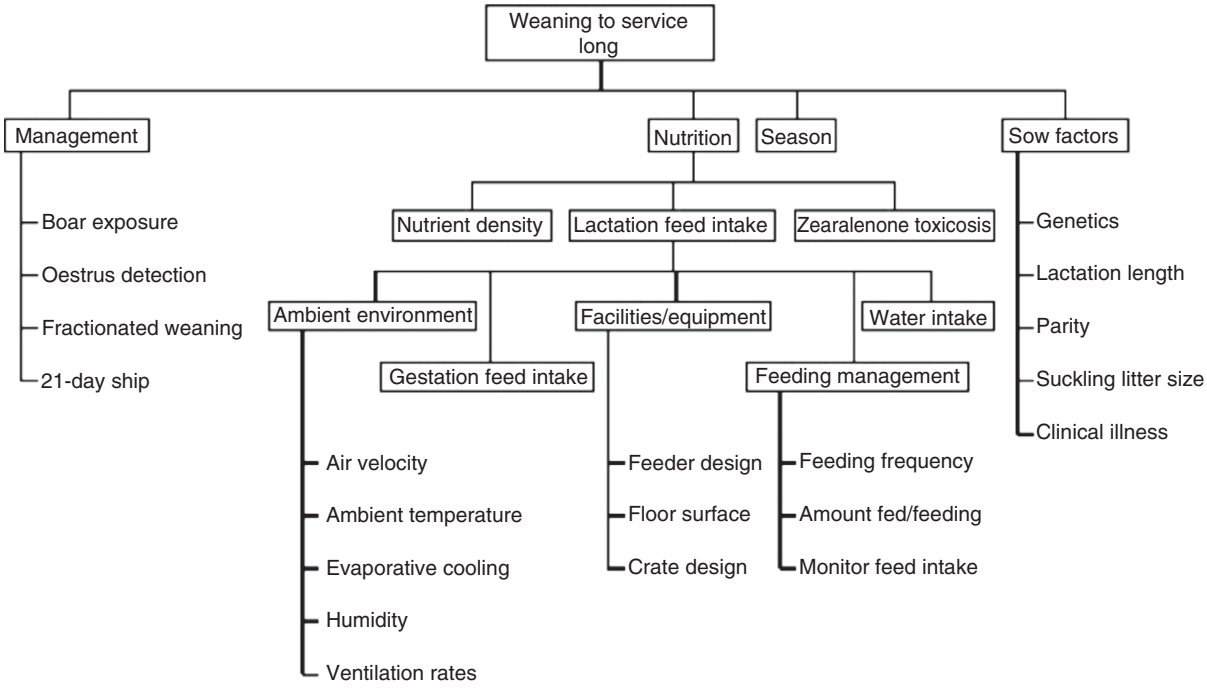

Fig 18.15 – Component analysis for a long weaning to service interval. (From PigChamp. Online. Available: http://www.pigchampinc.com. Courtesy of PigChamp and University of Minnesota.)

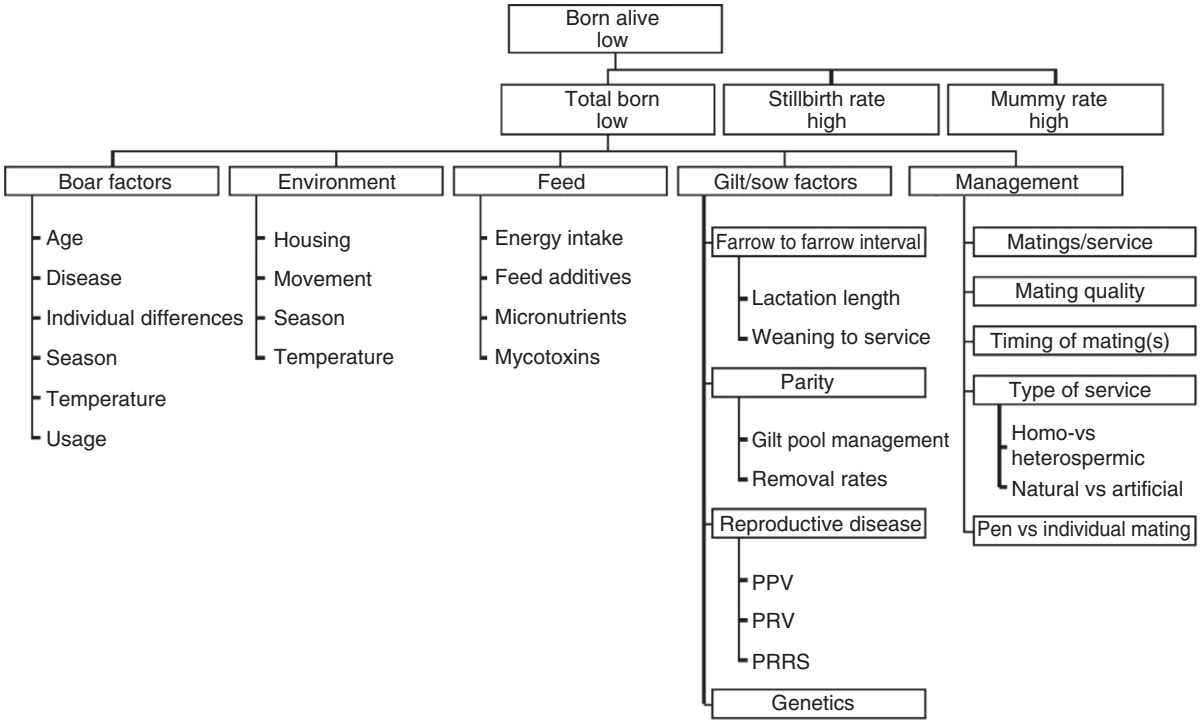

Fig 18.16 – Component analysis of pigs born alive. (From PigChamp. Online. Available: http://www.pigchampinc.com. Courtesy of PigChamp and University of Minnesota.)

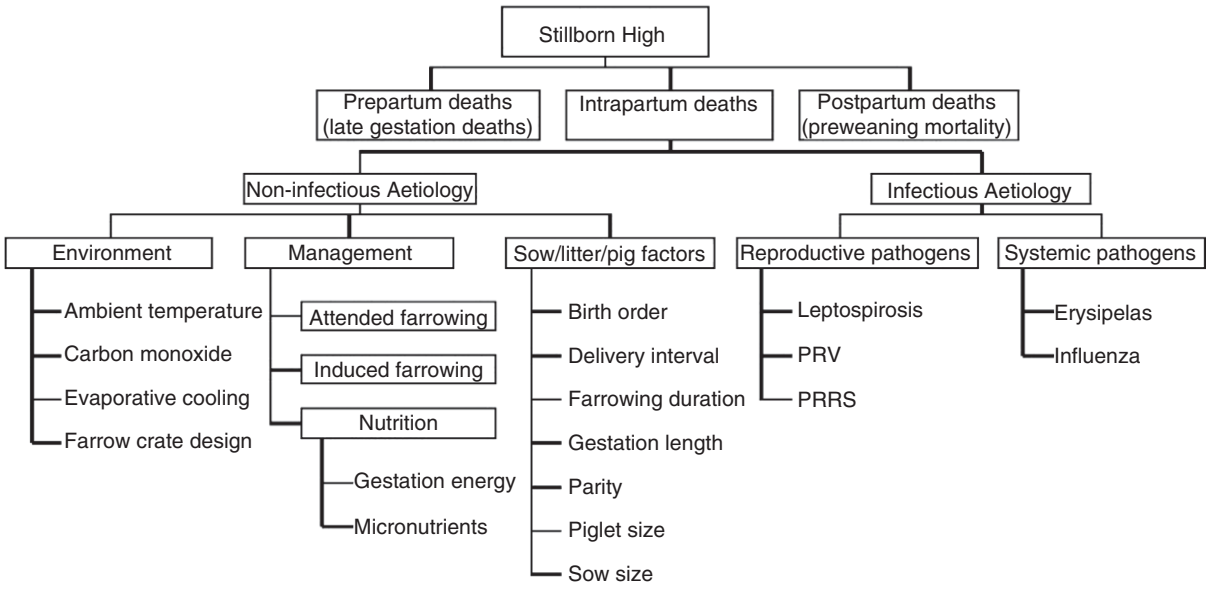

Fig 18.17 – Component analysis of stillbirths. (From PigChamp. Online. Available: http://www.pigchampinc.com. Courtesy of PigChamp and University of Minnesota.)

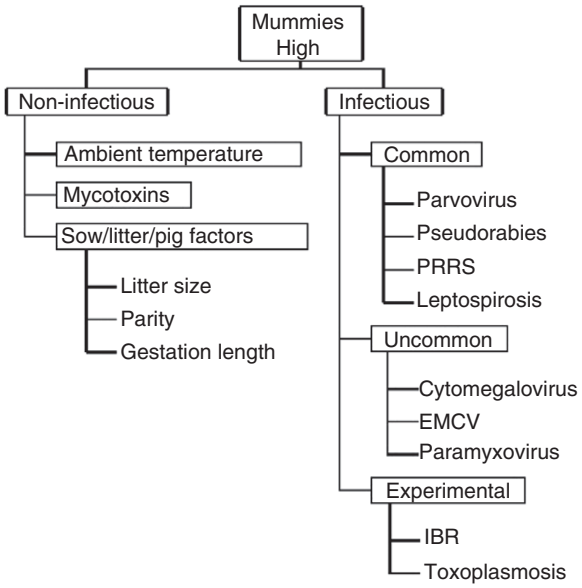

Fig 18.18 – Component analysis for mummified fetuses. (From PigChamp. Online. Available: http://www.pigchampinc.com. Courtesy of PigChamp and University of Minnesota.)

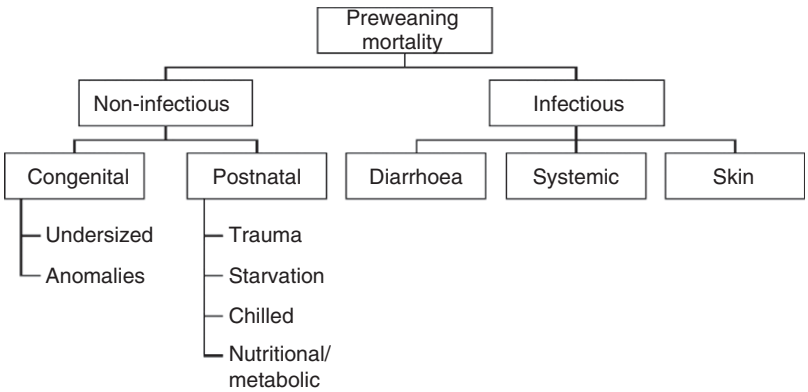

Fig 18.19 – Component analysis for preweaning mortality. (From PigChamp. Online. Available: http://www.pigchampinc.com. Courtesy of PigChamp and University of Minnesota.)

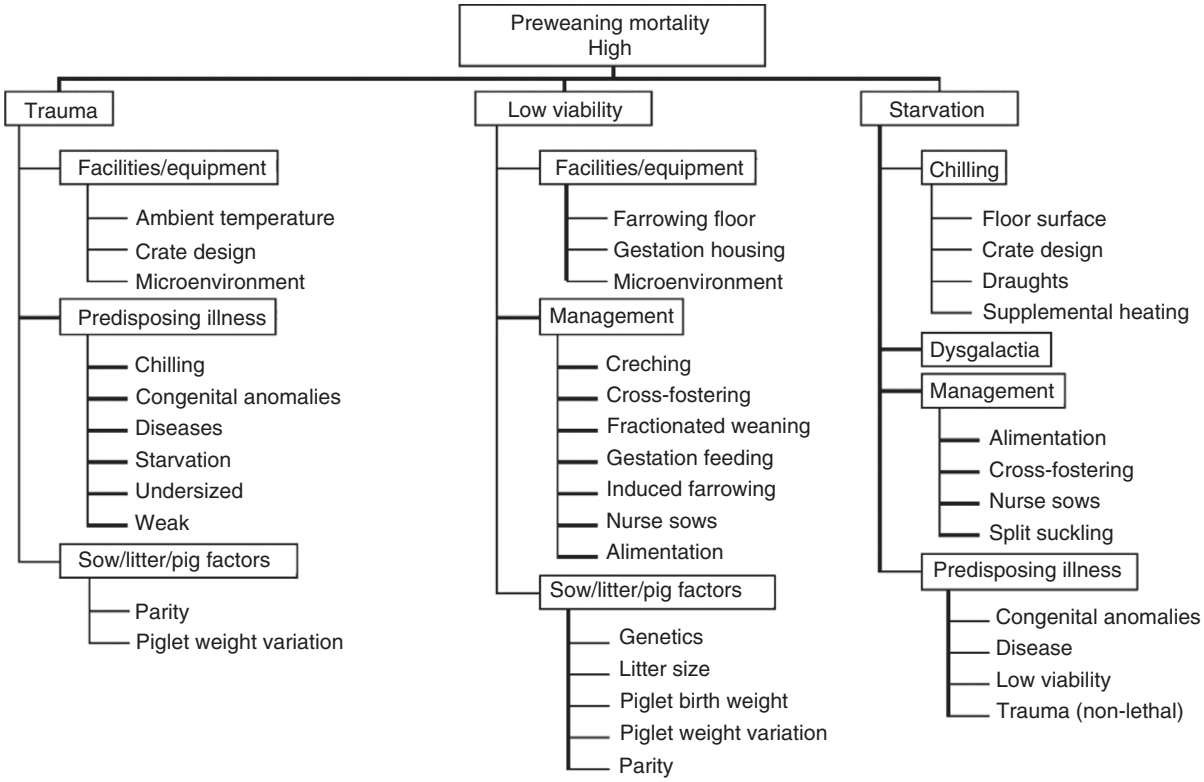

Fig 18.20 – Detailed component analysis of preweaning mortality factors. (Courtesy of PigChamp and University of Minnesota.)

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Cornell College of Veterinary Medicine's Consultant: a diagnostic support system for veterinary medicine. Online. Available: http://www.vet.cornell.edu/consultant/consult.asp

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Potter R 1995 Investigation into boar infertility. (1) Clinical examination. Pig J 35:28–33

Appendix 1

Notifiable diseases

The following pig diseases are notifiable in the UK, and the local divisional veterinary manager must be informed if there is any suspicion of their presence.

- African swine fever
- Anthrax
- Aujeszky's disease
- Classical swine fever
- Foot and mouth disease
- Rabies
- Swine vesicular disease
- Teschen's disease
- Vesicular stomatitis

Appendix 2

Office International des Épizooties lists A and B

By formal agreement, a large number of countries have agreed to report their status regarding important specified diseases to the Office International des Épizooties. This office collates and disseminates the information to other member states so that control and prevention measures can be initiated to reduce the impact of livestock diseases. There are two lists of diseases: list A and list B.

List A

List A swine diseases are diseases that have the potential for rapid spread and have serious economic or public health implications.

- Foot and mouth disease
- Swine vesicular disease
- Classical swine fever
- African swine fever
- Vesicular stomatitis

List B

In general terms, these are diseases of socio-economic and public health importance that spread less readily.

- Anthrax
- Atrophic rhinitis
- Aujeszky's disease
- Leptospirosis
- Porcine brucellosis
- Porcine cystocercosis
- Porcine reproductive and respiratory syndrome
- Rabies
- Teschen's disease
- Transmissible gastroenteritis
- Trichinellosis
- Tuberculosis

Appendix 3

Bibliography

The following books, journals, and web sites may be useful where further detail or information is required.

Books

Anonymous 1995 Good (pig) health manual. Pig Research and Development Corporation

Buddle RJ 2000 Differential diagnoses of diseases of pigs. University of Sydney Post Graduate Foundation in Veterinary Science, Sydney

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Muirhead MR, Alexander TJL 2002 Managing pig health and the treatment of disease. M5 Enterprises, Sheffield

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White M 2005 Pig ailments: recognition and treatment. Crowood Press, Marlborough

Journals

CABI Pig News and Information (Pigs)

Compendium of Continuing Education for the Practicing Veterinarian

In Practice

Journal of Swine Health and Production

The Pig Journal

UK Vet (Farm Animal)

Veterinary Clinics of North America: Food Animal Practice

Web sites

- Cornell College of Veterinary Medicine's Consultant: a diagnostic support system for veterinary medicine
  http://www.vet.cornell.edu/consultant/consult.asp
- Department for Environment, Food and Rural Affairs
  http://www.defra.gov.uk
- Iowa State University Swine Site
  http://www.vetmed.iastate.edu/departments/vdpam/swine/
- Office International des Épizooties
  http://www.oie.int
- Pig Diseases Information Centre
  http://www.pighealth.com
- The Pig Veterinary Society
  http://www.pigvetsoc.org.uk
- PigChamp
  http://www.pigchampinc.com
- The Pigsite
  http://www.thepigsite.com

Appendix 4

Pig vaccination

The text in this appendix has been adapted from the document Responsible Use of Medicines in Agriculture Alliance (RUMA) Guidelines: Responsible Use of Vaccines and Vaccination in Pig Production (www.ruma.org.uk) (Acknowledgement: Mark White) with permission. Tables 1, 2 and 3 are reproduced from this document with permission.

The relevant data sheets should be consulted before using any vaccine.

In most farm situations, vaccination against erysipelas and porcine parvovirus (PPV) will be essential.

Immunological responses to vaccination

Immunological responses to vaccination include humoral (largely IgG and IgM), mucosal response (IgA) (e.g. *E.coli*, *Lawsonia intracellularis*), cell mediated immunity (e.g. *Mycoplasma hyopneumoniae*) and colostrum (antibodies from serum and mammary tissue) (e.g. *E. coli*)

Maternally derived antibodies (MDA)

In the pig there is no direct transfer of circulating antibodies (IgM and IgG) across the placenta. Colostrum needs to be ingested as soon as possible after birth. The capacity to absorb immunoglobulins declines rapidly and by 24 hours of age absorption will not occur. IgA in colostrum/milk will continue to provide immunity. Vaccination of the sow prior to farrowing will increase the colostral antibody levels against the vaccinated organism(s) and boost piglet protection. The duration of protection depends on the quantity of colostrum consumed, the age of the piglet when colostrum is consumed, the concentration of antibodies in the colostrum, infectious challenge and the specific organisms against which the antibodies are active.

Maternally derived antibodies (MDA) can prevent response to vaccination. Exceptionally porcine parvovirus (PPV) maternally derived antibodies are very persistent and can block a response to any vaccination given before six months of age. If vaccination is cell mediated, maternal antibodies have very limited blocking effect on vaccinal response (e.g. *Mycoplasma hypopneumoniae* vaccines) and vaccination can begin early in life.

Targets of vaccination

The vaccine may protect the individual to which the vaccine is administered (e.g. *Mycoplasma hyopneumoniae* and *Lawsonia intracellularis* vaccines), the unborn litter by vaccination of the dam (e.g. PPV), the newborn piglet by vaccination of the dam and acquisition of MDA (e.g. *E. coli, Clostridium perfringens*) or both the vaccinated sow and her litter via MDA (e.g. erysipelas).

Vaccine failure

The majority of failures of vaccination regimes in pigs are the result of misdiagnosis, additional pathogenic agents, misuse of vaccines (storage, application and dosage), excessive challenge by field infection, strain variants not covered by the vaccine used (e.g. *E. coli*, erysipelas, *Haemophilus parasuis*), and failure of passive transfer. If a fault with the vaccine is suspected this should be reported to the VMD.

Authorized vaccines

Vaccines authorized for use in the pig in the UK have zero meat withdrawal periods following application.

Vaccines on cascade

Any vaccine used outwith its stated licensed conditions (e.g. vaccines licensed in other food producing species but used in the pig e.g. *Salmonella typhimurium* vaccine) can **only** be supplied and used under the direction of the veterinary surgeon attending the farm, and the standard meat withdrawal period of 28 days may apply following administration.

Importation of vaccines

Where significant disease problems occur within the UK and no effective product is available for the prevention

and control of disease, it is possible to import authorised products under special licence both from within and outwith the EU. These can only be applied for by a veterinary surgeon and take the form of a Special Import Certificate (SIC) (for an EU-authorised product) or a Special Treatment Certificate (STC) (non-EU product) granted by the Veterinary Medicines Directorate (VMD). A charge is made for granting such a licence. The licence may impose the standard withdrawal period on the product used.

A number of vaccines are authorised within the EU, but not in the UK, for significant diseases seen in this country and, where appropriate, it may be possible to import the product under licence for use on specific premises. Three particular diseases of pigs maybe relevant:

1. *Actinobacillus pleuropneumoniae* (APP)

This respiratory disease in growing pigs can be effectively controlled by vaccinating pigs during the early growing stage, typically six and 10 weeks of age. There are currently 13 serotypes of APP identified worldwide, of which a restricted number have been recognised in the UK. The single vaccine authorised in the Netherlands and elsewhere is a subunit vaccine based on four antigenic components common to all serotypes. In theory the vaccine thus covers all possible serotypes. Protection is likely to occur 2–3 weeks following the second injection and would be expected to protect pigs through to slaughter weight.

2. *Streptococcus suis* type II

Typically, disease associated with this bacterium occurs in the immediate post-weaning period (four to six weeks of age) causing meningitis, septicaemia and polyarthritis. Protection of the young piglet can be achieved by vaccination of the sow, relying on colostrally-derived MDA to provide passive immunity. Vaccination is achieved using a killed oil-based vaccine given six and two weeks prior to farrowing at the start of the programme, with single booster doses 2–3 weeks prior to subsequent farrowings. As a serotype specific vaccine, protection against disease caused by other *Strep. suis* serotypes is unlikely to occur.

3. Porcine circovirus type II (PCVII)

PCVII is an integral component of the post-weaning multisystemic wasting syndrome (PMWS) although it may not be either the sole or primary cause of the disease. Trial work in developing a killed vaccine for use in sows prior to farrowing indicates some protection against PMWS in weaned pigs. Currently, a vaccine has a provisional licence in some EU countries (e.g. France, Germany and Denmark) and it might be possible under the SIC/STC system to import the vaccine where the clinical need can be shown for a specific farm. The standard vaccination programme would involve a two dose primary course of vaccine to sows in late pregnancy followed by a single dose prior to subsequent farrowings. Guidance should be sought from the VMD over the possibility of importing such a product.

Non-disease vaccines

For a number of years immunological castration of pigs has been undertaken in Australia. The vaccine acts against gonadotrophin releasing hormone and temporarily induces vaccinates to destroy GnRH which then limits production of testosterone. The effect is to shrink the testicles, reduce male behavioural characteristics and cut down on boar taint. The vaccine may soon become available in the UK. The vaccine may have limited application in the UK as boar taint is generally not regarded as a problem in the carcasses of entire boars. However, the trend to increased slaughter weight may invite application of vaccination, particularly as physical castration of boars in the UK is banned under the major quality assurance schemes.

Two doses are essential for the vaccine to be effective, with an appropriate interval. The effect lasts for 4–6 weeks after the second dose in boars and thus needs to be used in the later stages of finishing. Operator safety is a priority when using this vaccine, as double accidental vaccination will have dramatic effects on sexual hormones in both male and female operators.

Vaccination against specific diseases

1. Porcine parvovirus (PPV)

It is the most common cause of the stillbirth, mummification, embryonic death and infertility (SMEDI) syndrome. The cost of disease relative to the vaccination costs is high As a result of widespread use of PPV vaccines, clinical disease is now rarely seen. The virus is ubiquitous within pig populations and can be spread in semen. It is therefore essential that all gilts are immune prior to breeding. This may occur by field exposure with life long immunity or vaccination. Exposure can be confirmed by serology. Where a new gilt herd is established there is a very high risk of PPV disease and thus vaccination is essential. Some PPV vaccines are also licensed for use in the boar with the aim of reducing viral excretion in semen. PPV has no adverse effect on the boar. Typically gilts would be vaccinated two to four weeks prior to first service but not before six months of age. Further single doses may be needed annually (depending on the product used) although this may be superseded by field challenge of the vaccinated gilt, imparting lifelong immunity.

2. Porcine reproductive and respiratory syndrome (PRRS)

This viral disease is widespread in the UK pig herd although some individual herds have been maintained PRRS free. In naïve herds the virus may cause devastating epizootics affecting fertility and piglet survival, but it is now commonly a chronic problem reducing sow productivity and increasing growing pig respiratory disease. Introduction of the virus to the herd may be airborne, by infected stock (e.g. replacement gilts) and in semen as well as by fomites. A naïve herd should be considered for vaccination if biosecurity is sub-optimal. Both live(breeding and growing) and killed vaccines (breeding) are available. Live vaccines are not recommended for use in a PRRS-free herd or pregnant animals. Vaccination of growing pigs must be delayed until MDA has largely waned (usually five to six weeks old). Short term excretion following live vaccination occurs and contact with susceptible pigs should be avoided.

In the breeding herd where PRRS disease is a regular or repeated problem, the low cost of vaccination gives a cost benefit ratio of 1:10 to 20 i.e. it is highly cost effective. In the feeding herd PRRS can act as a facilitator of other respiratory diseases and commonly acts in concert with *Mycoplasma hyopneumoniae*. The cost of vaccination would be easily outweighed by a reduction in growth rates delaying slaughter by five days taking no account of other specific treatments required and decrease in feed conversion efficiency.

3. *E. coli*

E. coli vaccination prior to farrowing only protects against neonatal enteritis and not postweaning disease. Colostral antibodies (IgA) prevent adhesion by pathogenic *E. coli* to the intestinal wall for the first 3–4 days of life. New herds, particularly indoors, are at high risk of neonatal *E. coli* disease and as such should be vaccinated.

In general, gilts are given two doses prior to farrowing (with the appropriate interval) and sows a single booster prior to each subsequent farrowing. In many herds, vaccination of the gilts alone is sufficient to control disease, but such a decision must be made on a case-by-case basis.

Neonatal *E. coli* disease can cause high levels of mortality in affected litters (over 50% in some cases) and the relatively low cost of vaccinating one sow to protect 10 piglets means that *E. coli* vaccines are highly cost effective. All *E. coli* vaccines are dead, consisting of a variety of strains covering the most common adhesions. Where known strains are a problem, it may be appropriate to choose a specific vaccine. Some vaccines also contain a toxin component. *E. coli* vaccines may be marketed as combination products, e.g. with erysipelas or clostridial vaccines.

4. Clostridial vaccines

Clostridia are soil and faecal borne organisms that sporulate and persist in the environment. Clostridial disease in the pig can take one of three forms: Clostridial enteritis associated with *Clostridium perfringens* types A, B, C and D in piglets, Aero chocolate liver in sows and older growing pigs (*Clostridium novyi*) and tetanus (rare). Where clostridial enteritis is encountered in young piglets vaccination of the gilt/sow prior to farrowing with a polyvalent clostridial vaccine is highly effective. These toxoid vaccines do not cover *Clostridium perfringens* type A disease.

As a general rule, indoor pig farms are low risk from clostridial disease and vaccination is usually only undertaken where there is a history of disease. However, the outdoor herd is at a much higher risk and normally sows would be vaccinated as a routine. To obtain an appropriate vaccine, the prescribing cascade may need to be consulted.

5. Erysipelas

The causative organism of erysipelas in pigs (*Erysipelothrix rhusiopathiae*) is widespread in vermin and wild birds. The pig is particularly susceptible to disease (sudden death/septicaemia, reproductive disease – abortion and infertility, chronic arthritis and endocarditis) and therefore all pig farms should be regarded as high risk and vaccination is recommended. In particular, straw barn-type accommodation for growing pigs, to which wild birds have access, creates a situation in which erysipelas is common. (It is rare in growing pigs in fully enclosed, slatted floor-type buildings). Vaccination – using inactivated vaccines – should be routinely applied to gilts and sows to protect both themselves and their offspring. Growing animals at high risk can be vaccinated from 6 weeks. The chronic arthritic form of erysipelas, which is often the most costly form of the disease in growing pigs, results from a type III hypersensitivity reaction and as such will not in itself be controlled by vaccination. However, a vaccination programme over time will reduce the overall bacterial challenge and therefore reduce the risk of pigs coming into contact with the organism, thereby reducing the chances of arthritis occurring.

6. *Mycoplasma hyopneumoniae*

Enzootic pneumonia (SEP) resulting from infection with *M. hyopneumoniae* is the most common respiratory disease seen in pigs and can often act as a trigger of other infectious diseases in the lung. Killed vaccines for use in the growing herd are now widely used. They are available in either single or two dose format, the immunity from which lasts up until slaughter in most situations. Programmes vary with vaccine and individual farm situa-

tions, but in most cases early vaccination (as young as one week of age) is appropriate depending on the vaccine used. The killed vaccines depend upon cell-mediated immunity and it is believed that maternally derived antibodies have minimal adverse effect on the piglets' reaction to vaccination. SEP vaccines are valuable tools in controlling population disease.

Examination of lungs of vaccinated pigs reveals that some disease is still evident but, as a general rule, vaccination will reduce the average percentage of lung tissue affected in a herd to less than five percent (SEP score two on the 55 scoring system).

7. *Lawsonia intracellularis*

This is the causative organism of the range of clinical enteric diseases that include ileitis, intestinal adenomatosis (PIA) and proliferative haemorrhagic enteropathy (PHE). Virtually all pig farms have been shown to be infected with the causative organism that can be a significant cause of lost growth and even mortality. The vaccine available is a live whole bacterial culture that is administered orally and stimulates a local, mucosal reaction in the intestine. It can be administered through the water supply, separate water troughs or by drenching of individual pigs. The water must not be chlorinated (mains water is), which kills the vaccine. The addition of skimmed milk powder can neutralize the chlorine. Antimicrobial treatment should be avoided at least 3 days either side of vaccination. Zinc oxide in the diet of pigs receiving vaccine does not interfere with vaccine efficacy. Where *L. intracellularis* alone is responsible for disease, vaccination would be expected to give a 3 to 5:1 benefit:cost ratio.

8. Atrophic rhinitis (AR)

Progressive atrophic rhinitis caused by toxin producing *Pasteurella multocida* type D is now an unusual disease in the UK. Vaccination using *P. multocida* toxoid, plus inactivated *Bordetella bronchiseptica* is a highly effective way of controlling the disease that results from infection in the young pigs (usually before 40 days of age). Therefore, the protocol employed is to vaccinate gilts and sows prior to farrowing and rely upon transfer of colostral immunity to protect the piglets after birth. Atrophic rhinitis vaccines should only be used in herds where the disease is known to occur. There is no need or justification for its use in the disease-free herd as the risk of introduction is low.

In an affected herd, the cost of AR can be very high with feed conversion efficiency depressed by up to 0.5 across the growing herd. When added to the costs of medication and additional mortality, the cost of the disease can exceed the cost of a vaccine programme by 20 – 30 times. Vaccination of the breeding herd is thus highly cost-effective in an affected herd.

9. Glasser's disease

Disease associated with *Haemophilus parasuis* infection is common in the UK. There are in excess of 20 different strains of the bacterium and currently there is no readily available method of serotyping isolates in the UK. Commercial vaccines rely on one or two specific strains from which there is little or no cross immunity to others.

The available killed vaccine is designed for use in the growing pigs, requiring two doses with an appropriate interval targeted for the second dose to be two weeks prior to challenge. This will then cover the growing pigs through to slaughter. Maternal immunity may interfere with vaccine efficiency and as such vaccination should not start before five weeks of age.

Unfortunately, much of the clinical disease seen occurs in younger pigs that cannot be vaccinated. Vaccine may be used off licence under the direction of the veterinary surgeon only, with sows vaccinated prior to farrowing (two dose primary course followed by single booster doses in subsequent pregnancies). The success of the vaccination programme will depend upon the vaccine containing the correct strain of *Haemophilus parasuis* for the farm's problem.

10. Aujeszky's disease – (AD) (Pseudorabies)

Vaccination for AD is only appropriate in Northern Ireland, where the disease has persisted. Eradication programmes are currently being undertaken in many European countries using a combination of vaccination and test and removal. Vaccines are live attenuated products, which have undergone gene deletion removing a glycoprotein. It is possible, with appropriate serological tests, to distinguish between field virus infections and vaccinates. This is important as part of herd eradication programmes based upon vaccination, test and slaughter,

11. *Salmonella typhimurium*

Clinical disease – usually seen in the form of diarrhoea in weaners – associated with *Salmonella typhimurium* is relatively uncommon in pigs. However, infection is widespread and as part of a national campaign, slaughter pigs are monitored serologically for the presence of previous infection. Heavily infected herds risk financial penalties at slaughter. No licensed vaccine is available for pigs; however, a live attenuated poultry vaccine for oral use is available but must be used only under veterinary direction, off licence.

The aim is to stimulate local IgA production to prevent adhesion to the villi of the intestine as the precursor to penetration and bacteraemia, which stimulates the humoral response, detected by the serological test.

Double dose vaccination via the water supply has proved effective at controlling clinical outbreaks of disease and may go some way to reducing the levels of *Salmonella* spp *in* the pig environment and allow a reduction in the serological incidence of infection. It would appear that vaccination does not increase the incidence of seropositive animals at slaughter, particularly if administered early in life (i.e. before seven weeks of age), suggesting that it does not stimulate a significant humoral response.

As a live oral product, *Salmonella typhimurium* vaccination is vulnerable to being inactivated if used concurrently with antimicrobials and/or mains water that is chlorinated. The inclusion of zinc oxide in the creep feed may interfere with the efficacy of this vaccine and should be avoided.

As the vaccine is not licensed for use in the pig, a standard withdrawal of 28 days should be applied to all pigs receiving it.

Pet Pigs

Assuming that the pet pig is not kept for breeding, then none of the diseases controlled by vaccinating the breeding sow are relevant (*E. coli*, AR, PPV). Furthermore, the main respiratory diseases (PRRS and SEP) are unlikely to be of significance to the individually kept pig and vaccination is usually unnecessary.

However, two diseases can be of significance in the pet pig and vaccination should be considered:

1. Erysipelas
2. Clostridial disease (especially *Clostridium novyi* and *Clostridium tetani*).

Further reading

NOAH Compendium of Data Sheets for Animal Medicines, published by NOAH and online at http://www.noahcompendium.co.uk

Table 1 Suggested vaccination regime for young growing pigs

Age/Stage of Production	Disease	Age of Vaccination + schedule	Comments
Preweaning/At weaning	SEP	Single dose from 7 days of age Or 2 doses 14+ days apart	Only SEP +ve farms or where high risk of disease breakdown exists
Post-weaning	Glasser's disease	2 doses 14 days apart from 5 weeks of age	Where disease is known due to appropriate strains included in vaccine
	PRRS	Single dose of live vaccine from 6 weeks of age	Only when PRRS clinical disease is present in growing herd or high risk
	Ileitis/*Lawsonia intracellularis*	Single dose post-weaning in water or 2 doses 2 weeks apart	Avoid in feed/water soluble antibiotics
	Salmonella typhimurium	Single dose post-weaning in water or 2 doses 2 weeks apart	Avoid antibiotic and zinc oxide treatment. Off licence use 28 day withdrawal period
	Erysipelas	Single dose from 6 weeks of age	Where disease is known in growing pigs or high risk e.g. future breeding animals
	Aujeszky's disease	Single dose from 14 weeks of age	Only Northern Ireland

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Table 2 Suggested vaccination regime for breeding herd purchasing replacement gilts

Stage of Production	Disease	Regime	Comments
Predelivery or in isolation	SEP	Single or 2 dose regime – 2 weeks prior to introduction	Only if naïve prior to supply or in specific high risk situation
	PRRS	Single live vaccine dose 3 weeks prior to introduction (or 2 doses of killed vaccine 6 and 3 weeks prior to introduction)	Only if naïve prior to supply or in specific high risk situation
35 kg delivery (3 months old)	Erysipelas	Single dose if not previously vaccinated	
90–100 kg delivery ($5^1/_2$–6 months old)	Erysipelas	1st dose of primary course	
	PPV + 2nd Erysipelas	4 weeks later at least 2 weeks prior to service	
	PRRS	Live vaccine 4 weeks pre-service or killed vaccine 6 and 3 weeks pre-service (if not vaccinated prior to delivery)	
	Aujeszky's	Single dose preservice	Northern Ireland only
During pregnancy	*E. coli*	2 doses prior to farrowing 2–6 weeks apart	2nd dose at least 2 weeks prefarrowing; interval depends on vaccine used
	PRRS	Single booster dose killed vaccine at 70 days gestation	Where killed vaccine has been given to maiden gilts
	Clostridia	2 doses 3 weeks apart, 2nd dose more than 2 weeks prefarrowing	Outdoors or where known risk indoors
	Glasser's disease	2 doses prior to farrowing (e.g. at 5 and 2 weeks prefarrowing)	Off licence use
	Atrophic rhinitis	2 doses prior to farrowing	Only where AR known
	Erysipelas	Single booster 2–3 weeks prefarrowing	

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Table 3 Suggested vaccine regimes for sows

Sows	Disease	Regime	Comments
Preweaning	PPV	Booster PPV dose	
	PRRS	Live vaccine booster (unless used off licence at 70 days gestation)	
Subsequent gestation	*E. coli*	Booster 2–4 weeks prefarrowing	Often not necessary beyond gilts
	Erysipelas	Booster 2–4 weeks prefarrowing	
	Clostridia	Booster 2–4 weeks prefarrowing	Where high risk or outdoors
	AR	Booster 2–4 weeks prefarrowing	Where disease known in the growing herd
	Aujeszky's disease	Boosters every 4 months	Northern Ireland only
	Glasser's disease	Booster 2–4 weeks prefarrowing	Off licence use

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Index

A

Abattoir monitoring, 45
Abdomen, 9
  distension, 3
  in piglets, 13
Abortion, tissue sampling, 246
Abscess, subcutaneous, 3, 8
Acepromazine, 54, 232
Actinobacillosis, 144–5
Actinobacillus pleuropneumoniae, 16, 17, 18, 206
Actinobacillus suis, 59
Actinobaculum suis, 155, 203
Acute septic metritis, 173
Aflatoxicosis, 109, 142
African swine fever, 184–5
Agalactia, 161–2, 173
  outdoor pig units, 204
Aminoglycosides, 222
Amoxycillin, 228
Ampicillin, 228
Anaesthesia, 230–41
  general, 6
  intramuscular injection, 232
  intravenous regional anaesthesia of lower limb, 233
  intubation and venepuncture, 231
  legislation, 230
  lumbosacral epidural, 232–3
  owner communication, 230
  preanaesthetic evaluation, 230
  premedication, 230
  preparations, precautions and aftercare, 230–1
Analgesia, 230
Anthrax, 192
Antimicrobials, 220–9
  aminoglycosides, 222
  cephalosporins, 222
  decision analysis, 224
  diterpines, 222
  drugs licensed, 228–9
  immunocompetence, 223
  lincosamides, 222
  macrolides, 221–2
  penicillins, 221
  resistance, 223
  sensitivities, 220–1, 223
  specificity, 224, 225–8
  spectrum of activity, 223–4
  sulphonamides, 221
  tetracyclines, 221
  see also individual drugs
Apramycin, 228
Arcanobacter pyogenes, 10, 124
Arthritis, 20
  chronic septic, 61–2
  outdoor pig units, 201
  see also Musculoskeletal disease
Arthrogryposis, 51
Artificial insemination, 174
Ascariasis, 110–11
Ascaris suum, 20, 110–1
Ascites, 9
Aspergillus flavus, 109
Aspergillus parasiticus, 109
Aspirin, 231
Assurance schemes, 29–41
  Assured British Pigs, 30–1
  environmental comfort, 40
  gases, dust and bacteria, 40–1
  sow condition scoring, 39–40
  veterinary health plans, 31–9
Assured British Pigs, 30–1
Asymmetrical hindquarters, 53
Ataxia, 4
Atresia ani (imperforate anus), 85
  surgery, 233–4
Aujeszky's disease (pseudorabies), 22, 132
Aural haematoma, 125, 234
Azaperone, 6, 232

B

Back muscle necrosis, 54
Bedding, 27
  poor, joint problems caused by, 66–7
Bilateral renal hypoplasia, 156
Biosecurity, 21–9
  carcass disposal, 28–9
  cleaning, disinfecting and deliveries, 29
  employees, visitors, tools and equipment, 28
  feed, bedding and water, 27
  herd location and perimeter security, 23
  intraherd security, 29
  replacement and new stock, 22–3
  transportation, 27–8
  wildlife, 23–7
Biotin deficiency, 68, 121
Bird trauma, 204
Bladder, post-mortem examination, 255
Blood biochemistry, 258–9
Blood sampling, 14, 242
  pet pigs, 218
Bloody gut, 105
Boar, 173–4
  accessory glands, 173
  external genitalia, 11
  frequency of use, 174
  infertility, 177–9, 273
    bleeding after service, 178
    clinical examination, 178–9
    inability to extrude penis, 178
    inability to fertilize, 178
    inability to mount, 177
    poor libido, 177
    reduction of sperm output, 178
  prepucial diverticulum, 173
  pseudohermaphrodites, 174
  puberty, 173
  semen collection, 174
  semen parameters, 174
Boar pen, 46–7
Bordetella spp., 206
Bordetella bronchoseptica, 16, 17, 74
Botulism, 136
Bowel oedema, 94–5
Brachyspira hyodysenteriae, 19, 29, 97
Brachyspira pilosicoli, 19
Breeding stock, 31
British Pig Executive, 21
British Pig Health Scheme, 22, 45
Bumble foot, 67–8
Burns, 124
Bush foot, 67–8
Butorphanol, 6, 231

C

Caesarean section, 171–2, 234–5
Campylobacter coli, 99
Cannibalism, 204
Capillary refill time, 7
Carbon monoxide poisoning, 154
Carcass disposal, 28–9
Carcass lymph nodes, 7
Cardiovascular disease, 143–7
  actinobacillosis, 144–5
  aetiology, 143
  clinical signs, 143–4
  control, 144
  diagnosis, 144
  epidemiology, 143
  Glasser's disease, 145
  mulberry heart disease, 145–7
  muscular dystrophy, 147
  swine erysipelas, 145
  tissue sampling, 245
  treatment, 144
  Veterinary Investigation Diagnosis Analysis, 144
Cardiovascular system
  haematology and blood biochemistry, 258
  post-mortem examination, 251–2
Castration, 235–6
Cefquinome, 228
Ceftiofur, 228
Cephalosporins, 222
Cerebellar lesions, 129
Cerebral lesions, 129
Cervical cord lesions, 130
Classical swine fever, 22, 89, 118, 132, 182–4
Clavulanic acid, 228
Claws
  broken, 67
  overgrown, 67
Cleaning, 29
Cleft palate, 85

Clinical investigation, 1–15
  environment, 2–3
  equipment, 1
  history of sick pig, 2
  observation, 3–4
  physical examination, 6–15
  restraint, 4–6
  signalment of pig, 2
  unit history, 1–2
Clostridial enteritis, 205
Clostridium novyi, 193, 203
Clostridium perfringens, 88, 205
  type A and B, 91
  type C (haemorrhagic enterotoxaemia), 89–91
Clostridium tetani, 128
Coagulopathies, 258
Coccidiosis, 102, 205–6
Code of Recommendations for the Welfare of Livestock, 40
Colibacillosis, 205
Colistin, 228
Colitis, 101
  spirochaetal, 99
Congenital tremor
  type A1, 133–4
  types A2–5 and type B, 134
Coughing, 4, 275
Cranial nerve lesions, 129
Creatinine kinase assay, 54
Crooked/kinky tail, 51
Cryptorchidism, 156
Cryptosporidia, 102–3
Cryptosporidium parvum, 102
Cystic kidneys, 156
Cystitis, 156–7

D

Danofloxacin, 228
Delayed puberty, 267
Delayed weaning to mating interval, 267, 269
Deliveries, 29
Demodectic mange, 117
Dermatosis vegetans, 114
Detomidine, 6
Diarrhoea, 4, 19
  porcine epidemic, 88
  spirochaetal, 99
Diazepam, 232
Differential diagnosis, 262–80
  conditions by age and system, 267, 268–9, 270–2
  mortality rates and intervention levels, 262, 263
  outcome of clinical examination, 262
  prevalence, 262, 263–5
  syndrome and sign, 267, 269, 272–6
  troubleshooting algorithms and component analysis, 276–80
  web sites, 262–7
    Consultant, 262
    Iowa State University Swine Site, 267
    Pigsite, 263, 266
Dihydrostreptomycin, 228
Disease control, 16
  programmes, 32
Disease, and pig performance, 16–21
Disinfection, 29
Diterpines, 222
Doramectin, 116
Droperidol, 54
Dust, 40–1
Dwarfism, 52
Dyspnoea, 70
Dystocia, 204

E

Ear, 8
Ear biting, 122–3
Ectoparasites, 32
  pet pigs, 217
  see also Mange; Louse infestation
Electrocution, 124
Employees, 28
Endoparasites, 32
  pet pigs, 217
Enrofloxacin, 228
Enteric colibacillosis, 2, 91–4
Enteric disease, 19–20
  sampling, 244
Enteritis
  clostridial, 205
  in piglets, 205
Entropion, 236
Environment, 45
Environmental comfort, 40
Environmental factors, 2–3
Enzootic pneumonia, 9, 71–4
  aetiology, 71–2
  clinical signs, 72–3
  control, 73–4
  diagnosis, 73
  differential diagnosis, 73
  epidemiology, 72
  incidence, 71
  treatment, 73
Eperythrozoonosis, 152
Eperythrozoon suis, 115
Epitheliogenesis imperfecta, 113–14
Equipment, 1
  security, 28
Ergot poisoning, 126, 160, 204
Erysipelothrix rhusiopathiae, 59
Escherichia coli, 19, 216
  see also Enteric colibacillosis
Euthanasia, 218, 243, 246
Exercising pet pigs, 215
Exophthalmus, 8
External genitalia, 11
  post-mortem examination, 249
Eyes, 8
  examination, 129

F

Faecal sampling, 243
Farrowing rates, 272
Fascioliasis, 204
Feed, 27, 30
  pet pigs, 213
Fertility
  herd, 179
  seasonal, 174
  see also Infertility
Fetal maldisposition, 169
Fetopelvic disproportion, 169–70
Fighting injuries, 124–5
Finishers
  poor growth rates, 275
  scours, 274–5
  sudden death, 275
Fitting, 4
Flank rubbing, 123
Florfenicol, 228
Flunixin, 231
Fly worry, 117
Foot
  care in pet pigs, 217–18
  lameness, 10
  lesions
    adult pigs, 67–9
    piglets, 67
  rot, 67–8
Foot and mouth disease, 62, 118, 185–6
Forelimb incoordination, 4
Fractures, 10, 63–4
  surgery, 236
Frenulum, persistent, 238
Frostbite, 124

G

Gait evaluation, 130
Gases, 40
Gastric torsion in sows, 108
Gastric ulceration
  fundic, 108
  pars oesophagea, 108
Gastroenteritis, transmissible, 86–7
Gastrointestinal disease, 83–111
  abdominal hernias, 109
  aetiology, 83
  aflatoxicosis, 109
  clinical signs, 83
  congenital and hereditary conditions, 85–6
    atresia ani (imperforate anus), 85
    cleft palate, 85
    macroglossus, 85
    short mandible, 85
  control, 84
  dealing with outbreaks, 103–4
  diagnosis, 84
  epidemiology, 83
  gastric ulceration
    fundic, 108
    pars oesophagea, 108
  helminth parasites, 109–11
    ascariasis, 110–11
    hepatosis cysticercosis, 111
    hyostrongylosis, 109–10
    liver fluke disease, 111
    lungworm disease, 111
    oesophagostomiasis, 110
    Trichuris suis, 111
    worm control programmes, 111
  hepatosis dietetica, 109
  infectious, 86–102
    bowel oedema, 94–5
    Campylobacter coli, 99
    Clostridium perfringens type A and B, 91
    Clostridium perfringens type C (haemorrhagic enterotoxaemia), 89–91
    colitis, 101
    enteric colibacillosis, 2, 91–4
    necrotic stomatitis, 101–2
    porcine epidemic diarrhoea, 88
    porcine proliferative enteritis complex, 99–100
    rotavirus, 88–9
    salmonellosis, 95–7
    spirochaetal colitis and diarrhoea, 99
    swine dysentery, 97–8
    swine fever, 22, 89
    transmissible gastroenteritis, 86–7
    vomiting and wasting disease, 87–8
    Yersinia infection, 100–1

intestinal haemorrhagic syndrome, 105
intestinal torsion, 108
intussusception, 109
protozoal, 102–3
coccidiosis, 102
cryptosporidia, 102–3
rectal prolapse, 9, 107–8
rectal stricture, 9, 106–7
stone swallowing, 109
thin sow syndrome, 104–5
treatment, 84
Veterinary Investigation Diagnosis Analysis, 84
Gastrointestinal tract, post-mortem examination, 253–5
General anaesthesia, 6
Glasser's disease, 4
cardiovascular, 145
musculoskeletal, 58
nervous system, 138
respiratory, 77–8
Granulomatous mastitis, 204
Greasy pig disease (exudative epidermitis), 118–19, 206
Growers
poor growth rates, 275
scours, 274–5
sudden death, 275

H

Haematology, 257–8
coagulopathies, 258
diseases affecting blood, 257
diseases causing anaemia, 257
leucocytosis and leucopenia, 257
plasma proteins, 258
platelets, 257–8
Haematoma, 3, 8
aural, 125, 234
vulva, 10
Haematopinus suis, 8, 114–5, 216
Haemolytic disease of newborn, 149–50
Haemophilus parasuis, 4, 10, 16, 17, 77, 128, 206
Haemopoietic disease, 148–54
carbon monoxide poisoning, 154
eperythrozoonosis, 152
haemolytic disease of newborn, 149–50
iron deficiency anaemia, 148–9
monensin poisoning (myoglobinuria), 153
navel bleeding in piglets, 151
nitrite/nitrate poisoning (methaemoglobinuria), 154
purpura haemorrhagica, 151
thrombocytopenic purpura, 150–1
vitamin B_1 deficiency, 152–3
von Willebrand's disease, 151–2
warfarin poisoning, 153
Halothane sensitivity test, 54
Head
examination, 8, 129
in piglets, 13
Head tilt, 4, 138–9
Heart, 8–9
post-mortem examination, 251–2
see also Cardiovascular system
Heat stress in outdoor pig units, 201
Heatstroke, 123–4
Hepatitis, necrotic, 203
Hepatosis cysticercosis, 111
Hepatosis dietetica, 109
Herd location, 23
Hernia, 109
inguinal, 236
scrotal, 3
umbilical, 3, 237
Hog cholera *see* Classical swine fever
Hoof trimming, 237
Hosepipe gut, 100
Hospital pens, 48
Housing, 198
pet pigs, 215
see also Outdoor pig units
Hyostrongylosis, 109–10
Hyostrongylus rubidus, 105
Hyperpnoea, 70
Hypoglycaemia, 139
Hypotrichosis (hairless piglets), 114

I

Ileitis, 19, 100
Immunocompetence, 223
Imperforate anus *see* Atresia ani
Inclusion body rhinitis, 80–1
Indoor herds, 45–8
boar pen, 46–7
dry sow yard, 46
farrowing area, 45–6
growers and finishers, 47–8
treatment and hospital pens, 48
weaners, 47
Infertility, 9–14
boar, 177–9, 273
bleeding after service, 178
clinical examination, 178–9
inability to extrude penis, 178
inability to fertilize, 178
inability to mount, 177
poor libido, 177
reduction of sperm output, 178
seasonal, 201–2
sow, 174–8
functional, 174–5
infectious, 175–6
leptospirosis, 176
porcine parvovirus, 176–7
porcine reproductive and respiratory syndrome, 177
SMEDI viruses, 177
Veterinary Investigation Diagnosis Analysis, 176
tissue sampling, 246
Inguinal hernia, 236
Inherited thick forelegs, 52
Intestinal haemorrhagic syndrome, 105
Intestinal parasites, 20
outdoor pig units, 204
see also Gastrointestinal disease
Intestinal torsion, 108
Intestines
haematology and blood biochemistry, 258
see also Gastrointestinal disease
Intraherd security, 29
Intussusception, 109
Iron deficiency anaemia, 148–9
Isospora suis, 102
Isowean systems, 16

J

Joint disease, 10
Joint ill, 60
outdoor pig units, 206
Joint lavage, 237

K

Ketamine, 6, 232
Ketoprofen, 231
Kidney
cystic, 156
post-mortem examination, 255
Kyphosis, 52

L

Lameness, 4, 9–10, 275–6
infectious causes, 56–63
outdoor pig units, 202–3
piglets, 13
see also Musculoskeletal disease
Laminitis, 68
Laparotomy, exploratory, 236
Lawsonia intracellularis, 19, 27, 100
Legless piglets, 51
Leg weakness syndrome *see* Osteochondrosis
Leptospira bratislava, 27, 202
Leptospirosis, 193–4
and infertility, 176
Leucocytosis, 257
Leucopenia, 257
Lincomycin, 228
Lincosamides, 222
Lincospectinomycin, 228
Litter savaging, 162
Litter size, 273
Liver
haematology and blood biochemistry, 258
post-mortem examination, 252
Liver fluke disease, 111
Loading, 31
Louping ill, 133
Louse infestation, 114–15
Lower motor neuron lesions, 129
Lungs, 9
haematology and blood biochemistry, 258
post-mortem examination, 251–2
Lungworm disease, 111
Lymphatic disease, 147–8
Lymph nodes, 7
post-mortem examination, 250
Lymphosarcoma, 147

M

Macroglossus, 85
Macrolides, 221–2
Malignant catarrh fever, 187
Malignant hyperthermia, 53
Mammary aplasia, 161
Mammary gland *see* Udder; Udder disease
Mange
demodectic, 117
outdoor pig units, 203
sarcoptic, 4, 20, 115–16
Mannheimia haemolytica, 17
Manure deposits, 198–9
Marbofloxacin, 228
Mastectomy, 237–8
Mastitis, 10–11
acute, 163–4
chronic, 165
granulomatous, 204
outdoor pig units, 204

Mastitis-metritis-agalactia syndrome (MMA), 10, 164–5
Mating, 166–7
Medetomidine, 232
Medicines, 32
Meloxicam, 231
Meningitis, 20
Meningoencephalocele, 134–5
Metastrongylosis, 82
Metastrongylus apri, 82
Metritis, 157–8
  acute septic, 173
  mastitis-metritis-agalactia syndrome, 10, 164–5
Midazolam, 232
Middle ear disease, 138–9
Milk let-down reflex, failure of, 162
Milk, poor production, 274
Monensin poisoning (myoglobinuria), 153
Morphine, 231
Mouth, 8
Movements, 31
Mucous membranes, 7
Mulberry heart disease, 145–7
Muscle
  damage, 10
  swelling, 3
Muscle injuries, 64
Muscular dystrophy, 64
Musculoskeletal disease, 49–69
  aetiology, 49
  clinical examination, 49
  clinical signs, 49
  congenital and hereditary, 50–6
    arthrogryposis, 51
    asymmetrical hindquarters, 53
    congenital porphyria, 52
    crooked/kinky tail, 51
    dwarfism, 52
    inherited thick forelegs, 52
    kyphosis and scoliosis, 52
    legless piglets, 51
    osteochondrosis, 54–5
    pietrain creeper syndrome, 52
    polydactyly, 51
    porcine stress syndrome, 53–4
    slipped epiphysis of femoral head, 55–6
    splay leg, 50–1
    syndactyly, 51
  control, 50
  diagnosis, 50
  epidemiology, 49
  foot lesions, 67–9
  fractures, 63–4
  infectious causes, 56–63
    Actinobacillus suis, 59
    chronic septic arthritis, 61–2
    foot and mouth disease, 62
    Glasser's disease, 58
    joint ill in neonatal piglets, 60
    Mycoplasma hyorhinis, 56–7
    Mycoplasma hyosynoviae, 57
    Streptococcus suis type 1, 58
    Streptococcus suis types 2 and 14, 58–9
    swine erysipelas, 59–60
    swine vesicular disease, 62–3
  muscle injuries, 4
  muscular dystrophy, 64
  osteitis and proliferative osteitis, 66
  osteochondrosis, 10, 54–5, 101
  osteodystrophia fibrosa, 65–6
  osteomalacia and osteoporosis, 65
  pressure-induced, 66–7
  rickets, 65
  treatment, 50
  Veterinary Investigation Diagnosis Analysis, 50
Musculoskeletal system, post-mortem examination, 255
Mycobacterium avium-intracellulare tuberculosis, 196
Mycobacterium bovis tuberculosis, 196–7
Mycoplasma hyopneumoniae, 16, 17, 71–2, 206
Mycoplasma hyorhinis, 10, 56–7
Mycoplasma hyosynoviae, 10, 20, 57
Mycoplasmal arthritis, 201
Mycotoxic nephropathy, 160
Mycotoxins, 159–60

N

National Pig Association, 21
Navel bleeding, 151
Neck
  examination, 8
  in piglets, 13
Necrotic hepatitis, 203
Necrotic stomatitis, 101–2
Neomycin, 228
Nervous signs, 275
Nervous system disease, 128–42
  aetiology, 128
  aflatoxicosis, 142
  bacterial, 135–9
    botulism, 136
    Glasser's disease, 138
    middle ear disease, 138–9
    Streptococcus suis, 136–8
    tetanus, 135
  clinical signs, 128
  congenital, 133–5
    meningoencephalocele, 134–5
    tremor type A1, 133–4
    tremor types A2–5 and type B, 134
  control, 130–1
  degenerative, 139–40
  diagnosis, 128
  epidemiology, 128
  injury, 139–40
  metabolic, 139
  selenium toxicity, 141–2
  treatment, 130
  Veterinary Investigation Diagnosis Analysis, 131
  viral, 131–3
    Aujeszky's disease (pseudorabies), 132
    louping ill, 133
    rabies, 132–3
    swine fever, 132
    Talfan disease, 131
    Teschen disease, 131
    vomiting and wasting disease, 131–2
  vitamin deficiency, 141
  water deprivation (salt poisoning), 140
Neurological examination, 128–30
Neurological signs, 4
Nicotinic acid deficiency, 121
Nipples, 13
  inversion, 162
  necrosis, 162
  see also Udder; Udder disease
Nitrite/nitrate poisoning (methaemoglobinuria), 154
Normal values, 259–60
  adults, 6
  piglets, 6
  pot-bellied pigs, 216
Notifiable diseases, 281
Nystagmus, 8

O

Obstetrics and reproduction, 166–79
  normal reproduction, 166–8
    mating, 166–7
    normal birth, 167
    sow, 168
    synchronization and induction of birth, 168
  porcine dystocia, 168–72
    aftercare of sow, 172
    causes, 168
    deviation of uterus, 169
    end of farrowing, 171
    fetal maldisposition, 169
    fetopelvic disproportion, 169–70
    indications for caesarean section, 171–2
    management, 170–1
    maternal excitement, 169
    obstruction of birth canal, 169
    signs of, 170
    uterine inertia, 168–9
  postparturient problems in sow, 172–3
    acute septic metritis, 173
    agalactia, 173
    recumbency, 172
    retention of fetal membranes, 172
    uterine prolapse, 172–3
    vaginal prolapse, 172
  see also Infertility
Ochratoxicosis, 160
Ocular discharge, 8
Oesophagostomiasis, 110
Oesophagostomum spp., 105
Oestrous cycle, 166
Oestrus, 166
Office International des Épizooties lists, 282
Organic pig production, 207–9
  aims of, 207
  conversion, 207
  organic certification, 207
  organic standards, 208–9
  see also Outdoor pig units
Osteitis, 66
  proliferative, 66
Osteochondrosis, 10, 54–5
  outdoor pig units, 201
Osteodystrophia fibrosa, 65–6
Osteomalacia, 65
Osteoporosis, 65
Outdoor pig units, 31, 198–211
  characteristics, 198
  checklist, 209–11
  clinical conditions, 200–7
    abdominal catastrophes, 203
    farrowing sows, 204
    intestinal parasites, 204
    lameness, 202–3
    necrotic hepatitis, 203
    outdoor weaners, 206–7
    piglets, 204–6
    pyelonephritis, 203
    replacement breeding stock, 200–1
    reproductive performance, 202

skin conditions, 203–4
sows, 201–2
paddock systems and housing, 198
site management, 200
site selection, 198–200
veterinary health plans, 207
see also Organic pig production
Outdoor weaners, 206
Ovariohysterectomy, 238
Ovary, post-mortem examination, 255
Overlying, 205
Oxytocin, 168

P

Paddocks, 198
Panniculus test, 130
Pantothenic acid deficiency, 121, 141
Paracetamol, 231
Parakeratosis, 121
Parvovirus, 22
Pasteurella multocida, 15, 16, 17, 72, 78–9
Patellar reflex, 130
Pathogens, routes of transfer, 24–6
Pedal withdrawal reflex, 130
Penicillins, 221
Penis
damage to, 201
prolapse, 238
sucking, 123
Pentobarbital, 232
Perimetery security, 23
Perineum, 9
in piglets, 13
Peripheral nerve lesions, 130
Peritoneal cavity, post-mortem examination, 252–3
Peritonitis, 9
Pet pigs, 212–19
behaviour, 213–14
blood sampling, 218
chemical restraint and anaesthesia, 218
clinical examination, 215–16
handling, 215
history, 215
normative values, 216
physical examination, 215–16
diseases, 216–17
endoparasite/ectoparasite control, 217
euthanasia, 218
exercise, 215
feed, 213
feeding, 214
foot care and tusk trimming, 217–18
housing, 215
identification rules, 213
legal requirements, 212–13
nutritional deficiencies, 214–15
pig-walking licences, 213
reproduction, 214
surgical procedures, 218
therapeutics, 217
vaccination, 217
Phenoxymethyl penicillin, 228
Photosensitization, 204
Physical examination, 6–15
adult and growing pigs, 6–11
sow and litter, 11–13
Phytophotodermatitis, 125–6
Pietrain creeper syndrome, 52
Pig board, 4, 5
Piglets
abdomen, 13
behaviour, 11–12
carboxyhaemoglobin in, 154
chest, 13
enteritis, 205
foot lesions, 67
hairless (hypotrichosis), 114
head and neck, 13
health, 46
hypoglycaemia, 139
joint ill, 60
legless, 51
navel bleeding, 151
outdoor pig units, 204–5
overlying, 205
physical examination, 11–13
post-mortem examination, 246–7
skin, 12–13
starvation, 205
sunburn, 206
tail necrosis, 13, 123
Pig pox, 117–18
Pig units, history, 1–2
Pig-walking licences, 213
Pityriasis rosea, 8, 127
Plasma proteins, 258
Platelets, 150, 257–8
Pleura, post-mortem examination, 251
Pleuropneumonia, 9, 76–7
Pneumonia, enzootic, 9, 71–4
Polydactyly, 51
Polymerase chain reaction test, 54
Polysystemic diseases, 180–97
aetiology, 180
African swine fever, 184–5
anthrax, 192
classical swine fever, 22, 89, 118, 132, 182–4
clinical signs, 181
Clostridium novyi infection, 193
control, 181
diagnosis, 181
epidemiology, 180
foot and mouth disease, 185–6
leptospirosis, 193–4
malignant catarrh fever, 187
porcine dermatitis and nephropathy syndrome, 16, 19, 118, 157, 190–2, 266
porcine reproductive and respiratory syndrome, 16, 17, 70–1, 81, 177, 187–8
postweaning multisystemic wasting syndrome, 16, 19, 82, 188–90
swine erysipelas, 8, 59–60, 118, 145, 194–6
swine vesicular disease, 118, 186–7
treatment, 181
tuberculosis, 196–7
Poor growth rates, 275
Population medicine, 16–48
Porcine circovirus 2, 17
see also Porcine dermatitis and nephropathy syndrome; Postweaning multisystemic wasting syndrome
Porcine dermatitis and nephropathy syndrome (PDNS), 16, 19, 118, 157, 190–2, 266
Porcine dystocia, 168–72
aftercare of sow, 172
causes, 168
deviation of uterus, 169
end of farrowing, 171
fetal maldisposition, 169
fetopelvic disproportion, 169–70
indications for caesarean section, 171–2
management, 170–1
maternal excitement, 169
obstruction of birth canal, 169
signs of, 170
uterine inertia, 168–9
Porcine epidemic diarrhoea, 88
Porcine parvovirus, 176
Porcine pasteurellosis, 78–9
Porcine proliferative enteritis complex, 99–100
Porcine reproductive and respiratory syndrome (PRRS), 16, 17, 70–1, 81, 177, 187–8
Porcine respiratory coronavirus, 82
Porcine respiratory disease complex, 18
Porcine stress syndrome, 53–4
Porphyria, congenital, 52, 114
Post-mortem examination, 247–56
external carcass, 247–9
suckling piglet, 249
weaner, grower and adult, 249
gastrointestinal tract, 253–5
heart and lungs, 251–2
kidney and bladder, 255
liver, 252
lymph nodes, 250
mammary gland and external genitalia, 249
musculoskeletal system, 255
peritoneal cavity, 250, 252–3
peritoneum, 250
pleura, 251
positioning of carcass, 250
skull, 255–6
thoracic cavity and pericardium, 250
uterus, vagina and ovaries, 255
Posture, 130
Postweaning multisystemic wasting syndrome (PMWS), 16, 19, 82, 188–90
Pot-bellied pigs *see* Pet pigs
Predation, 206
Pregnancy
diagnosis, 167
failure to maintain, 273
vaginal discharge, 158
see also Obstetrics and reproduction
Prepucial diverticulitis, 158–9
Prepucial diverticulum, 173
distension, 3
removal, 238
Pressure-induced problems, 66–7
Pressure injuries, 126
Preweaning deaths, 274
Procaine penicillin/dihydrostreptomycin, 228
Production systems, 16
Progressive atrophic rhinitis, 74–6
Prolapse
penis, 238
prepuce, 238–9
rectal, 9, 107–8, 239
uterine, 172–3, 240
vaginal, 172, 239–40
Proliferative enteropathy, 100
Proprioception, 130
Pruritus, 4
Pseudohermaphroditism, 156, 174
Pseudorabies, 22, 132
Puberty in sow, 166
Pulse, 7
Purpura haemorrhagica, 151
Pyelonephritis, 9, 156–7
outdoor pig units, 203

R

Rabies, 132–3
Rectal examination, sow, 11
Rectal prolapse, 9, 107–8
  surgery, 239
Rectal stricture, 9, 106–7
Renal agenesis, 156
Replacement and new stock, 22–3, 274
Reproduction *see* Obstetrics and reproduction
Residue monitoring, 31
Respiratory disease, 70–82
  aetiology, 70
  clinical signs, 70–1
  control, 71
  diagnosis, 71
  enzootic pneumonia, 71–4
  epidemiology, 70
  Glasser's disease, 77–8
  inclusion body rhinitis, 80–1
  metastrongylosis, 82
  outdoor pig units, 206–7
  and performance, 17–19
  pleuropneumonia, 76–7
  porcine pasteurellosis, 78–9
  porcine reproductive and respiratory syndrome, 16, 17, 81
  porcine respiratory coronavirus, 82
  postweaning multisystemic wasting syndrome, 16, 19, 82
  progressive atrophic rhinitis, 74–6
  sampling, 245
  swine influenza, 79–80
  treatment, 71
  Veterinary Investigation Diagnosis Analysis, 72
Respiratory rate, 7
Respiratory system, 4
Restraint, 4–6
Retention of fetal membranes, 172
Return to service, 269, 272
Rhinitis
  inclusion body, 80–1
  progressive atrophic, 74–6
Riboflavin deficiency, 121
Rickets, 65
Rickety rosary, 65
Ringworm, 8, 119–20
  outdoor pig units, 203–4
Rotavirus, 88–9
Routine (advisory) visits, 41–5
Runts, 3

S

Salmonella spp., 19
Salmonella cholerae-suis, 96, 118
Salmonella typhimurium, 19, 96
Salmonellosis, 95–7
Salt poisoning, 140
Sampling, 242–6
  aerobic bacteriology, 243
  anaerobic bacteriology, 243
  biochemical/toxicological analysis, 243
  blood samples, 242
  cytology, 243
  faeces and skin, 243
  packing and sending, 242
  selection of animals, 242
  tissues, 243
Sarcoptes scabiei, 8, 20, 113
Sarcoptic mange, 4, 20, 115–16
Scoliosis, 52
Scrotal hernia, 3
Sedation, 6, 232
Selenium toxicity, 126, 141–2
Semen collection, 174
Semen parameters, 174
Short mandible, 85
Skeletal muscle, 259
Skin, 4
  colour, 7–8
  discoloration, 4
  examination, 7
  haematology and biochemistry, 259
  injuries, 8
  lesions, 8
  piglets, 12–13
  sampling, 243
  temperature, 7
  texture, 8
Skin diseases, 112–27, 275
  aetiology, 112
  bacterial, 118–19
    greasy pig disease (exudative epidermitis), 118–19
    salmonella cholerae-suis, 118
    spirochaetal granuloma, 118
    swine erysipelas, 8, 59–60, 118
  clinical signs, 112
  control, 113
  diagnosis, 112–13
  epidemiology, 112
  fungal, 119–20
  inherited, 113–14
    congenital porphyria, 114
    dermatosis vegetans, 114
    epitheliogenesis imperfecta, 113–14
    hypotrichosis (hairless piglets), 114
  parasitic, 114–17
    demodectic mange, 117
    fly worry, 117
    louse infestation, 114–15
    sarcoptic mange, 4, 20, 115–16
  physical causes, 121–6
    aural haematoma, 125
    burns, 124
    electrocution, 124
    fighting injuries, 124–5
    flank rubbing and penis sucking, 123
    frostbite, 124
    heatstroke, 123–4
    phytophotodermatitis, 125–6
    pressure injuries, 126
    sunburn, 123
    tail and ear biting, 122–3
    tail necrosis, 13, 123
    teat necrosis, 123
  pityriasis rosea, 8, 127
  toxic causes, 126
  treatment, 113
  Veterinary Investigation Diagnosis Analysis, 113
  viral, 117–18
    classical swine fever, 118
    foot and mouth disease, 118
    pig pox, 117–18
    porcine dermatitis and nephropathy syndrome, 16, 19, 118
    swine vesicular disease, 118
  vitamin- and mineral-related abnormalities, 120–1
    parakeratosis and zinc deficiency, 121
    vitamin A deficiencies, 120
    vitamin B deficiency, 120–1
Skin sensitivity test, 129
Skull, post-mortem examination, 255–6
Slipped epiphysis of femoral head, 55–6
SMEDI viruses, 177
Snares, 4, 5
Snout, 8
  deviation, 3, 74
Sow
  behaviour, 11–12
  breeding life, 166
  gastric torsion, 108
  health, 46
  infertility, 174–8
    functional, 174–5
    infectious, 175–6
    leptospirosis, 176
    porcine parvovirus, 176–7
    porcine reproductive and respiratory syndrome, 177
    SMEDI viruses, 177
    Veterinary Investigation Diagnosis Analysis, 176
  litter savaging, 162
  oestrous cycle, 166
  oestrus, 166
  outdoor pig units, 201–2
  physical examination, 11–13
  puberty, 166
  rectal examination, 11
  thin sow syndrome, 104–5
  see also Obstetrics and reproduction
Sow condition scoring, 39–40
Sow unwell, 162
Space requirements, 30
Spectinomycin, 222, 228
Spirochaetal colitis and diarrhoea, 99
Spirochaetal granuloma, 118
Splay leg, 50–1
Staphylococcus hyicus, 13, 118
Starvation, 205
Stillbirth, tissue sampling, 246
Stomach
  haematology and blood biochemistry, 258
  see also Gastrointestinal disease
Stone swallowing, 109
Streptococcus suis, 4, 10, 17, 20, 128, 136–8
  type 1, 58, 136–7
  types 2 and 14, 58–9, 137–8
Sudden death, 276
Sulphonamides, 221
Sunburn, 4, 123
  outdoor pig units, 201, 206
  in piglets, 206
Surgical procedures
  amputation
    digital, 233
    uterus, 233
  anaesthesia *see* Anaesthesia
  atresia ani, 233–4
  aural haematoma, 234
  bladder retroversion, 234
  caesarean section, 234–5
  castration, 235–6
  entropion, 236
  exploratory laparotomy, 236
  fractures, 236
  hernia
    inguinal, 236
    umbilical, 237
  hoof trimming, 237
  joint lavage, 237
  mastectomy, 237–8
  ovariohysterectomy, 238
  persistent frenulum, 238
  prepucial diverticulum removal, 238

prolapse
penile, 238
prepuce, 238–9
rectal, 239
uterus, 240
vaginal, 239–40
tail docking, 240
tooth clipping, 240–1
tusk trimming, 241
undescended testicles, 241
vasectomy, 241
Swellings, muscle, 3
Swine dysentery, 2, 97–8
Swine erysipelas, 8, 59–60, 118, 145, 194–6
acute, 195
chronic, 195
outdoor pig units, 201
peracute, 194–5
Swine influenza, 17, 79–80
Swine vesicular disease, 118, 186–7
Syndactyly, 51

T

Tail biting, 122–3
Tail docking, 240
Tail holding, 4, 5
Tail necrosis, 13, 123
Talfan disease, 131
Teat necrosis, 123
Temperature, 7, 30
Teschen disease, 4, 130, 131
Tetanus, 135, 206
Tetracyclines, 221, 228
Thin sow syndrome, 104–5
Thiopental, 232
Thoracolumbar cord lesions, 130
Thrombocytopenic purpura, 150–1
Tiamulin, 228
Tilmicosin, 228
Tissue sampling, 243
Tolfenamic acid, 231
Tooth clipping, 240–1
Transmissible gastroenteritis, 86–7
Transport, 27–8, 31
Treatment pens, 48
Trichophyton mentagrophytes, 8, 119, 203
Trichophyton verrucosum, 119
Trichuris suis, 111
Trimethoprim, 222
Trimethoprim/sulphonamide, 228
Tuberculosis, 196–7
Mycobacterium avium-intracellulare, 196
Mycobacterium bovis, 196–7
Tulathromycin, 228
Turkey egg kidney, 183
Tusk trimming, 217–18, 241
Tylosin, 228

U

Udder, 10–11
conformation, 4
post-mortem examination, 249
see also Nipples
Udder disease, 161–5
acute mastitis, 163–4
aetiology, 161
agalactia, 161–2
chronic mastitis, 165
clinical signs, 161
control, 161
diagnosis, 161
epidemiology, 161
mastitis-metritis-agalactia syndrome, 164–5
treatment, 161
Veterinary Investigation Diagnosis Analysis, 161, 162
Umbilical hernia, 3
surgery, 237
Undescended testicles, 241
Upper motor neuron lesions, 129
Urinary tract, 9
haematology and biochemistry, 259
Urogenital disease, 155–60
aetiology, 155
clinical signs, 155
control, 155
cystitis and pyelonephritis, 156–7
diagnosis, 155
epidemiology, 155
hereditary abnormalities, 156
metritis, 157–8
mycotoxins, 159–60
porcine dermatitis and nephropathy syndrome, 16, 19, 118, 157
prepucial diverticulitis, 158–9
treatment, 155
urolithiasis, 157
vaginal discharge in pregnancy, 158
Veterinary Investigation Diagnosis Analysis, 156
vulval damage, 158
Urolithiasis, 157
Uterine inertia, 168–9
Uterine prolapse, 172–3
surgery, 240
Uterus, post-mortem examination, 255

V

Vaccination, 32
pet pigs, 217
Vaginal discharge, 10, 158
Vaginal prolapse, 9, 172
surgery, 239–40
Vagina, post-mortem examination, 255
Valnemulin, 228
Vasectomy, 241
Ventilation, 30
Vestibular lesions, 129
Veterinary health plans, 31–9
aims of, 31
disease control programmes, 32
ectoparasites and endoparasites, 32
formulation of, 31
medicines, 32
organic pig farms, 209
outdoor pig units, 207
production and target figures, 33–4
recording, monitoring and reviewing, 32
Veterinary Investigation Diagnosis Analysis
cardiovascular disease, 144
gastrointestinal disease, 84
infertility, 176
musculoskeletal disease, 50
nervous system disease, 131
respiratory disease, 72
skin diseases, 113
udder disease, 161, 162
urogenital disease, 156
Veterinary practices, 31
Visitors, 28
Vitamin A deficiency, 120, 141
Vitamin B deficiency, 120–1
Vitamin B_1 deficiency, 152–3
Vomiting and wasting disease, 87–8, 131–2
Von Willebrand's disease, 151–2
Vulva, 10
haematoma, 10
Vulval biting, 204
Vulval damage, 158
Vulvovaginitis, 159

W

Warfarin poisoning, 153
Water, 27, 30, 41
Water deprivation, 140
Water intake, 229
Waterlogging, 198
Weaners, 47
deaths, 274
scours, 274
Wheelbarrow test, 130
Wheezing, 9
Whey bloat, 105
Wildlife, 23–7
Worm control programmes, 111

X

Xylazine, 231, 232

Y

Yersinia spp., 100–1
Yersinia enterocolitica, 101
Yersinia pseudotuberculosis, 19, 101

Z

Zearalenone poisoning, 159
Zinc deficiency, 121
Zoonosis action plan, 22, 31, 45