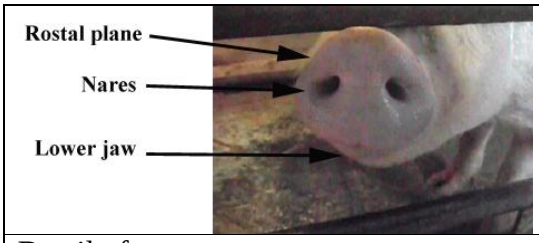
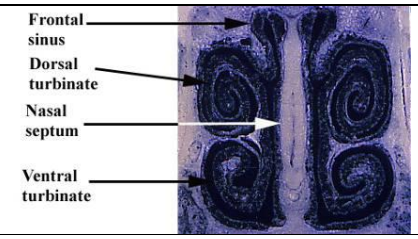
Disorders of the chest

Disorder present in	Australia	Europe/Asia	North America
Clinical anatomy of the chest			
Actinobacillus pleuropneumonia	Yes	Yes	Yes
Actinobacillus suis	Yes	Yes	Yes
Enzootic (Mycoplasma) pneumonia	Yes	Yes	Yes
Glasserôs Disease	Yes	Yes	Yes
Mulberry Heart	Yes	Yes	Yes
Pneumonia Pasteurellosis and Streptococci	Yes	Yes	Yes
Porcine Reproductive and Respiratory Syndrome Virus	No	Yes	Yes
Porcine Respiratory Coronavirus	No	Yes	No
Post-weaning sneezing ï see chapter on head	Yes	Yes	Yes
Salmonellosis ï see chapter on intestinal tract	Yes	Yes	Yes
Swine Influenza Virus	No (yes)	Yes	Yes

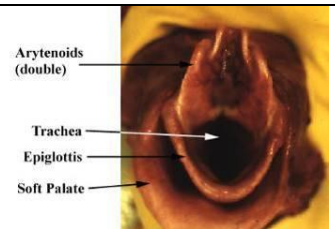
CLINICAL GROSS ANATOMY OF THE CHEST
-RESPIRATORY TRACT AND HEART



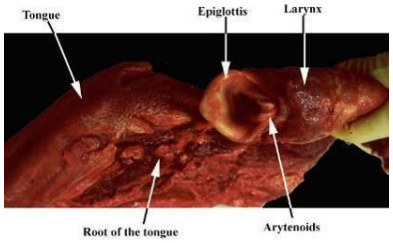
Detail of nose



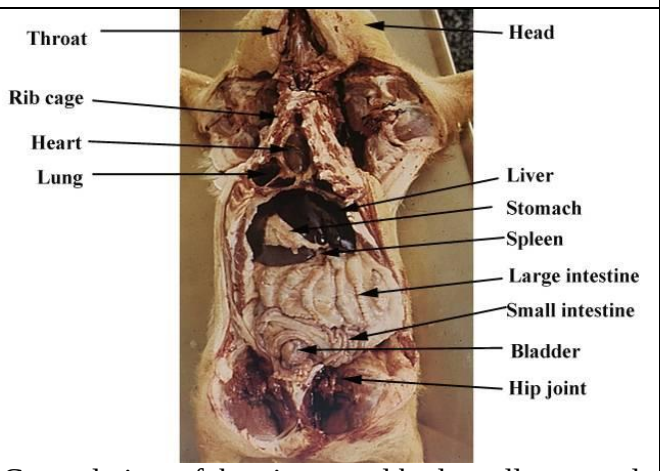
Cross-section of the nose



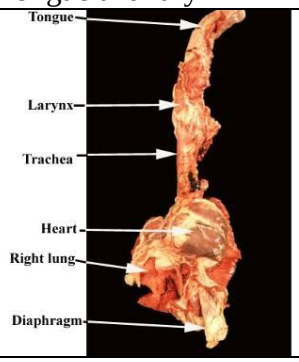
Entrance to larynx



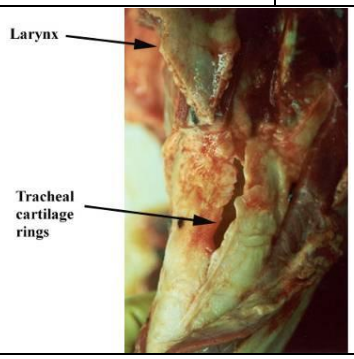
Tongue and larynx



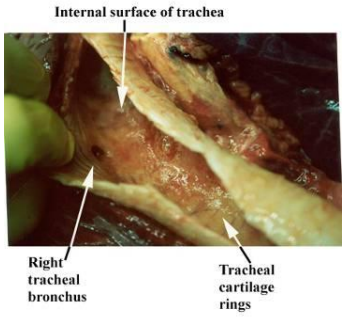
General view of the pig ventral body wall removed



The pig respiratory tract

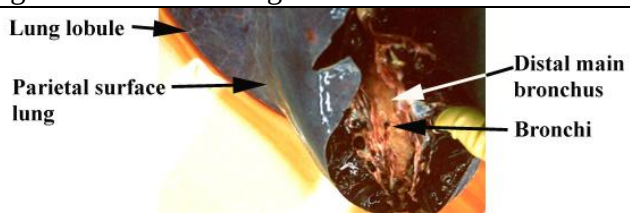


Detail of tracheal rings

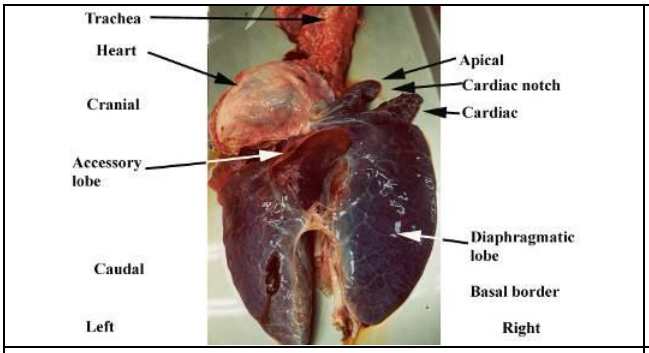


Detail of right tracheal bronchus

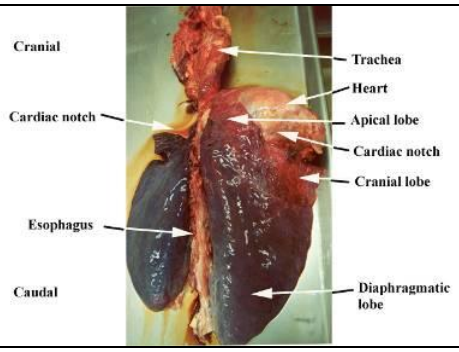
Chest cavity, ventral wall removed



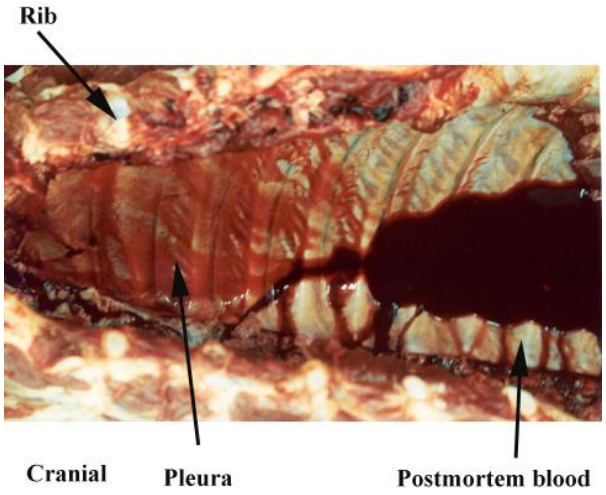
Detail of end of bronchi cut surface



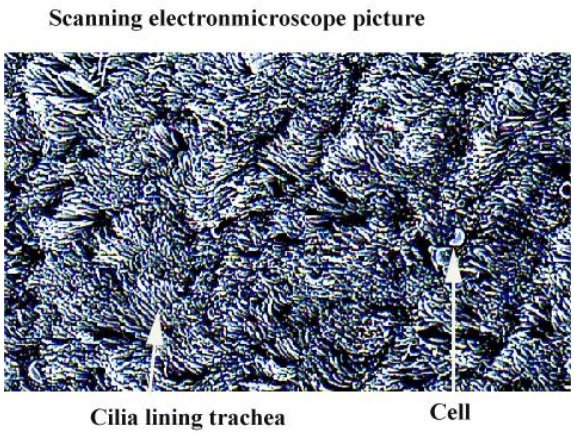
Lungs dorsal view



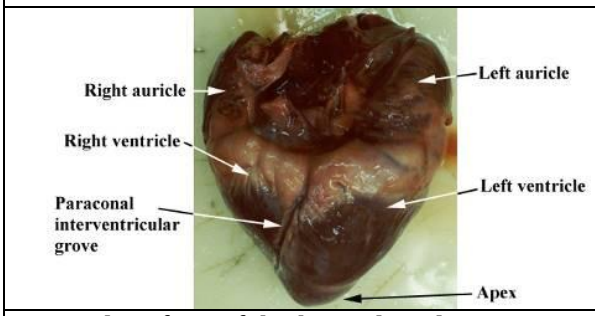
Lung ventral view



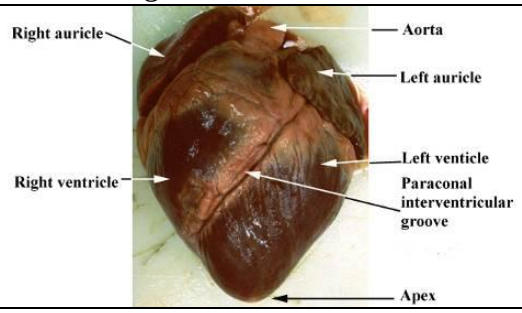
Detail of pleural surface



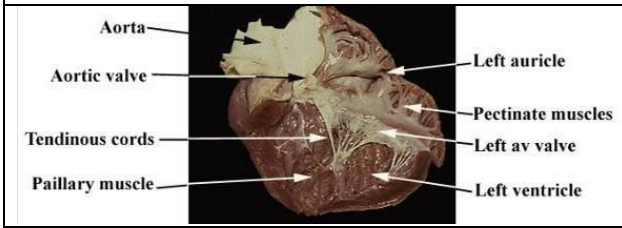
Scanning electronmicroscope view of surface of trachea showing the cilia



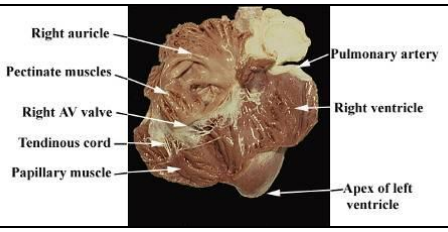
Parietal surface of the heart dorsal view



Parietal surface of the heart ventral view



Heart opened, left side



Heart opened, right side.

ACTINOBACILLUS PLEUROPNEUMONIA

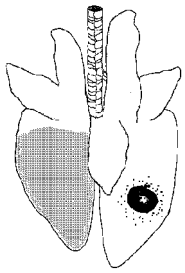
Other names	APP, Haemophilus Pleuropneumonia, HPP
Causal agent	*Actinobacillus pleuropneumoniae* a bacteria. There are currently 15 varieties (serovars) described. The severities of the clinical signs differ between the different serovars and presence of other factors. Serovars 1, 5, 9, 10 and 11 are considered to be more virulent than others but there are a lot of local variations. Serovars 3, 6 and 8 predominate in the UK. In the US 1, 5 and 7 most important. Cross reactions with 1, 9 and 11; 2, 6 and 8; 4 and 7. Note *Actinobacillus suis, A. porcitonsillarum* and *A. rossi* may also cause some cross-reaction complicating diagnosis. PCR will help classification
Age group	All ages are susceptible, mostly 20 to 100 kg pigs show the disease on farms
Clinical signs (varying from death to nothing)	
Peracute 	Sudden illness. May occur in individuals only. The animals lie down a lot and have a high rectal temperature of 41.5ÅC. The animals are anorexia and generally do not drink. Their extremities become cyanotic (purple) and are cold to the touch. The cyanosis can spread to the whole of the body. Severe breathing difficulties, often with mouth breathing, develop before the animal dies. Blood stained froth can be seen at the mouth and nostrils. Death generally occurs within 24 to 48 hours
Acute	Several animals in the group (may be 70%) present depressed and lie down a lot. They have a high rectal temperature of 40.5 to 41ÅC. They are anorexia and often do not drink either. The animals present with severe respiratory distress often with a cough and mouth breathing. Cyanosis is apparent, particularly if the animal is asked to move. Vomiting may be seen. Abortion can occur in sows
Subacute	There can be an intermittent cough in a group of pigs. There is a general loss of appetite resulting in reduced growth. With serovar type 3 arthritis, endocarditis (heart valve lesions) and abscesses may be seen
Chronic	In chronically affected herds (most herds), daily weight gain may not be affected. An intermittent cough may be heard
Implications of the disease	
	From 15 to 30% of pigs are affected in a group and at times up to 100% may die. Depending on the severity of the disease daily gain may be reduced by up to 30%
Infectivity	
	The disease is mainly spread by pig to pig contact
	Airborne spread via droplets is possible over a short distance
	Survival in the environment is very short unless protected by mucus or organic material, when it can be extended to a few days
	Recovered pigs can act as carriers
Stress factors	
	Moving pigs
	Mixing pigs
	Overcrowding
	Rapid changes in temperature, insufficient ventilation, high Relative Humidity
	Taking blood and other tests

Incubation period

	Very variable, high exposure can result in death within a few hours, other cases can take a few days

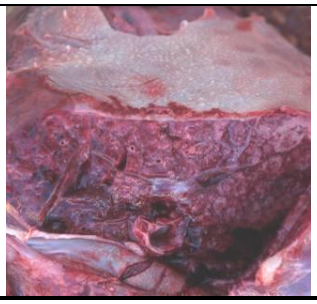
Post-mortem Lesions

	Most of the lesions are confined to the respiratory tract. In the peracute and acute cases there is the presence of well demarcated focal dark and solid pneumonic areas particularly in the caudal area of the lung, but lesions can be found also in the cranial lobe. Lesions may have a red haemorrhagic outer edge and a necrotic (dead) centre. Pleurisy is generally very obvious and may be seen adherent to the lesion. Fluid around the heart (pericardial effusion) may also be seen. In the subacute form, there may be pleurisy with no other lung lesions In the chronic form, there may be a few individuals with pleurisy, but most pigs demonstrate no gross changes. There are three known toxins ApxI strong haemolysin and cytotoxic, ApxII is weak haemolysin and mildly cytotoxic and ApxIII is a strong cytotoxin

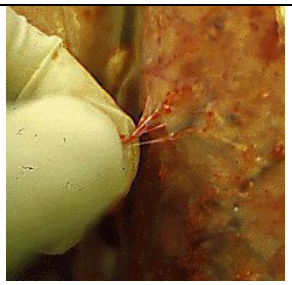


Distribution of the lesions

Gross appearance of the lung



Cross section of the lung showing the yellow necrotic areas



Pleurisy

In the drawing the shaded area indicates the area of lung generally diseased by pleuropneumonia. Illustration of an acute pleuropneumonia lesion

Diagnosis

	Bacteriological confirmation needed in peracute, acute and subacute cases.
	Pleurisy can occur through other diseases, notably Glasser's.
	Detection of antibodies generally unrewarding clinically, but may be worth considering when needing to tract the disease through a herd. Note pigs can go serologically negative after a while.

Treatment

Individuals affected	Individual Antimicrobial injections, but you must be vigorous and quick in peracute and acute outbreaks. Treat other pigs in the same area as well. Ceftiofur or tulathromycin can be very effective.
	In-feed and water medication may be ineffective initially as the seriously affected pigs do not eat or drink
	Short acting drugs such as Ceftiofur or tulathromycin are very effective initially, followed up by long acting amoxycillin. Once the animals start to eat and drink other medication routes can be considered
Control	All-in/All-out programmes
	Purchase breeding stock from farms with good health programmes
	All purchased stock to go through isolation facilities
	Partial depopulation may be needed in herds with repeated acute or subacute problems.
	Vaccines may be useful either autogenous or commercial, but do require a farm specific programme to be worked out. Commercial vaccines can be very disappointing
	Elimination programmes may be possible via medication protocols using tulathromycin

Common differentials

	Swine Fevers, Erysipelas, Salmonellosis, Streptococcal septicaemia, Glassers disease and other abscess forming organisms in the chronic forms.

Zoonotic

	None

Actinobacillus pleuropneumoniae APP - check list

Farm: **Date:**

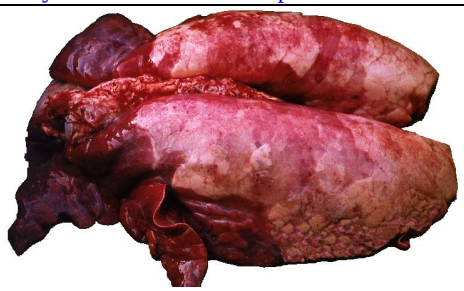
		Check
Undisturbed group of pigs	Examine and record lying pattern of undisturbed pigs ï picture/video	
	Behaviour of pigs around the water supply - picture/video	
	Behaviour of pigs around the feeders - picture/video	
Stock	Clinical pigs respond to tulathromycin (Draxxin), ceftiofur (Excenel), penicillin	
	Is Tilmicosin (Pulmotil) used in-feed or water medication.	
	Record post-weaning antibiotic use	
	Any APP vaccine used?	
Check for PRRSv	Blood results ï note 21 days antibody delay	
PMWS/PCVAD	Were pigs vaccinated at weaning ï PCV2	
	Check vaccine purchases correspond to weaning numbers	
	Check medicine storage ï 2-8°C	
Post-mortem	Ensure diagnosis correct - picture/video	
	APP actually being isolated ï which type is isolated	
	Slaughterhouse reports	
Sick pigs	Treatment of sick pigs	
	Movement of recovered pigs	
	Collect true mortality and morbidity figures	
Check weaning age and weight	Age at weaning	
	Weight at weaning	
Immunology	Feedback programme	
Pig flow	Collect true weaning/farrowing/breeding and gilt numbers by batch	
All-in/all-out	AIAO by pigs, water, feed, floor, air and medicine	
Medicines	Ensure needles and syringes not used between different batches	
Water	Flow ï 700 mls	
	Height suitable for the pigs	
	Number of drinkers	
	Cleaning programme between batches	
Floor	Stocking density	
	Cleaning programme between batches	
Air	Temperature variation	
	Relative humidity (50-75% RH)	
	High dust and endotoxin issues	
	Note level of slurry under slats ï air flow from underneath slats?	
	Draughts in the ñproposed sleeping areaò	
	Examine defecation pattern of pigs	
	Smoke buildings and record air movement patterns - picture/video	
	Cleaning programme between batches	
Feed	Feed space and feeder management	
	Problem coincides with a change in diet/feed type and feed size	
	Cleaning programme between batches	
Other problems	PRRSv, PCVAD/PMWS and Mange are classical examples of problems.	
	Eliminate any additional stressorsï weighing, tagging, bleeding	

ACTINOBACILLUS SUIS

Other names	This has historically been classed as secondary agent or even normal inhabitant of the mouth and throat, but as herd health has improved it is possible that a specific disease/characteristics may be associated with *Actinobacillus suis*
Causal agent	*Actinobacillus suis* a bacteria. Distinctive from *Actinobacillus pleuropneumoniae*
Age group	Sows with mastitis. Piglets with sudden death. Growing pigs with respiratory problems
Clinical signs	
Adult sows	*A. suis* has been associated with chronic mastitis and a few other abscessations
Nursery pigs	Septicaemia and sudden death
Recent problems	Pig found dead. High fever, lethargy and anorexia. Some skin discoloration.
Infectivity	
	Carried in the tonsils and nostrils of healthy pigs of any age
	It has also been found in the vagina
Stress factors	
	It is likely that all of the stress factors involved in the post weaning respiratory complex is also going to be involved
Post-mortem Lesions	
	Acute necrohaemorrhagic lesions scattered throughout the lung. There may be pleurisy, pericarditis, peritonitis and septicaemia. The lesions look very much like acute pleuropneumonia
Diagnosis	
	Culture of the organism
	Histopathology of the lesion
Treatment	
Individual	Because of the rapid onset of the disease in grow finish animals treatment can be difficult
	With sows with chronic mastitis cull from herd
Common differentials	
	Actinobacillus pleuropneumonia. Other causes of mastitis
Zoonotic	
	None

ENZOOTIC (MYCOPLASMA) PNEUMONIA

Also called	Virus pneumonia. Mycoplasma pneumonia. EP. PRDC ï Porcine Respiratory Disease Complex.
Causal agent	*Mycoplasma hyopneumoniae.* A mycoplasma does not have a cell wall
Occurrence	A disease commonly seen in growing and finishing pigs Note enzootic pneumonia may not require *Mycoplasma hyopneumoniae* ï it just describes the clinical condition
Complicating factors	
Bacteria	There are a number of bacteria and mycoplasma which can infect the lung, particularly after the effects of the mycoplasma on the mucocilary escalator. These include pasteurella, streptococci and *Actinobacillus pleuropneumoniae* (APP). *Haemophilus parasuis* (Glassers disease) also plays a contributing role in post-weaning respiratory disease.
Viruses	These include PRRSv, Swine Influenza, Circovirus II and Porcine Respiratory Coronavirus (PRCV). Aujeszkyôs Disease (Pseudorabies) may play a pivotal role.
Others	Parasites- Ascaris, lungworm
Environmental factors	There are many factors for which the stockperson is responsible. These include:
Air	Excessive 24 hours temperature variations. Draughts. High ammonia levels
Floor	Overstocking. Rough floors
Water	Poor water flow. Insufficient drinkers
Feed	Dusty feed. Poor feed availability
Clinical signs	Coughing, with or without fever (with fever 40.5 to 41C implies a complicated enzootic pneumonia), laboured breathing, variable growth rates, unthrifty appearance, reduced appetite and increased post-weaning mortality If naïve herd breaks, sows may abort and die.
Transmission	The disease can move via the air from infected farms to adjacent farms within 3 km. On infected farms, the disease is transferred from the sow/gilt to her offspring; sows may still have mycoplasma in her nose at parity 8. Infected pigs spread the disease by droplet spread from nose to nose contact and coughing pigs. One cough can spread the disease 4 metres, assuming the mycoplasma can survive the cough
Incubation	With high level of infection incubation takes 5 days. With a moderate level of infection incubation may take 4 to 6 weeks
Effects of enzootic pneumonia	Depending on the extent, Enzootic pneumonia can reduce daily liveweight gain by 17% and increase feed conversion by 14%. In other cases causes death. Enzootic pneumonia can also have a significant effect on PRRSv infections making them more serious to the weaner by encouraging macrophages into the lung.

Pathogenesis	*Mycoplasma hyopneumoniae* graze the cilia on the trachea and bronchi (the windpipe). The cilia are important as they help to protect the lung from particles (dust and disease). Once the disease enters the lung it causes areas to collapse and the pig progressively becomes short of air. The collapsed areas become infected with other diseases and the pig finally succumbs to the disease load. The mycoplasmas have an effect on macrophages and reduce their ability to kill and digest other pathogens. There is a significant effect of coinfection risk with PRRSv and Aujeszkyôs disease which will potentiate the clinical signs
	 The diseased collapsed areas are darker than the normal light parts of the lung
 The normal lung floats while the diseased (atelactic) lung sinks	
Diagnosis	Slaughterhouse examination
	Examination of the serum by ELISA and PCR. Immunohistochemistry of tissue sections
Treatment and control	Greater than 70% of normal health herds are infected by *Mycoplasma hyopneumoniae*. Because the disease is so widespread, control and treatment is complicated
EP - ve herds	Where herds are set up from EP-ve pigs, these herds have much less problems with respiratory disease. Maintenance of the EP status takes a lot of time and planning. Siting of such a pig farm is fraught with difficulty as the mycoplasma can spread 43km through the air
EP +ve herds	
Eradication	Difficult both practically and economically as herds can be re-infected quickly. May even be impossible on certain units.
	Programme utilizing tulathromycin can be designed
Antibiotics	Antibiotics limit the effects of the disease. However, subsequently to viruses becoming involved in pig respiratory disease, antibiotics are proving less effective
Herd management	Improvements in the environment of the pig greatly help to reduce the stress factors. In particular improvements in ventilation and a reduction in the stocking density should be attempted
Disease management	Partial depopulation, cleaning and repair of the growing/finishing phase has helped considerably. This may be combined with all-in/all-out, effective pig flow and 2 or 3 site production systems
Vaccination	Mycoplasma hyopneumoniae vaccines significantly help to reduce the effect of the disease. The vaccine is administered between 7 to 10 days of age and at weaning (21-28 days), but awareness need to be made regarding the maternal antibody levels provided from the sow Do not vaccinate the sow to raise maternal antibodies. Vaccinate gilts and boars as part of their introduction period.
Zoonotic	
	None

ENZOOTIC (MYCOPLASMA) PNEUMONIA

The approximate relationship between lung damage/scoring system at slaughter at 95 kg and daily liveweight gain and food conversion ratio

	Lung Lesion	DLW Reduction		FCR increase	
		%	gr./day	%	Value
	0/55 NEGATIVE	0	0	0	0
	2/55 MILD	-4	-25	0	0
	10/55 MILD	-7	-50	+5	0.15
	15/55 MODERATE	-11	-80	+8	0.25
	20/55 MODERATE	-15	-100	+11	0.35
	30/55 SEVERE	-20	-130	+14	0.40
	55/55 SEVERE	-22	-560	+17	0.50

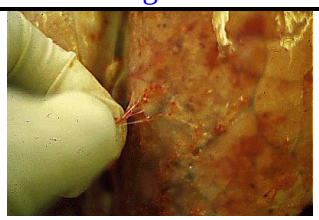
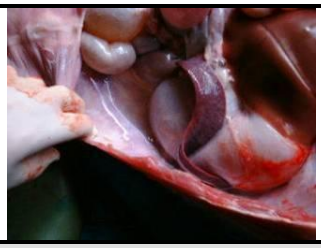
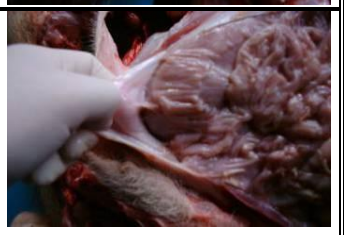
The estimates of reduction in DLW and FCR are based on Straw 1989 using pigs with 700g/day over the finishing period and a FCR of 3.
The lungs are shown from the front, with the intermediate lobe superimposed for completeness

GLASSER'S DISEASE

Causal agent	*Haemophilus parasuis* alone or a combination with various streptococci spp and *Mycoplasma hyorhinis*. Bacteria. There are at least 15 types of *H. parasuis*, many are none virulent and the types have little immunological similarities.
Age group	All ages are susceptible, however generally causes disease in weaned pigs
Clinical signs	
Naive herds Very rare	The disease creates a devastating acute meningitis
	This is normally seen when a naive adult is introduced to a normal herd.
	Within 48 hours the adult demonstrate severe pneumonia, depression, anorexia, high rectal temperature 42°C.
	Terminally the animal demonstrates incoordination, prostration, meningitis and dies. Death can occur very quickly after arrival
Normal Herds Acute	Usually sudden and often affects the better pig. The animal presents with depression, anorexia, the rectal temperature rises to 40.5C. Cyanosis may appear on the extremities. The animal may appear as if walking is painful. Terminally meningitis may be seen. Quite often the animal presents only as a sudden death
Chronic	Loss of part of the ear associated with failure of the circulation supply to the ears
	Wasting piglets who fade and die or grow very poorly
	Can be found as a diagnosis in late growers who die
  	
Weaners running off with Glasser's disease	
Infectivity	
	The organism lives in the nasal cavities of most normal piglets/weaners
Stress factors	
	Since PRRSv introduction, Glasser's disease has become more common/severe
	Vit E deficiency is often associated with the disease
	Environmental stress can play a role, characterised by draughts, chilling and a damp environment. In particular if the nursery is poorly set up. This places a great stress on the newly weaned piglet
	Variation in diurnal temperatures or poor adherence to cooling curves
Incubation period	
	Can be within 24 hours

Post-mortem Lesions

	The organism infects all the serosal membranes and produces a polyserositis. The clinical signs are dependent on which serosal membrane is affected
	The following organs are covered in a serosal membrane:
Heart	The disease causes pericarditis with both tags and fluid around the heart
Lungs	The disease creates extensive pleurisy
Intestines	The disease infects the abdominal cavity results in peritonitis
Joints	When the joints become infected synovitis and arthritis with swollen joints are seen
Brain	The meninges of the brain become infected resulting in a meningitis

At slaughter pleurisy tags can still be present			Chronic pericarditis
Acute peritonitis can appear as increased volume of peritoneal fluid			Chronic peritonitis with adhesions between intestinal loops and peritoneum

Diagnosis

	Clinical and postmortem signs
	Culture of the organism is difficult and requires special media. The presence of antibiotics in the pig makes isolation additionally difficult
	PRC is available, but does not differentiate pathogenic and not. Note almost all pigs are positive anyway

Treatment

	Antimicrobial agents, in particularly penicillin or amoxycillin based, initially via the water supply. However death can be very rapid before treatment can be initiated
	Removal of as many stress factors as possible
	Good gilt introduction routines to reduce PRRSv and Swine Influenza flair ups
	Vaccination is possible. Autogenous vaccines are often more effective owing to the large number of serotypes and little protection between the different types. Note a farm can be infected with multiple serotypes

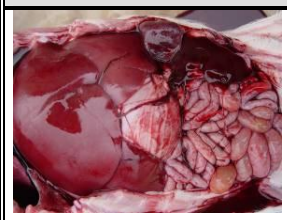
Common differentials

	Actinobacillus pleuropneumonia, Vit E deficiency

Zoonotic

	None

MULBERRY HEART DISEASE

Causal agent	Vitamin E/ Selenium deficiency
Age group	Post weaning, normally about 15-30 kg.
Clinical signs	
Acute	Sudden death. Generally the best pig(s) in the group.
	No other clinical signs.
Infectivity	
	Not infective to other pigs
Post-mortem Lesions	
	Large amounts of fluid around the heart and lung
	Haemorrhage and pale areas in heart muscle
	Fluid in the abdomen with pieces of fibrin
	Pale muscle areas in the leg and back
	The liver may be enlarged and mottled with areas of haemorrhage and possible rupture (as shown left)
Diagnosis	
	Post mortem findings
	Histological examination of the liver, heart or damaged muscle
	Serum samples may be difficult to interpretate
Treatment	
Affected group	Inject with 70 IU Vitamin E. May need selenium, note selenium can be very toxic
	While Vitamin E is a fat vitamin Water soluble preparations are available
	Increase Vitamin E in the feed to 150 g/tonne.
Control	Review vit E concentrations in the feed
	Check environment, remove stress factors
	Examine for Glasserôs disease
	Review feed storage, Vitamin E destroyed by high moisture and mycotoxins
	Review genetic and breeding stock
Common differentials	
	Glasserôs Disease, Actinobacillus pleuropneumonia, Oedema disease and Streptococcal septicaemias
Zoonotic implications	
	None

PNEUMONIC PASTEURELLOSIS AND STREPTOCOCCI

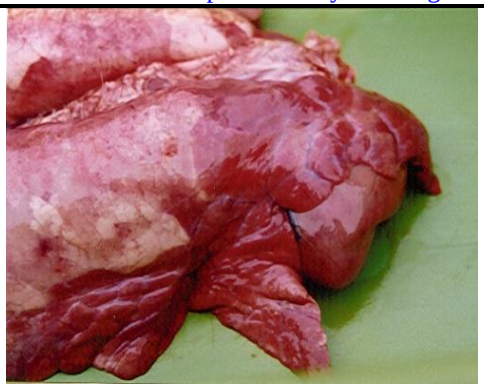
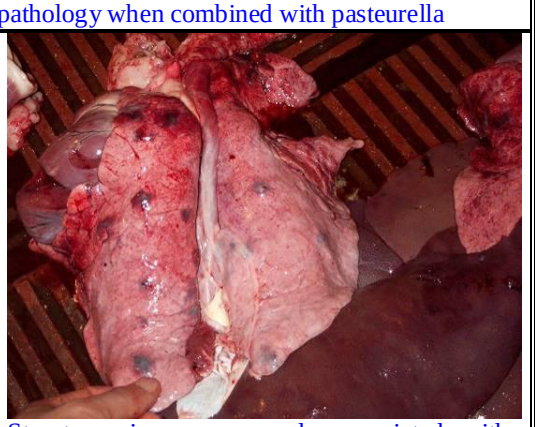
Causal agent	*Pasteurella multocida* and various species of streptococci, *typically Strep. suis*. Both are bacteria. Note toxigenic forms of *P. multocida* are associated with Infectious Progressive Atrophic Rhinitis.
Age group	Clinically affects the growing and finishing pig
Clinical signs	
	Generally represents the final stage of the post-weaning respiratory syndrome
Acute form	This is most commonly associated with *P. multocida* serotype B. The animal presents with dyspnoea, laboured breathing, thumping, high fever 42.2ÅC (108ÅF) prostration and finally death. Purple discoloration of the abdomen is not uncommon.
Subacute	Pleuritis, coughing, difficulty in breathing. In chronic cases the pig can lose a lot of weight. The pigs may have only poor or no growth with serious consequences in pig flow.
Chronic	Occasional cough, thumping and little or no fever. Generally affects pigs from 10-16 weeks of age (25-50 kg)

A pig with chronic pneumonia, where pasteurella and streptococci will be playing a role in killing the pig.

Nearly all of these pigs will be carrying pasteurella and streptococci in their noses.

Infectivity	
	Piglets are infected with streptococci from the sow within hours of birth, some may be infected intrauterine. Pasteurella may be acquired within 5 days of birth.
	The bacteria are very common and probably a normal inhabitant of the pig's nasal flora
	Nose to nose contact most common route of infection
	Aerosol infection is possible.
	Rodents may carry or transmit pasteurella
	The disease may be spread around the body via the blood stream after tail biting or feet damage.
Stress factors	
	All the normal stress factors involved in the post-weaning respiratory complex with draughts, chilling, damp environment, overstocking, mixed age groups and moving pigs are classic stressors.
Incubation period	
	The disease can be very quick as the organisms may be already established in the pig.

Post-mortem Lesions

	Generally part of the enzootic pneumonia complex with and superimposed on the lesions resulting in cranial consolidation. Severe cases may also present with pleurisy and abscessation. Note pasteurellosis in association with other viral agents in particular PRRS or Swine Influenza can produce lesions closely resembling *Mycoplasma hyopneumoniae* (Enzootic pneumonia) even on EP negative herds. Note water deprivation and high ammonia concentrations can produce very similar gross pathology when combined with pasteurella
Post-mortem findings	 Pasteurella and streptococci are commonly isolated from cases of enzootic pneumonia  Streptococci are commonly associated with pulmonary abscessation

Diagnosis

	Isolation of the organism. Note this can be complicated by the fact that both pasteurella and streptococci are very common in the respiratory tract. Also isolation can be complicated by concurrent antibiotic therapies. The isolation of pasteurella and streptococci does not mean they were the causes of the problem. However, they would have been significant in the animal's death.

Treatment

Individual	Antimicrobial agents, however, combinations are often required as the disease is rarely a primary agent.
	Vaccination against *Mycoplasma hyopneumoniae* has significantly reduced the effects of pasteurellosis.
Control	All-in/all-out
	Ventilation ï avoid draughts and high ammonia concentrations
	Avoidance of temperature fluctuations
	Stocking rate controls
	Reducing other causes of pneumonia and other respiratory conditions
	Minimal mixing and sorting
	Reducing building and pen size
	Enhance drinking water supplies
Vaccination	Generally disappointing

Common differentials

	Actinobacillus pleuropneumonia, *Salmonella choleraesuis*. Enzootic pneumonia. Most other respiratory diseases.

Zoonotic

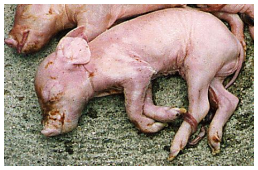
	None

PORCINE RESPIRATORY CORONAVIRUS

Causal agent	A coronavirus closely related but distinct from Transmissible Gastroenteritis Virus (TGEV) and Porcine Epidemic Diarrhoea (PED)
Clinical Signs	Clinical signs may go unnoticed or seen as a mild respiratory infection coupled with a fever and loss of appetite for a couple of days. There are no intestinal disorders associated with PRCV
Pathogenesis (course of the disease)	PRCV infects the respiratory tract. This is distinct from TGEV which infects the intestinal tract. Transmission is by air/contact/coughing not through faecal spread. Transmission can occur over many miles
Diagnosis	Unless specific methods are used can be difficult to distinguish from TGEV resulting in misdiagnosis. Specific monoclonal antibodies are used to distinguish between TGEV and PRCV
Control	There are no known control strategies to control the virus once on the farm. Eradication may be achieved though hysterectomy or segregated early weaning protocols. However, in countries with PRCV re-infection is highly likely
Importance	On its own PRCV causes few, if any clinical signs. However, it may play a role in the Post-weaning Porcine Respiratory Complex, especially when combined with other respiratory agents such as Swine Influenza (SIV) and Porcine Reproductive and Respiratory Syndrome (PRRSv)

PORCINE REPRODUCTIVE AND RESPIRATORY SYNDROME VIRUS

Other names	PRRS, Blue Ear Disease, Mystery Swine Disease, Swine Infertility and Respiratory Syndrome (SIRS), Porcine Endemic Abortion and Respiratory Syndrome (PEARS)
Causal agent	Virus. Porcine Reproductive and Respiratory Syndrome Virus ï RNA enveloped
Age group	Adult: Clinical signs generally reproductive, mild fever and anorexia
	Piglets through to finishing: clinical signs generally associated with secondary infections
Clinical signs	
Naive herds	Reproductive losses and a decreased farrowing rate
	Early farrowings, at 105 to 112 days
	Increase in stillborn, mummified and weak liveborn pigs
	Increased pre-weaning mortality often associated with increase in bacterial infections for example diarrhoea and greasy pig disease
	Increased numbers of unthrifty pigs post weaning
	Increased nursery mortality often associated with an increase in bacterial infections for example post-weaning diarrhoea and meningitis
On established herds	
Neonatal Pigs	Respiratory Distress Unthrifty and failure to thrive Increased secondary bacterial infections- scour and pneumonia
Growing pigs	Increased mortality Decreased appetite Fever Rough hair coat, unthrifty pigs
	Increased respiratory problems, pneumonia and atrophic rhinitis
	Increased secondary bacterial infections for example meningitis, Greasy pig disease
Adults	North America strains can cause major reproductive problems with massive abortions

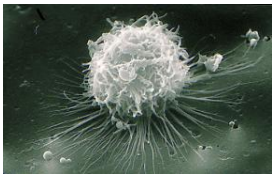
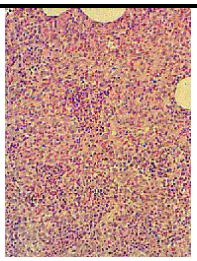
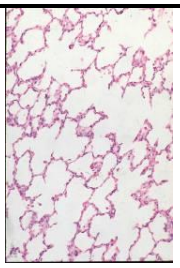


Stillborn and late mummified piglets

Sick pig with complicated PRRSv

Abortions

Infectivity	
	The virus particles have an envelope and rapidly becomes inactivated in the environment and in the presence of disinfectants
	Pig to pig contact the major means of spread, through infected faeces, urine and milk to piglets without colostral antibodies
	Transmission through needles and insects is possible, especially when blood transfer occurs
	Air transmission possible, but mainly when major outbreaks are present
	While virus particles are seen in boar semen for 90+ days and experimentally gilts can contract the disease through insemination, many thousands of inseminations from serologically positive boars to naive herds has not resulted in the spread of disease. Therefore the risk through AI is very small indeed
	When the disease first enters a country or new area, the level of disease locally can be very high and aerosol spread possible. Once the disease has stabilised in an area the risk of disease spread by semen or air is reduced.

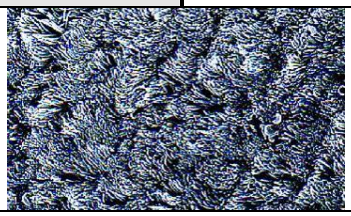
Post-mortem Lesions				
	There are very few visible post-mortem changes associated with PRRSv, majority of the signs relate to secondary infections. Histologically the major finding is an interstitial pneumonia and lack of air spaces. The disease selectively kills the lung macrophage, essential for the defence of the lung. The macrophages are killed or damaged for 26 days. After 7 weeks of age the alveolar macrophage becomes more resistant to PRRSv infection			
				
Post-mortem findings	Healthy plump macrophage	Flattened dying macrophage	Collapsed lung with interstitial pneumonia	Healthy lung ï lots of open alveoli
Diagnosis				
	PRRSv is suspected on the basis of the clinical signs			
	The presence of PRRSv on a unit is confirmed by the use of antibody tests. However, it can take 2-3 weeks for the antibody level to rise before the test becomes positive. Unfortunately the antibodies may also disappear 6 months after exposure.			
	Examination of the lung tissue by histology -immunohistochemistry can reveal the organism in the lung			
	PCR examination of tissues, in particular used for semen examination			
	Gene sequencing can be useful to monitor epidemiology of PRRSv between farms			
Treatment				
Infected Herds	There is no specific anti viral treatment for PRRSv infection			
	The treatment regimes aim to minimise the effect of secondary infections. Aim to keep the pigs warm and in the draught free environment and possibly increase feed density to compensate for the anorexia. Review the control measures for the secondary infections with the practice			
Control	SEW programmes can help to control the spread of the disease around the farm and minimise the effect of the disease on the farm's economy			
	All-in/ all-out and hygiene are essential precursors to controlling the disease			
	Current live vaccines result in excretion from vaccinated pigs and therefore cannot be used on PRRSv negative herds. The use of live vaccines in incoming breeding animals in PRRS +ve herds helps to maintain farm stability. The vaccinated stock must be kept separate from the farm until sheading has stopped			
	Home (Autogenous) vaccines from serum or tonsilar scrape therapy may be utilised to help gilt and boar introduction programmes. These should be restricted to the single farm			
	Gilts and boars must be stabilised before service. Discuss introduction programmes			
Vaccines	Dead vaccines generally confer no or little protection in naive animals, but it will reduce excretion of virus and assist reducing farm clinical signs in infected herds. Modified live vaccines ï these can be very variable in response depending on the modification carried out. Several MLV can cause severe clinical signs without field virus. In addition, there can be little protection provided for heterology virus strains. Allowing sufficient time between vaccination and field infection essential part of control. MLV general reduce excretion of virus particles.			
	Review fly and mosquito control programmes			
PRRS-ve herds	Before purchasing breeding or other incoming stock ensure you match serostatus. Unfortunately the testing procedures are not 100% accurate. Practice on-farm AI collection, do not rely on a commercial AI stud			
Common differentials				
	The clinical signs associated with Swine Influenza can mimic many of the signs of PRRSv			
Zoonotic				
	None			

SWINE INFLUENZA

Other names	Swine Flu, Flu, Influenza
Causal Agent 	Swine Influenza Virus. This belongs to the Influenza type A virus group. In the USA H_1N_1, H_3N_2 and reassortant H_1N_2 are problematic. The US has also developed a novel H_1N_1 with avian internal genes. Currently common varieties within the UK are H_1N_1, H_1N_1 (195852), H_3N_2 and H_1N_2. There are 16 H and 9 N types providing a lot of potential. There are at least 7 different pig adapted influenza viruses. The genome is divided into 8 segments.
Age group	All ages can be affected
Clinical signs	
 	Naive herds Explosive outbreaks with all or many animals becoming ill at the same time. Disease much more common in the Spring and Autumn Animals present with inactivity, depression, huddling/pilling. They are anorexic. The animals often are mouth breathing and breathing is laboured. When the animals are moved many cough, some uncontrollably (paroxysm coughing). They often have a nasal discharge and the eyes are puffy. Their rectal temperature increases to 40.5-41.5ÅC. As the disease progresses loss of weight may be seen. Mortality is generally low. The high rectal temperature in breeding stock can result in abortions, infertility (a boar can become sub-fertile for 6 weeks), production of small weak litters and increased stillbirths. Recovery generally starts 5 to 7 days after the first clinical signs
	Established Herds Annual re-infection appears, possibly from carrier pigs or the natural spread to younger naive pigs who present few signs in the summer months.
Infectivity	
	The disease is much more predominant in the spring and autumn months, however antibody investigations reveal little seasonal trend implying pigs get sick during the summer months without presenting with many signs
	Spread is mainly from pig to pig via the movement of animals, introduction of breeding stock
	Cross-Infectivity between human, pig, duck and turkey strains can occur
	Humans can transmit the disease to pigs and *vice versa*
	Virus can survive in the environment for a very short period of time.
Stress factors	
	Moving pigs
	Mixing pigs
	Poor isolation facilities
	Marked diurnal (day and night) temperature fluctuations
	Overstocking
Incubation period	
	1 to 3 days. Can be as short as 4 hours

Post-mortem Lesions

There may be few lesions seen in uncomplicated cases. There may be firm lobular lesions with interlobular oedema. Associated lymph nodes may be enlarged. The trachea can be filled with froth.
The Swine Influenza causes problems because it damages the lining of the trachea destroying the mucocillary escalator



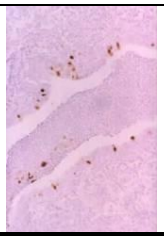
The normal appearance of the cilia

Effect of swine influenza removing vast areas of the cilia leaving the underlying cells open to infection and making it more difficult for the lungs to clear themselves of infection

Diagnosis

Clinical signs

Use of histology from fresh lungs and stained with antibody stains
For example, the picture shows the microscope view, the brown areas represent swine influenza



Paired blood samples checked for antibody concentrations (21 days apart), note maternal antibodies may persist for 2-4 months

Treatment

During outbreak	Cover all services with AI from a third party source
	No specific treatments available, all treatment regimes supportive
	Careful nursing in the farrowing house essential. Must ensure the farrowing house is draught free.
	All-in/All-out will limit the spread of the disease
	Provide fresh clean drinking water
Control	Avoid ducks and turkey contamination's/contact including staff
	Use disinfectants when cleaning buildings
	Vaccines
	Do not allow clinically affected people to work with pigs or in AI stations

Common differentials

Enzootic pneumonia. Other causes of reproductive problems. PRRSv

Zoonotic

Swine Influenza may rarely affect human beings. Note Humans can affect pigs ï the A(H1N1)2009 outbreak for example