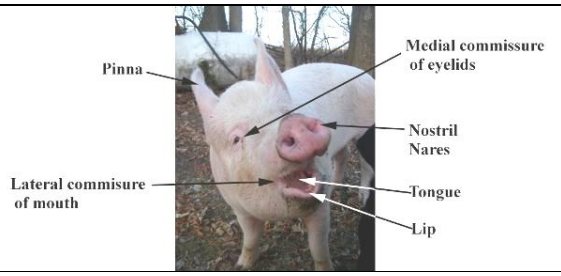
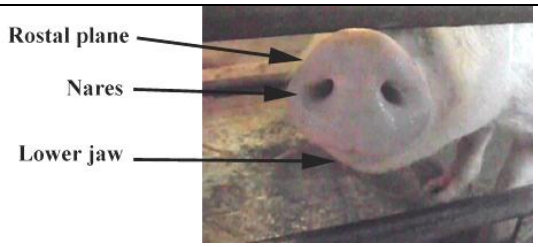
Disorders of the head

Disorders present in	Australia	Europe/Asia	North America
Clinical gross anatomy of the head			
Progressive atrophic rhinitis	Yes	Yes	Yes
Congenital tremor	Yes	Yes	Yes
Conjunctivitis	Yes	Yes	Yes
Meningitis	Yes	Yes	Yes
Post-weaning sneezing	Yes	Yes	Yes
Other conditions:			
Middle Ear	Yes	Yes	Yes
Aural Haematoma	Yes	Yes	Yes

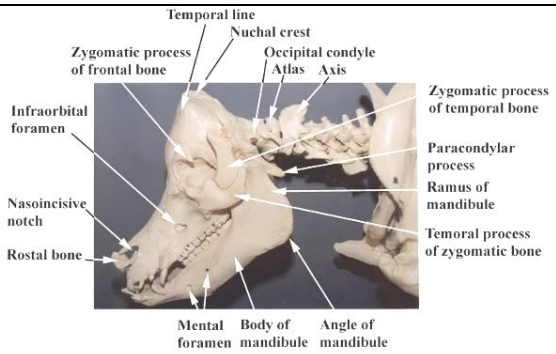
Clinical gross anatomy of the head



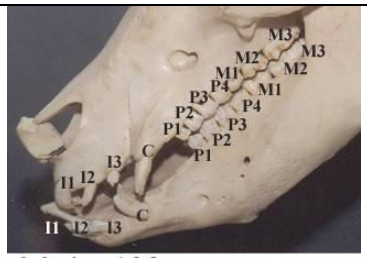
The head from the front



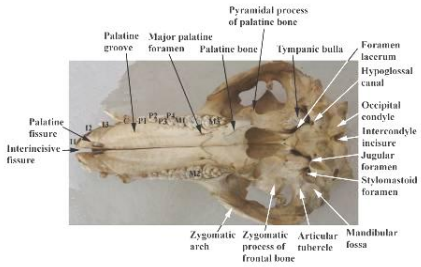
Detail of the nose



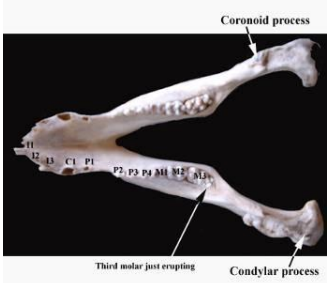
The skeletal head lateral



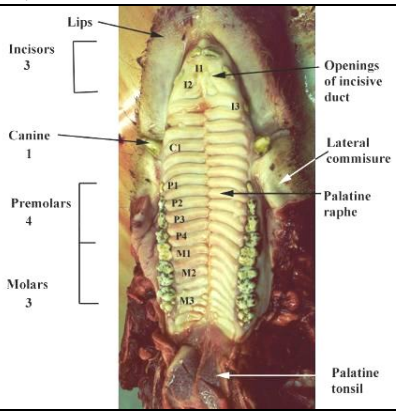
Detail of the teeth in the skull



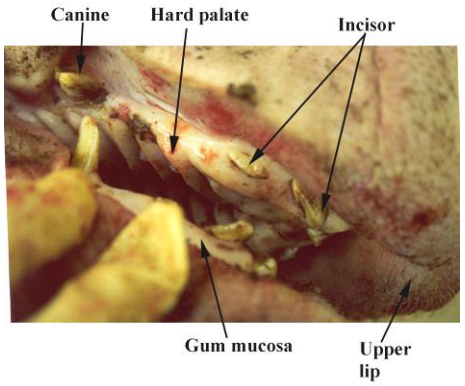
The skull, ventral view



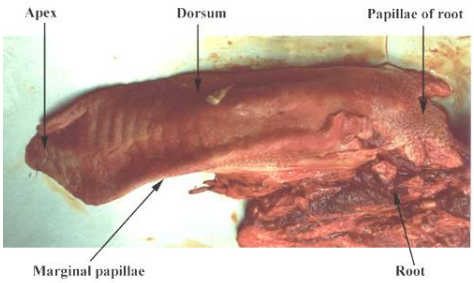
The lower jaw, dorsal view



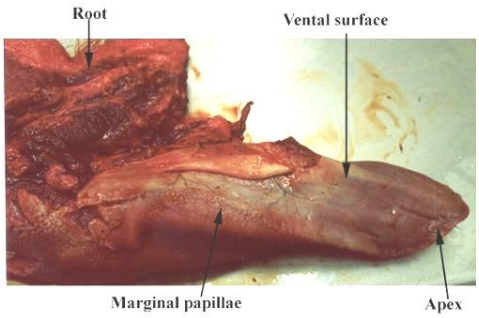
The hard and soft palate



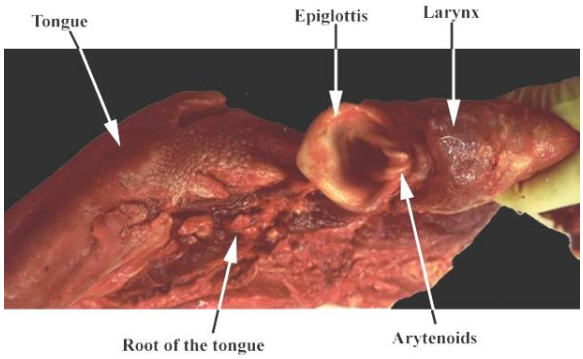
Detail of the teeth and gums



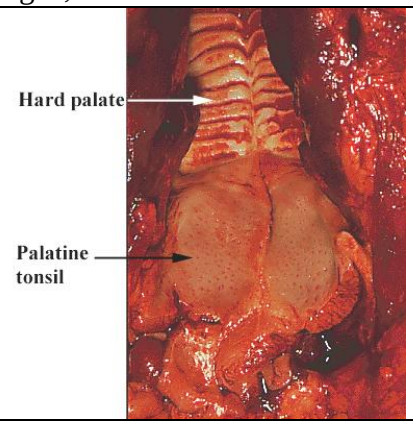
The tongue, dorsal view



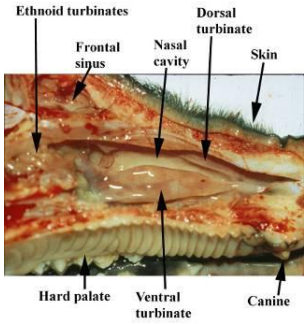
The tongue, ventral view



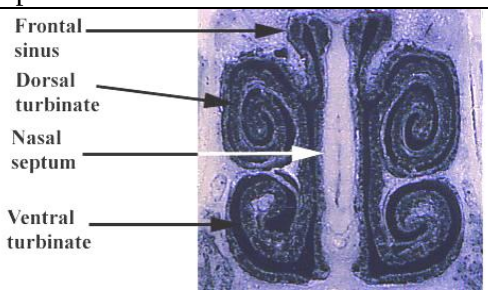
The tongue and larynx



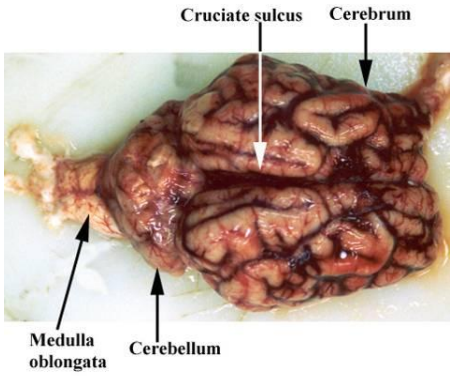
The palatine tonsils



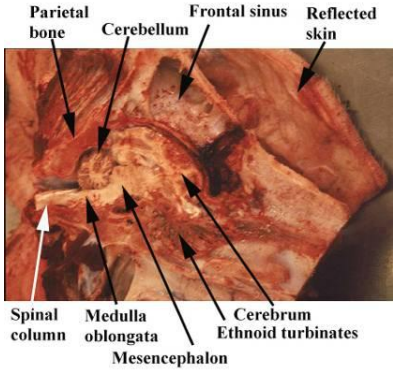
Longitudinal section of the nasal cavity



Cross section of the nasal cavity



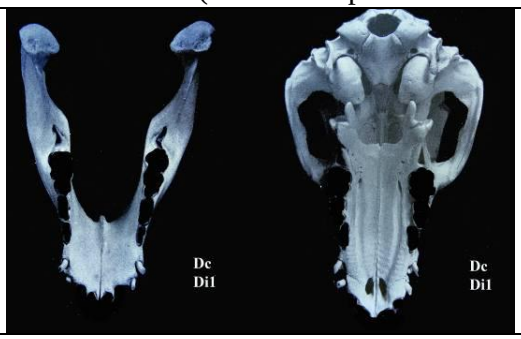
The brain, dorsal view



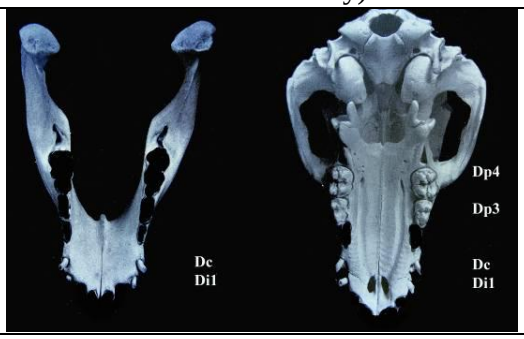
Longitudinal section of the cranial vault

Aging piglets, weaners and growers by their teeth

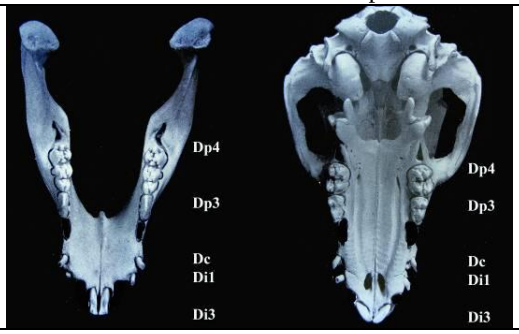
(the non erupted teeth have been blacked out to create clarity)



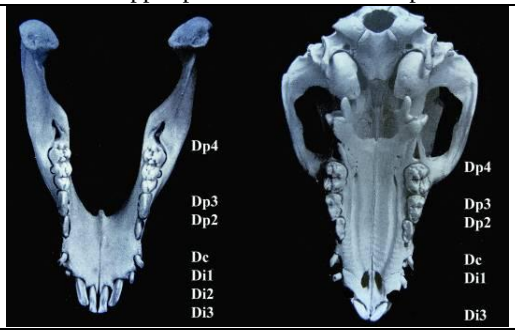
Birth ï the incisors 3 and canines are present



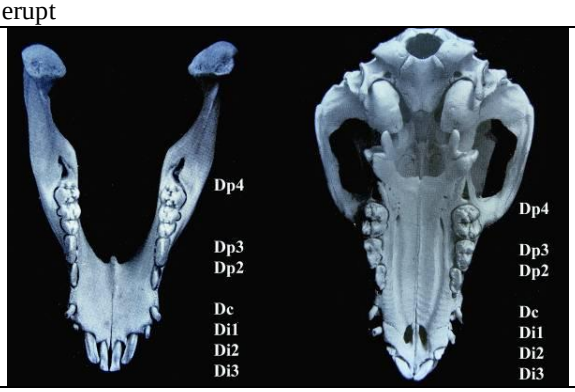
1 week ï the upper premolars 3 and 4 erupt



2 to 4 weeks: lower premolars 3 and 4 and incisors 1 erupt



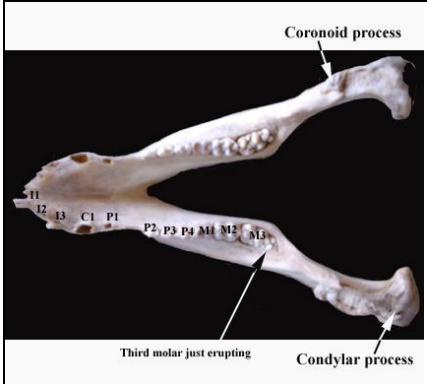
5 to 7 weeks: Premolars 2 and lower incisors 2 erupt

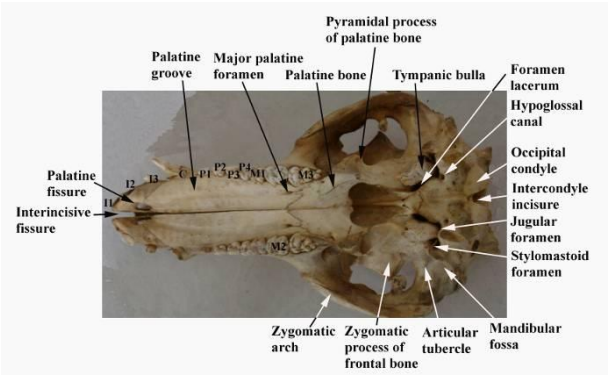


6 to 12 weeks upper incisors 2 erupt

Permanent teeth eruptions

4 to 6 months	M1
8 to 12 months	M2 and I3
9 to 10 months	C
12 months	I1
12 to 15 months	P1, P2, P3, P4
16 to 20 months	I2
18 to 20 months	M3





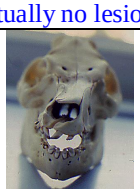
Adult lower and upper jaw with teeth labeled

INFECTIOUS PROGRESSIVE ATROPHIC RHINITIS

Other names	Atrophic Rhinitis AR PAR
Causal agent	Toxigenic forms of *Bordetella bronchiseptica* combined with toxigenic *Pasteurella multocida* mainly types D and A. Both of these agents are bacteria
Age group	The deviation may be seen in any pig older than 10 kg. However, clinical signs usually seen in pigs up to 5 months of age
Definition	Atrophic rhinitis covers any disorder that results in shrinkage of the nasal turbinates which may be accompanied by deviation of the face. Many factors may result in atrophic rhinitis: - dust, ammonia, disease agents and genetics. Infectious progressive atrophic rhinitis is the major category of Atrophic Rhinitis that requires to be controlled on pig units
Clinical signs	
	Note virtually all herds are infected with *Bordetella bronchiseptica* and therefore occasional moderate turbinate atrophy is normal
	Sneezing and snuffling in farrowing area and then throughout the growing phase
	Nose bleeding (epistasis) usually unilateral
	Snout deformation
	Growth retardation, which may be up to 7% loss of daily gain, but this is very variable
	Turbinate atrophy possibly with septum deviation
	Tear staining causing brown streaks on the face originating from the medial canthus of the eye. In modern breeds this is quite common without atrophic rhinitis
	Non immune piglets infected in the first week of life show most severe signs
	Pigs infected after 4 weeks show less severe lesions
	Pigs infected after 9 weeks of age show virtually no lesions

Twisted face and blood at nostrils

Twisted snouts in a group of pigs



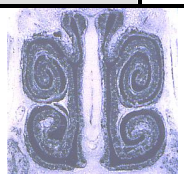
Skull of a pig with atrophic rhinitis

Acquired atrophic rhinitis

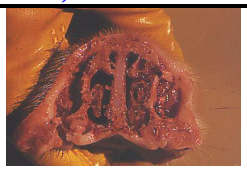
Infectivity	
	Pig to pig
	Droplet infection
	P. multocida can survive for 8 days in water, 6 days in liquid manure and 49 days in nasal washings
	Piglets are infected from their mother's mouth and possibly from the vagina during birth
	The disease may be transmitted by air between 200 and 1000 m
	Litters in the farrowing area are rapidly infected from a shedding mother and also at weaning
Stress factors	
	Re-occurrence, failure of vaccination or even first clinical signs on previously normal herds may occur when the unit is severely stressed by viruses such as PRRSv, PMWS or Swine Influenza

Post-mortem Lesions

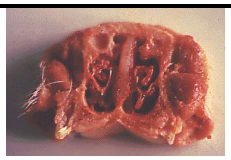
	Distortion of the face
	Atrophy of the nasal turbinates
	The atrophy can be scored particularly by sawing the nasal cavities at the level between the 1st and 2nd pre-molar teeth. Note a straight cut is required. This is indicated by the lateral canthus of the mouth (commissure)



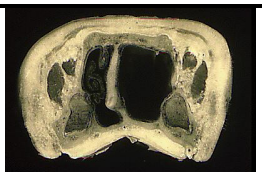
Normal turbinates



Atrophic score 3



Atrophic score 4



Score 5 + Deviation

Diagnosis

	All herds with clinical infectious progressive atrophic rhinitis as a problem are infected with Toxigenic *Pasteurella multocida*; however, not all herds with toxigenic *P. multocida* have clinically significant atrophic rhinitis. 20% of normal 'non clinical' herds may actually be infected
Slaughterhouse check	By sectioning the nose. However, the tests only look for 'atrophic rhinitis' not the infectious progressive atrophic rhinitis
Toxin check	Detection of the toxigenic *Pasteurella multocida* by examination of samples obtained by nasal and/or throat swabs ï test pigs at 4, 8 and 12 weeks of age.

Treatment

Affected individuals	Feed wet food
	Antibiotics will not reduce deviation but will help support the pigs if co-infected with other secondary pathogens
Control	
Acute outbreaks	Consult with your veterinarian for precise details, the following is a guide
	Vaccinate sows and gilts with a vaccine containing the toxin and *B. bronchiseptica* and *P. multocida*
	Administer in-feed antibiotics to sows for the last month of pregnancy
	Administer tetracycline LA or tulathromycin to piglets at 3 and 10 days of age
	Medicate weaner feed with tetracycline 800 g per tonne
	It is possible to vaccinate piglets within the first week of life
Essential other requirements	Improve general management, All-in/all-out programmes. Allow the sow herd age to rise Review breeding animal purchases. Reduce stocking rates. Reduce gas and dust concentrations. Move wet feeding finishing herd
Controlled	Vaccinate all sows and gilts as appropriate
Elimination	Depopulation and restock from a known free herd only possible option. Clean, disinfect and fumigate all buildings and leave for 8 weeks. Note eliminate mice and rats. The disease may rarely be transmissible over 1000 m
	It is possible to save genetic material by SEW programmes to set up a new unit followed by depopulation and restocking of old unit

Common differentials

	Sneezing in farrowing area may be due to a range of other organism in particular uncomplicated *B. bronchiseptica* and porcine Cytomegalovirus.
	Facial deformity can occur through other defects such a genetics, dust, ammonia and behaviour (playing with drinkers or bars for example)

Zoonotic Implications

	Note it is possible that humans may become infected with toxigenic *P. multocida* and this may result in a variety of upper respiratory diseases, tonsillitis and rhinitis

CONGENITAL TREMOR

Agent	A variety of events, diseases, toxins etc can cause a piglet to present with congenital tremor	
	1	The most common cause is an unidentified viral agent
	2	There are a number of genetic causes ï Landrace trembles, Saddleback tremors
	3	There are other known congenital infections for example Classical Swine Fever Virus which can result in the production of trembling piglets
This technical note will concentrate on the most common cause		
Clinical signs	The problem classically occurs in naïve gilt litters from newly introduced stock.	
New born piglets	Piglets are born trembling in all the muscles. They may be unable to walk and often are unable to suckle efficiently. Once attached to the teat by the stockman the piglet will suckle vigorously, but once let go by the stockman the piglet will often shake itself away from the teat. Excluding sporadic cases it is normal that there will be an increase in pre-weaning mortality and diseases, in particularly overlaids, increased lameness from trauma and outbreaks of scour. A trembling piglet which has problems suckling will clearly have problems ingesting sufficient colostrum. Mortality rates of 75 to 100% are common. Sometimes the trembling will be noticeably reduced when the piglet is asleep. In the piglets which survive, the clinical signs subside with age. However, the tremble is often noticeable if the animal is watched over time and then relaxes, then mild muscle trembles (fasciculation) will be evident over the ears and muscles on the back.	
Start up herds	The problem can be very severe and potentially disastrous with almost 100% of litters presenting with congenital tremor piglets	
Closed herds And new stock	In small closed herds it is possible for the resident animals to be or become naive. Under these circumstances the introduction of a new boar or other gilts will invoke the problem in piglets born to any pregnant sow. In these circumstances the farmôs original sows and gilts have problems.	
Sporadic cases	Congenital tremor is seen occasionally on most farms as an incidental occurrence and probably results from gilt or possibly sow which has by chance remained naive until she became pregnant.	
All other age groups	There are no clinical signs to born piglets, weaners, growers or adults who become infected for the first time. The disease only affects the foetus.	
Infectivity	The agent would appear to be very infective. However, on modern farms disease transmission can be slow and it is possible for the disease to die out on a farm. For this reason nucleus farms may produce naive gilts. All materials are infective ï faeces, placenta, nasal droplets, macerated piglets/foetuses and possibly semen	

Pathogenesis (how the disease occurs)	A naive gilt or sow becomes infected while pregnant. It would appear that getting infected at any time during gestation will result in the production of trembling piglets. Once the female is immune / positive the second and subsequent litters will not demonstrate clinical signs.	
Diagnosis	There is no test for the virus. Diagnosis is achieved by clinical signs alone.	
Postmortem examination	There are no gross postmortem findings. In cases of Swine Fever a reduction in the size of the cerebellum (part of the brain) may be noted (hypocerebellum). In the other causes there are no clear postmortem changes that can be identified.	
Treatment and Control		
Piglet with trembles	There is no specific treatment. The stockman can only provide help and assistance to the piglet with trembles. For example:	
	1	Helping them to obtain colostrum, even by stomach tubing
	2	Keeping the piglets warm
	3	If necessary euthanasing the piglets and using the gilt as a nurse sow
	4	Do not breed from the infected piglets as it is possible they will produce trembling piglets themselves.
Control		
1	Adequate introduction programmes to the newly arrived gilt. A minimum of 6 weeks between arrival and first service	
2	Ensure the gilts are adequately ómmunisedô to the farmôs diseases using faeces and if clinical problems are present placenta and macerated foetuses and dead piglets. It is essential to óinfectô all naive gilts before they become pregnant	
3	In startup units /repopulation ensure that there is no evidence of the previous livestock. In particular all faecal material must be removed. Do not use old needles/syringes or medicine bottles. Dispose of all old clothing	
4	On start-up units, obtain feed-back material from the AI source and feed to gilts prior to first mating ï for example dead semen	
5	Do not cull unnecessarily the gilt/sow which had the trembling piglets as they should not produce affected piglets again	
6	If there are any signs of Classical Swine Fever, immediately call your veterinarian	
7	If hereditary congenital tremor is believed to be the cause avoid mating the sow and boar or siblings in subsequent matings	
Zoonotic		
	None	

CONJUNCTIVITIS

Causal agent	Unknown. Probably a combination of *Bordetella bronchiseptica* and *Chlamydia psittici*.
Age group	Weaners and progresses into grow/finish and young adults
Clinical signs	
	Sneezing and runny eyes in the late farrowing house and nursery.
	Conjunctiva of both eyes become injected and inflamed
	Third eyelid prolapses. Once prolapsed condition seems to become static.
	Many only affect individuals, but on some farms all pigs in the pen are affected.
	The pigs otherwise do not seem affected by the condition

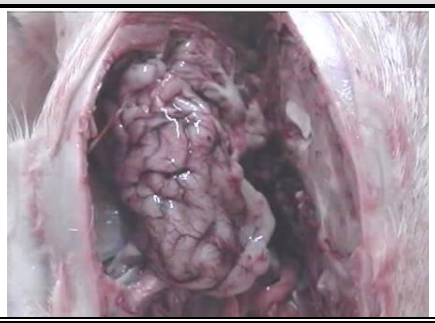
Growing pigs with conjunctivitis

Both eyes are equally affected

Note prolapse of the third eyelid

Infectivity	
	Unknown
Transmission	
	Probable nose to nose contact. Organisms isolated are very common on all farms with or without the problem
Post-mortem lesions	
	Severe conjunctivitis with prolapse of the third eyelid.
	Sclera and cornea unaffected
	Tear staining on face.
	Young pigs moderate to severe purulent rhinitis
Diagnosis	
	Clinical signs
	Chlamydia suspected as a secondary invader following swollen exposed conjunctiva
Treatment	
Individual/group	Review Progressive atrophic rhinitis vaccine programme.
	Test and eliminate toxigenic *Pasteurella multocida* as a problem
	Antimicrobials appear to have little effect. Overuse of antimicrobials may encourage Chlamydia as a problem
Control	Review management of the farm buildings
	Avoid chilling and draughts
	Reduce ammonia concentrations in the air
	Reduce dust levels in the grower and finishing house ï cover feeders, consider wet feeding
	Minimise the clinical effects of PRRSv
Common differentials	
	Progressive Atrophic Rhinitis. Swine Influenza
Zoonotic	
	None

MENINGITIS

Causal agent	The classical cause of meningitis in piglets, weaners and growers is *Streptococcus suis* II. However, several other bacteria can cause meningitis namely *Haemophilus parasuis* and septicaemic *Escherichia coli* or Bowel Oedema (F18)
Age group	Any. Meningitis classically occurs between 3-60 kg.
	Haem. parasuis in naive finishing and adult pigs can cause a devastating acute fatal meningitis
Clinical signs	
Acute	The pig becomes incoordinated often with uncontrolled eye movements
	Rectal temperature is increased to 40-41°C
	As the condition progresses the pig will fall over and thrash with all four legs on the floor.
	The pig may traumatise itself during these thrashing movements
	Death can occur quickly especially if the pig is stressed. In the farrowing house the condition can then be misdiagnosed as an overlaid pig
	If treatment is late a neurologically damaged pig may result
Normal pig	No clinical signs, the organism lives on the tonsils and upper respiratory tract of the normal healthy pig
 	Two growing pigs with meningitis
Infectivity	
	Most pigs are infected
Transmission	
	From the mother at birth by nose to nose contact
	Between pigs by nose to nose contact
Post-mortem Lesions	
	None particularly obvious. Meningeal congestion and tags are suspicious but most veterinarians examine so few brains it would be difficult for them to differentiate between the normal range of meningeal changes. The picture shows the brain in a sectioned head. Take a piece of brain and meninges for histology in all <30kg pigs 

Diagnosis	
	Culture of *Streptococcus suis* II from cerebrospinal fluid (CSF)
	Using an 18 gauge 1.5 inch needle. Place the pig in a dorsal position. Flex the head over the edge of the post-mortem table. Feel for the atlas occipital junction. Swab the skin with surgical spirit. Insert the needle and ówalkô the needle down the brain case off the occipital joint. Attach a sterile 2 ml syringe. 1 ml of clear CSF should be readily easy to extract
	The reason for using CSF is that *Strep. suis* is widespread on the skin and upper respiratory tract and post-mortem knives are easily contaminated leading to a misdiagnosis
	Histological examination of the meninges including a Gram stain
Treatment	
Individual	The pig is dying
	Treatment must be rapid and vigorous
	Ceftiofur 3mg/kg by intramuscular injection is the medicine of choice
	Treat with procaine penicillin every 12 hours until clinical signs subside
	A few pigs can develop a procaine allergy and go into anaphylactic shock
	If weaned isolate the pig. If not weaned place in creep area
	Keep in a darken room
	If fits are extreme give a sedative as necessary
	Provide water by mouth from a syringe is necessary. Note a pig drinks 1 litre per 10 kg per day so a few syringe fulls is not sufficient
Control	Most pigs carry *Strep. suis* on their tonsils and upper respiratory tracts. The causal factor that results in the clinical signs is that the pig is subjected to too much stress or other disease
	Draughts particularly in farrowing houses and nurseries
	Check colostrum management when meningitis occurs in piglets. Review feedback programme
	Wet and cold environments, common if weaners placed into cleaned houses. In outdoor situations use of damp mouldy straw often precipitates a meningitis ócutbreakô
	Excessive change in temperature in the pigs sleeping area
	Pigs being moved and mixed resulting in disturbed social hierarchy
	Gas levels being too high. Carbon monoxide poisoning from gas heaters
Common differentials	
	Haemophilus parasuis and septicaemic *E.coli.* Intoxication with brewer products. Water deprivation. Porcine Stress Syndrome. Trauma to the head. Aujeszkyôs Disease.
Zoonotic Implications	
	Strep. suis can rarely cause a fatal meningitis in man following infection into a cut from the pigôs skin typically following cutting a finger following injection of a pig. It is an occupational hazard

Streptococcus suis meningitis in weaners- check list

Farm: Date:

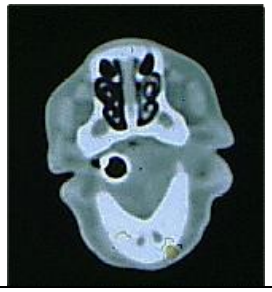
		Check
Undisturbed group of pigs	Examine and record lying pattern of undisturbed pigs ï picture/video	
	Behaviour of pigs around the water supply - picture/video	
	Behaviour of pigs around the feeders - picture/video	
Stock	Clinical pigs respond to ceftiofur or penicillin	
Check for PRRSv	Blood results ï note 21 days antibody delay	
PMWS/PCVAD	Ensure that the pigs were 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	
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 groups.	
Water	Flow ï 700 mls	
	Height ï 13-30cm	
	Number of drinkers	
	Cleaning programme between batches	
Floor	Nursery pigs need 0.3 m^2 per pig (to 30 kg)	
	Cleaning programme between batches	
Air	Temperature cooling curve 30°C entry	
	Cooling 1°C per week to 24°C	
	Relative humidity (50-75% RH)	
	Gas heating ï check colour of flame and CO concentration	
	Many farms with a meningitis problem staff have ñheadachesò	
	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 (50 mm per pig 30 kg) and feeder management	
	What is the Vit E concentration in the feed? Ideally 250mg/kg	
	Gruel feeding regime 3-4 days post-weaning	
	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 stressors at weaning ï weighing, tagging, bleeding	

POST-WEANING SNEEZING

Causal agent	Multiple bacteria and viruses. Includes, Bacteria- *Pasteurella multocida*, *Bordetella bronchiseptica*, *Haemophilus parasuis*, *Actinobacillus pleuropneumoniae*, *A. suis*, streptococci spp., pseudomonas spp., proteus and other environmentally originated bacteria. Various mycoplasmas. Chlamydia involved in the conjunctivitis. Viruses - Inclusion Body Rhinitis and PRRSv
Age group	Classically 10-30 days post-weaning
Clinical signs	
	The group of weaned pigs present with mild to severe sneezing. The symptoms will progressively reduce within 2-3 weeks.
	The sneezing may progress to middle ear disease.
	Conjunctivitis may be seen in several pigs. May be severe.
	  
Infectivity	
	Mixing of the ónormalô microflora of the nasopharynx between different litters of piglets.
Transmission	
	Nasal contact.
Post-mortem Lesions	
	A non-progressive rhinitis. Conjunctivitis. The nasal cavity may be filled with purulent material.
Diagnosis	
	Clinical examination. Weaners rarely die specifically associated with post-weaning sneezing.
Treatment and Control	
Treatment	Mixed infection. Specific treatment supportive and generally unrewarding. Often consider almost ónormalô on most farms.
Control	All-in/all-out
	Review management of the farm building. Avoid chilling and drafts.
	Review PRRSv stablisation programme
	Combine litters pre-weaning
	If sneezing also in farrowing house ï review atrophic rhinitis controls
Common differentials	
	Progressive Atrophic Rhinitis.

MIDDLE EAR DISEASE

This condition is characterized by the pig walking with a pronounced head tilt. Radiological examination reveals an abscess and occlusion of the middle ear on the side of the tilt. There is some evidence that *M. hyorhinis* may play a role in the pathogenesis. Culture at post-mortem tends to reveal a mixed bacterial population. The condition is more common in weaners who develop colds and sneezing 10-20 days post-weaning. Treatment of individuals may be unrewarding. Injections of ampicillin have been effective. Control is to avoid chilling and drafting pigs. However, the problem tends to be sporadic.



A CT scan on a middle ear case, demonstrating occlusion of the right middle ear ï left indicated by the black circular hole

In sows the condition may occur due to misplacement of drip cooling systems ï which drip into the sowôs ear.

AURAL HAEMATOMA

Characterized by a swelling of the external pinna (ear). The swelling is soft and fluctuated. The swelling is associated with blood leaking under the skin of the ear. The condition is associated with head shaking ï fighting and mange are important predisposing factors.

Treatment is varied depending on the farm

Do nothing ï the lesion with resolve with time, but the ears can be very heavy and they may become infected

Open the ear, but there is a risk of the pig bleeding to death if the internal bleeding has not stopped

Placing a band around the base of the ear and the ear will drop off.

This ear has healed after the resolution of the blood clot