DIAGNOSIS AND CLASSIFICATION OF PNEUMONIAS IN SWINE

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INTRODUCTION

Respiratory disease in housed farm animals is a problem of intensification and increasing herd size. The economic pressure to increase stocking rate within a giver air space will always mean that respiratory disease will be a significant cause of economic loss. It produces increased mortality, morbidity, culling rates, failure to grow, loss of breeding animals, expenditure on sanitation, drugs, treatments, hygiene and possibly restocking and leads to uneven production complicating all in/all out sysrems. There are also increased costs associared with slaughtering - delays on the line (for example, due to pleural stripping), loss of viscera and even carcases and also possible adverse effects on grading of carcases.

BACKGROUND FACTORS

Respiratory disease is the resukt of a complex interaction betwwen three major factors. Firstly the environment, secondly the host and its general and special defenc› mechanisms, and thirdly the micro-organisms, both saprophytic and pathogenic within and outside the host, and their complex relationship with the host and the environment. The important point is that respiratory disease is multifactorial (Done, 1982, 1988). The five most important factors are the presence of primary pathogens, seconday bacteria, management technique, environmental hazards and the age structure of the herd. Recently, the expansion of outdoor pig production in the UK has meat that the extensive system has reduced the severity of chronic bacterial pneumonias, but has probably not helped in the fight against recent epizootic disease since it is difficult to arrange simultaneous infection in large extensive herds, and therefore there is often the presence of both susceptible and resistant stock in the same unit with the possibility of recycling of waves of infection.

CLINICAL SIGNS

There are basically four clinical signs specifically related to the respiratory tract (see Table 1). Sneezing is associated with the upper respiratory tract (nares, nasal cavity), coughingwith the large airways such as larynx, trachea and bronchi (large airway disease) and dyspnoea with disease of smaller bronchi, bronchiles and alveili (Done et al, 1994). Performance can be adversely affected in a variety of ways - reduced feed efficiency, reduced daily gain, increased days to slaughter, and these are indicators of chronic disease such as enzootic pneumonia and seconday infections following primary viral disease (swine influenza and PRRS). The association of the major pathogens with the increasing age of the pig is shown in Table 2. It is also important to realise a variety of other signs in other systems can indicate severe disease that may affect the respiratory system. For example, the haemorrhages that may be seen throughout the respiratory that indicate the vasculitis caused by Hog Cholera or African Swine Fever viruses may be more easily visualised through the more avident skin lesions. Almost all the major systematic epizootics can be detected by attention to the signs in systems other than the respiratory system.

UPPER RESPIRATORY TRACT

The subject of this paper is the diagnosis and classification of pneumonias, but it is important to realise that there are two important factors that may influence the severity of disease in the lower respiratory tract. Firstly, environmental pollution is a significant hazard to the finishing pig. Levels of in excess of 20 ppm, dust levels in excess of 10mg/m3, bacterial counts in excess of 106-108 colony forming units per cubic metre have been described and high levels of endotoxin level (Backbo, 1990) pose a tremendous background environmental challenge for the pig lung.

Secondly, there is no doudt that conditions affecting the upper respiratory tract including congenital facial deformities such as brachygnathia, acquired abnormalities such as mandibular malalignmente and infectious disease such as paranasal abscesses or persistent conjunctivitis and porcine cytomegalic virus infections affect the ability of the upper respiratory tract to maintain a'clean environment' below the larynx. Obviously, this is worse in atrophic rhinitis, where the initial infection with B. bronchiseptica produces sufficient epithelial damage for subsequent colonisation by toxigenic strains of P. multocida. There deleterious effect are most severe in grande 3 and 4 atrophic rhinitis, where there are severely damaged conchal bones and in grade 5 atrophic rhinitis where they have been completely destroyed, and under these circumstances the lower tract has to assume some of the functions of the upper respiratory tract.

GROSS PATHOLOGY

Chest

Generally, the gross pathology of the thoracic respiratory system is seen as one of two major patterns, either consolidation or pleurisy. Consolidation simply means an accumulation of fluid and/or cells the lung and is seen macroscopically as a colour change from flesh to deep pink or plum. It typically affects the cranial lobes of the lung, usually in the sequence right cranial, left cranial, right middle, right intetmediate and left and right caudal lobes and is usually indicative of enzootic pneumonias (M. hyopneumoniae infection). Gross lesions of this type are usually indicative of chronic problems with widespread secondary

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bacterial invasion and are easy to diagnose only on a macroscopic picture.

It is also important to realise that the presence of lesions at slaughter does not indicate economic loss. All it indicares is there has been some loss of pulmonary lobule due to inflammatory reactions.

Heart Lesions

It is essential to examine the heart properly during gross examination of the chest, because manyof the problems of the lung have their origin in the circulation and particularly the heart.

Haemorrhagic lesions (petechiae) may indicate hog cholera, ASF, salmonellosis or erysipelas.
For this reason, it is essential to examine the heart for signs of pericarditis indicative of H. parasuis or S. suis infections, myositis indicative of encephalomyocarditis virus infection, or myodegeneration with haemorrhage indicative of Mulberry Heart Disease. Examination of the endocardium may show evidence of vegetative endocarditis associated with streptococci or erisypelas. A large amount of pericardial affusion may be associated with S. suis or H. parasuis infections or even fumonisin intoxication. It may or may not be accompanied by blood. Later in these two major infections of serosal surfaces (H. parasuis and S. suis there may be increased serofibrinous exudation and ultimately organissation of the exudate.

Lung Lesions - Viruses

An absence of macroscopic consolidation, or the presence of minimal consolidation associared with the presence of clinical disease suggests that tgere may be a viral infection (Done et al, 1991). In my experience in the UK, uncomplicated viral infections of the pig lung are often not accompanied by gross lesions and are only detectable on microscopic examination. There are two major infections of the lung in this group. The first of these, and of worldwide importance, is porcine reproductive and respiratory syndrome (PRRS) (Done et al 1988, 1992, 1993; Paton et al 1992). This generally does produce a slightly heavy, rubbery lung with no obvious lesions unless there is significant secondary infection. In there cases the bacterial pneumonias certainly mask any gross lesions of pneumonias associated with PRRS alone. The most commonly found lesions in PRRS affected herds are those of S.suis, H. parasuis and P. multocida.

Swine Influenza

In the UK we have been relatively free from swine influenza viruses. We received classical HlNl in 1985, probably from Denmark, and it went repidly through the UK pig population in 1985-1986 causing minimal clinical disease, gross pathology and microscopic pathology. It was closely followed by classical H3N2 strains and again this produced minimal changes and many pigs seroconverted without clinical signs as the disease passed through the national herd.

However, since 1992 we have experienced a much more virulent form of swine influenza (HlNl/195852) in which there has been substantial secondary infection often characterised by extensive consolidation, mucopurulent exudate, oedema and even haemorrhage. It is difficult in many instances to decide whether primary infection or the secondary infection has been responsible for the gross or microscopic lesions (Brown et al, 1993).

Porcine Respiratory Coronavirus (PRCV)

There are many strains of the virus. There is a considerable variation in pathogenicity from no disease to lethal pneumonia in SPF pigs (O'Toole et al, 1989, van Nieuwstadt and Pol, 1989). The virus does have the potential to predispose pigs to secondary invaders. In many instances there is an activated infection in the piglet as a result of swine influenza or PRRS infection.

Pseudorabies (PRV)

The clinical signs depend on the strain of PRV, the challenge dose and the age of the pigs infected. Often the signs are similar to flu, and some demonstrate CNS disorders. There is typically a high morbidity with low mortality. Pigs have fevers, are depressed and anorectic at the onset. Sneezing, coughing, 'thumping' and nasal discharge may be seen.

Porcine Paramyxovirus (PPMV)

A swine paramyxovirus has been associated with a moderate interstitinal pneumonia and a mild encephalitis (Janke et al 1992).

Proliferative Necrotising Pneumonia (PNP)

This condition has been described in Canada by Dea et al (1992) and Girard et al (1992) and is characterised by grey to plum colored lung with a rubbery feel and a generalised lymphadenopathy. Microscopic lesions consist of necrosis of terminal bronchioles, alveolar exuadation, Type II pneumonocyte proliferation, interstitial infiltration with mononuclear cells and proteinaceous material in alveolar lumens. We have not yet seen this disease in the UK, but I believe it has spread into the USA.

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Bacterial Pneumonias

In most instances bacterial pneumonias are a result of recrudescence of latent or carried infections, secondary to environmental or managemental malpractices or follow primary viral invasion and danage. Therefore a wide variety of bacterial agent may be isolated from these cases, and these are a reflection of the health status of the herd and the general environmental cleanliness.

However, there are instances where primary bacterial pneumonias may occur and these primary pathogens include M. hyopneumoniae, A. pleuropneumoniae, H. parasuis and B.bronchiseptica. Most of the other bacteria will not prodece pneumonia when inoculated intra-tracheally into susceptible pigs. Pneumonia can also reslt as a consequence of septicaemia or septic embolism from bacteria such as A. pyogenes, E. coli, A. suis or S. cholerae-suis.

Actinobacillus pleuropneumoniae

Many pigs carry theA. pleuropneumoniae orgamis in the nasal cavity, nasopharynx or tonsils, and outbreaks of disease may follow stress or adverse management. There are many strains belonging to 12 major serotypes. There is considerable variability between strains in virulence and pathogenicity with 1, 5, 9, 10 and 11 being the most virulent, but 3, 6 and 8 predominate in the UK.

All pigs may be affected and the clinical course can be peracute, acute, sub-acute or chronic. The clinical pictures vary from sudden death to lethargy, high temperature, cyanisis, dyspnoea and recumbency with high temperature.

In peracute and acute cases the gross lesions are virtually pathognomic with a necrotising pleuropneumonia thet is often haemorrhagic and which is usually accompanied by a severe pleurisy. the lung colour is very variable and the consistency of the lung is also very variable. The pleural fibrin often lies in sheets and may appear yellowish in colour. The interlobular septae are often distended by fibrin. The airways may be hyperaemic or blood stained and the tracheobronchial lymph nodes may also be enlarged, oedematous and sometimes haemorrahagic.

As the lesions become chronic so the degree of fibrosis increases and organisation takes place so pleural adhesions develop.

Mycoplasma hyopneumoniae

This agent only survives in swine and is the major cause of enzootic pneumonia (other contributors include H. parasuis, M. hyorhinis and M. hyosynoviae). It is rarely seen in pigs under 5-6 weeks, but we have seen thar in many cases when the pigs die of other causes, inspection of the lung reveals a wet exudative lung that is heavy and totally consolidated without producing any clinical signs.

The gross lesion seen in the abattoir is an exudative pneumonia - (consolidation) that is primarily seen in the cranial lobes of the lung. Many of the lobules may be plum-red or purplish in colour. Many of the airways are filled woth frothy mucus. In the later stages the lobules become grey in colour, as many more inflammatory cells infiltrate the lung. There is some attempt at the moment to use the term 'mycoplasmosis' to describe M. hyopeumoniae and 'enzootic pneumonia to describe M. hyopeumoniae and other secondary bacterial infections, and this may have something to recommend it.

Haemophilus parasuis

Grossly this agent produces a widespread pleurisy, pericarditis and peritonitis with arthritis and meningitis in pigs as a primary infection. It also contributes to the swine pneumonia complex as a secondary incader. It may be an opportunistic lung infection of low virulence that can cause suppurative bronchopneumonia secondaries to M. hyopneumoniae where the lesions are indistinguishable from enzootic pneumonia. In our opinion it is associated with stress whether it is movement, weaning, diet or environmental change.

B. bronchiseptica

This is not commonly found as a lung pathogen in the UK, but it may produce a chronic fibrinous pneumonia that organises with fibrosis of all tissues . It may be a secondary invader to enzootic pneumonia especially in herds with a severe rhinitis problem. It is probably a primary pathogen in young pigs, where it may cause a primary bronchitis in pigs under 4-6 weeks of age. Gross lesions are fairly characteristic with cranial and ventral lesions and in the acute cases these are red in colour, but in more chronic cases the lesions are grey as the fibrosis progresses.

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P. multocida

It is the most common invader of the pig lung. Widely recorded as a secondary infecrion, particularly following PRRS and swine infuenza, it may also accur as a primary diagnosis in highly stressed pigs as it is a normal inhabitant of the pig's respiratory tract that proliferates rapidly from the nasopharynx and tonsil.

a wide variety of gross lesions are seen, but they are generally similar to those on enzootic pneumonia. In some cases there may be much more haemorrhage and the lewsions may resemble APP, but in other cases where the serofibrinous exudation is greater, the lesions resemble more closely those of H, parasuis or S. suis polyserositis.

Streptococcus suis

This is a major organism isolated from the lung. Since the occurrence of PRRS it seems to be isolated even more frequently. It is not surprising that it is commonly isolated from the lung as it is normally carried in the tonsil, nasopharynx or nasal cavity.

The gross lesions are indistinguishable from those of enzootic pneumonia in most cases, but sometimes the lesions are similar to those of polyserositis caused by P. multacida or M. hyosynoviae. The serotype of the S.suis dose not appear to influence the lesions seen.

Mycoplasma hyorhinis

It is still not certain whether this is a major pathogen or not. It may contribute to enzootic pneumonia lesions and may also cause diffuse pleuritis. Gross lesions in experimental inocuations are similar to those of M. hyopneumoniae.

Septicaemias

Salmonellosis

Salmonella is a significant cause of pneumonia when there is often an early septicaemia following oral ingestion. The gross lesions are highly suggestive since there is skin discolouration with extensive cyanosis. There are lesions in other systems which help to suggest salmonellosis (gut, lymph nodes etc), but in many cases the gross lung lesions resemble an oedematous enzootic pneumonia.

Actinobacillus suis

This organism is associated with a widespread embolic septicaemia in all ages of pigs, but paricularly suckling and recently weaned pigs. It is often associated with repopuation of units where there appears to be poor immunity in recently introduced pigs.

Actinomyces pyogenes

Often in pigs a bacteraemia follows a localised infection and as a result there is an embolic pneumonia. It often co-exists with an osteomyelitis, valvular endocarditis or arthritis. The gross lesions are usually focal, multifocal, or confluent microabscesses. It is also isolated from lesions of enzootic pneumonia.

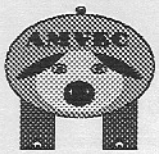
HISTOPATHOLOGY AND DIAGNOSIS

The respiratory tract is a branching system that becomes much smaller as division proceeds distally. There is a progressive loss of the supporting structures so that by the alveolar level only a squamous epithelium remains. There are two particularly weak points in the system. Firstly, the turbinates are especially prone to damage from inhaled debris and by damage from materials inhaled during rooting. Secondly, the bronchiolar region is protected by less efficient defences than are bronchi and alveoli. It is no coincidence that several of the major pig disorders (atrophic rhinitis affects turbinates; mycoplasma, swine influenza affects the terminal bronchioles) occur in these two sites.

It is also of considerable significance that there are two major group of pathogen. One that attaches to the surface epithelium, eg Bordetella bronchiseptica, P. multocida or Mycoplasma hyopneumoniae) and the other group that is a powerful producer of toxins, eg P. multocida or A. pleuropneumoniae. In addition, many other substances also exert a deleterious effect on defence mechanisms, and in particular noxious gases, particularly ammonia, may penetrate to peripheral parts of the lung and destroy cilia at all levels of the tract.

Virus Diseases - PRRS

The lesions caused by the UK strain of PRRS (Humberside agent) have been minimal and involve a loss of bronchiolar cilia and occasional epithelial cells with some blebbing of the surface epithelium of ciliated epithelial cells and a loss of luminal alveolar macrophages with an accumulation of interstitial mononuclear cells leading to the development of a mild to moderate



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interstitial pneumonia.

The absence of other lesions in the UK pigs affected by PRRS has been quite extraordinary. We have not seen lesions in foetal tissues, placentae or other systems (eg thymitis, splenitis or cardiovascular and CNS system lesions). There is a considerable in pathogenicity of the various strains. some cause indetectable disease, others moderate disease (3-5 days duration) and some cause severe persistent (7-21 days) pneumonia with thumping, cyanosis and mild CNS disease. Some isolates also cause rhinitis, encephalitis, myocarditis and nephritis. Some isolates also indice no detecable microscopic lesions.

Swine Influenza
In the UK, the classical HlNl and H3N2 strains have prodiced a typical interstitial pneumonia that is indistinguishable from that produced by PRRS infection. However, the 1992 variant (195852) produces an altogether different set of lesions characterised by a severe, extensive necrotising bronchiolitis and alveolitis, but in particilar a severe bronchitis. There were also pools of necrotic debris in the alveoli and a whole variety of bronchiolar abnormalities including sloughing, separation, dysplasia, hyperplasia and necrosis. Bronchiolitis obliterans is a particular feature in many bronchioles 2-4 days post infection.

Porcine Coronavirus
Often produces only a slight interstitial pneumonia with an occasional mild bronchiolitis. The most consistent feature is the presence of occasional syncytia in the alveoli and sometimes the bronchiles. They are not always present.

HISTOPATHOLOGY - BACTERIAL DISEASES

Actinobacillus pleuropneumoniae (APP)
The microscopic lesions are almost disgnostic of infection with this agent. The reaction is primarily fibrinous. Fibrin coats the pleurae and the alveoli are filled with fibrin. Fibrin also distends the alveoli. Thombi form in the lymphatics and small blood vessels. Many alveoli are necrotic and often alveolar destruction is so severe that micro-abscesses form. Neutrophils are largely absent or necrotic because they are destroyed by the toxins.

In chronic cases these esions are much more fibrotic and in many cases there is also considerable secondary infection.

Mycoplasma hyopneumoniae
Microscopic lesions in infections caused by this agent are basically bronchiolar associated lesions with characteristic bronchiolar lymphocytic cuffing. Cilia are often missing, and the epithelia are hyperplastic. Many neutrophils invade the alveolar lumina and the interstitial tissue.

Bordetella bronchiseptica
If one sees afibrosing suppurative alveolitis then it is likely that the cause will be B. bronchiseptica.

P. multocida
The microscopic lesions are of a severe suppurative alveolitis with abscessation and often extensive necrosis, sometimes with haemorrhage. Often there is a widespread pleurisy with extensive serofibrinous exudation, again often with haemorrhage.

S. suis
The microscopic lesions cannot be differentiated from those caused by other secondarybacterial lung pathogens, and in particular those caused by P. multicida.

A. suis
Microscopic lesions in this septicaemia include septic thrombo-emboli in vessels. There may also be extensive haemorrhage, necrosis and inflammation.

APPROACH TO DIAGNOSIS
Aliving animal sacrificed at the height of clinical disease will always provide a better diagnostic opportunity than a pig that has been dead for several hours. Blood for serology and blood culture for septicaemia, and possibly haematology, can be collected from this animal for a more complete diagnosis.

To investigate pneumonia cases effectively, and this is particularly in respect to post-weaning respiratory syndrome, you cer-

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tainly need a complete and detailed clinical history. You need if possible some live pigs, three acutely affected being the minimum, and carry out the postmortem examination yourself. If this is not possible, tissues both fixed and fresh from lung, turbinate, brain and heart will do, accompanied by fresh spleen and tonsil. Blood samples from twenty contemporary piglets taken at weaning and again three weeks later can be taken for serology and virus isolation if you suspect PRRSV.

It is essential on opening the thorax to take samples for subsequent microbiological examination without contamination. If the chest has been opened with the animal on its back, and the sternum and sternebrae removed by incision through the costochonbral junctions, then there is relatively easy access to the chest.

Fluid should be aspirated directly on opening (pericardial and pleural), samples for virological examination should be collected at the same time and then the individual tissues should be sterilised by flaming with a Bunsen burner before incision and sampling. Samples as far as possible should be taken from lesions to include the advancing edge of the lesion and thereby increase the chance of recovery of micro-organisms. Cotton swabs do not help in the isolation of bacteria, but the use of transport media certainly does increase the chances of recovery of organisms.

Nasal swabs, again from approximately 20 pigs, can be collected for virus isolation (particularly for PRCV and SIV infection).

A summary of the gross and microscopic pathology and diagnostic methods used in the study of pneumonia in pigs is shown in Table 3.

SUMMARY

The macroscopic anatomy and pathology of the chest of the pig will be described. Those diseases which produce generalised body lesions and significant thoracic pathology will be briefly mentioned (ASF, hog cholera,erysipelas, salmonellosis). Disorders affectig the heart which also cause lung abnormalities, principally cardiovascular changes, will also be briefly discussed (endocarditis, Mulberry heart disease, encephalomyocarditis, virus infections). The major part of the paper will describe changes in the lungs associated with viral diseases (PRRS and swine influenza) and bacterial diseases (Actinobacillus pleuropneumoniae, Pasteurella multocida, Haemophilus parasuis and mycoplasma hyopneumoniae infections The classification will be illuminated throughout.

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SIGNOS	ENFERMEDADES O CONDICINES PROBABLES
Signos sistemicos o generalizados	Epidemias importantes: Fiebre Porcina Clasica, * Peste Porcina Africana, * Enfermedad vesicular, Enfermedad de Aujeszky
Secreción nasal y esturnudo	- Contaminación - Altos niveles de amonia - Altos niveles de polvo Enfermedad de Aujeszky - Citomegalovirus porcino - Rinitis (Bordetella bronchiseptica) - Rinitis (Pasteurella multocida) - *A veces Virus hemoaglutinante de la encefalomielitis
Tos	- Neumonia - enfermedad de vias respiratorias con infecciones secundarias - Influenza Porcina - Bronquitis asociada con Bordetella - Neumonia Enzootica - Ascaris suum - Metastrongylus
Disnea	- Lechones - anemia, Enfermedad de Aujeszky - * Toxoplasmosis - *Sindrome respiratorio y reproductivo del cerdo - Influenza porcina pura - Coronavirus respirAtorio porcino
Aumento en dias al mercado	- Enfermedad cronicas - Rinitis atrofica - Neumonia enzootica - Infecciones bacterianas secundarias cronicas

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EDAD	ENFERMEDAD
0 - 7 días	- Enfermedad de Aijeszky - *Enfermedad del cerdo roncador - Rinitis por citomegalovirus porcino - Rinitis por B. bronchiseptica - Rinitis por P. multocida
7 días al destete	- Enfermedad de Aujeszky - Todas las anteriores - * Sindrome respiratorio y reproductivo del cerdo - Influenza porcina - Infeccion con Streptococcus - Infeccion con H. parasuis
Post - destete a engorda	- Todas las anteriores - M. hyopneumoniae - M. hyosynoviae - Salmonelosis
Engorda	- Todas las anteriores - Actinobacillus pleuroneumoniae - Actinobacillus suis
Adultos	- Pasterelosis - Anemia

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NEUMONIA	CAUSA	PATOLOGIA MACROSCOPICA	PATOLOGIA MICROSCOPICA	DIAGNOSTICO
A: HEMORRAGICAS	Fiebre Porcina Clasica	Hemorragias en todo el tracto respiratorio	Petequias, equimosis, edemas y vasculitis	Hist. clinica Flurescencia directa de bazo, Tonsila, riñón Aislamiento viral de Ileum
	Peste Porcina Africana	Mismas lesiones	Mismas lesiones	Inmunofluorescencia directa
B: VIRALES	Enfermedad de Aujeszky	Rinitis fibrinonecrotica, neumonia (tonsilitis, laringitis, traqueitis, ganglios edematosos y hmorragicos) (kluge et al, 1992)	Lesiones en sistema nervioso central (SNC), lesiones en pulmón, bronquiolitis necrozante + fibrina + corpusculos de inclusión intranucleares en cel. epit.	Signos clinicos: aborto, fiebre, lesiones en SNC. Aislamiento viral de tonsilas, pulmón y bulbo olfatorio. Serologia
	Citomegalovirus Porcino	Rinitis, conjuntivitis neumonia ocasional	Infiltración de mononucleares, corpusculos de inclusion	Signos clinicos, edad, histopato logia (Edington, 1992)
	Sindrome del Ojo azul (SOA)	Conjuntivitis mucosas congestionadas ojo azul. Lesiones no especificas	Neumonia Intersticial	Signos clinicos de SOA, serologia prueba de IHA, aislamiento viral en cel. de riñón
	Virus de la encefalomiocarditis	Cerdo cianotico. Fibrina en pleura. Pericarditis. Areas blanquesinas pequeñas en corazón.	Miocarditis con infiltración de selulas linfoides y plasmaticas	Records que muestran falla reproductiva y altas mortalidades. Ailamiento viral de corazón (Dea et al 1991).
	Coronavirus respiratorio porcino	No hay signos, con pulmon tipo goma.	Bronconeumonia intersticial con necrosis de vias aereas	Historia clinica serologia

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B: VIRALES (Cont.)	Paramixo virus porcino	Lesiones semejantes a neumonia enzootica. Enfermedad del SNC	Neumonía Intersticial	Aislamiento a partir de pulmón o cerebro. Inmunofluorescencia (Paul et al 1992 Janke et al 1992)
	Neumonía proliferativa y necrozante en Canada. Posible influenza porcina H1N1	Lesiones tipicas de Neumonía enzootica clasica	Proliferacion de células alveolares tipo 11. Necrosis de los espacios aereos	Serologia aislamiento viral (Dea et al 1992, Girard et al 1992)
	Sindrome reproductivo y respiratorio porcino	Puede haber aborto, mortina tos, muerte decoloración de la piel, pulmones cafe rojizo, a veces no hay lesiones	Neumonía Intersticial (lesiones vasculares, placenta, SNC bazo y timo)	Aislamiento del virus en macrofagos alveolares. Prueba de inmunoperoxidasa en células infectadas (Frey et al 1992)
	Influenza porcina H1N1 clasica H3N2	Consolidación, congestión, exudado mucopurulento. Pulmon con 50% de consolidacion. Puede haber pleuritis. Puede no haber falla reproductiva	Bronquiolitis necrosante, hemorragia, lesiones leves en Inglaterra Neumonía intersticial	Morbilidad alta, mortalidad baja. Aislamiento viral. Serologia prueba de inhibición de la hemoaglutinación
	Influenza porcina H1N1 (195852) Nueva variante	Severa neumonia catarral con bronquitis. Tonsilitis, conjuntivitis ganglios aumentados de tamaño	Bronquitis extensiva, bronquiolitis, alveolitis	Igual que la anterior pero la serologia debe hecerse usando el especifico.

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C; NEUMONIAS BACTERIANAS, INFECCIONES PRIMARIAS				
a) Necroticas hemorragica	Actinobacillus pleuroneumonia	Hiperagudo, sin lesiones, muerte subita. Diferentes grados de pleuritis y neumonia pilmones rojos hemorragicos, focas o multifocas más comune en lobulos diafragmaticos Edema luego pleuritis aguda o cronica. Neumonia serofibrinosa a veces artritis y meningitis	Necrosis hemorragica, pleuritis fibrinosa, hemorragia lobular. destrucción alveolar. Vasculitis con neutrofilos, despues infiltración de mononucleares.	Signos clinicos y aislamiento de la bacteria en tryptosa agar o en agar chocolate (Nicolet 1990 Bertram 1990)
	Salmonella la más comun cholerasuis	Lesiones de proceso sep ticemico. Pulmones edematosos, rojos y brillantes, a veces hemorragicos. La patologia es muy variable	Hemorragia, bronconeumonia necrozante, a veces solo neumonia intersticial	Signos clinicos, aislamiento del agente en MacComkey o tryptosa agar. Lesiones necroticas en higado. Ulceras en ileum (Schwartz 1991 Wilcock et al 1992)
	Antinobacillus suis (eguuli)	Usualmente seoticemia fulminante. Sindrome hemorragico difuso. Pleuroneumonia con adherencias entre lobulos. Puede haber infartos rojos en piel, aborto, artritis, meningitis y metritis	Lesiones troboembolicas en pulmon por cocobacillus. Puede ser hemorragicas.	Muerte subita, lesiones en piel, aislamiento de la bacteria en tryptosa agar o en MacConkey (Christenson 1986 Sanford etal 1990)
b) con pleuritis que varia, pudiendo ser hemorragica, necrotica o cronica.	Pasteurella multocida	Bronconeumonia purulenta, incremento de moco en traquea. Puede haber: septicemia, pleuritis, pericarditis meningitis o rinitis atrofica.	Puede haber abscesos, bronquiectasia, alveolitis necrosante, exudado serofibrinoso y pleuritis.	Aislamiento bacteriano en medios enrriquecidos (Hoie et el 1991, Falk et al, 1991)
	H. Parasuis	Lesiones difusas, artritis, meningitis, efusiones serosas, miositis..Lesiones de neumonia enzootica, pleuritis, poliserositis,poliartritis, neumonia fibrinosa.	Neumonia supurativa severa pleuritis supurativa severa.	Aislamiento de la bacteria esto puede ser complicado por lo delicado de la bacteria. Requiere de procesarse rapidamente se recomienda agar chocolate .
	Migración de ascarisPasteurella multocida	Hemorragias petequiales	Hemorragias, exudado eosinofilico	signos e historia clinica

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C: Bronconeumonias con abscesos	Infecciones secundarias a neumonía; pleuritis, septicemia, P. multocida, S suis, H. parasuis	Abscesos en pulmon. Focal multifocal o miliar. Micro o macro abscesos, otros tejidos con abcesos	Severa neumonia supurativa	Aislamiento bacteriano
	A. pygenes	Mismas lesiones más metritis, mastitis, endocarditis, neumonía, infecciones por heridas.	Superación, exudado necrotico. Microabscesos	Aislamiento bacteriano (Yager 1992)
	E. coli	Mismas lesiones	Mismas lesiones	Igual que la anterior
	S. aureus	Mismas lesiones	Mismas lesiones	Igual que la anterior
Streptococcus	Betahaemoliticum, zooepidemicus, suis	Abortos, abscesos, endocarditis, linfadenitis, mastitis, septicemia, neumonia.	Bronquilitis supurativa, alveolitis.	Aislamiento de la bacteria en agar chocolate o triptosa agar (galina et al 1992, Rivera - Reams 1992)