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Common respiratory diseases in commercial South African piggeries

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Respiratory diseases are of great economic importance in commercial piggeries. It has a negative impact on a piggery's efficiency in that it causes an increase in mortalities, an increase in medication usage, a decrease in growth and an increase in FCR (feed conversion ratio).

Respiratory diseases in pigs can be classified into two broad categories based on the disease's extent and duration. Category one would be those diseases that affect many animals and are usually very serious, but they tend to be of short duration. These diseases are usually very costly. Category two type diseases tend to be persistent problems with ongoing economic losses. Although mortality varies between the different diseases in the group, they all cause economic damage from an adverse and uneven effect on the growth rate, decreased feed efficiency, and additional costs of drugs.

Examples of diseases in the two categories would be:

Category 1	**Category 2**
Swine influenza	Mycoplasmal pneumonia
Classical swine fever	Pleuropneumonia
Pneumonic form of: pseudorabies virus, circovirus associated disease, PRRS	*Glaesserella (Haemophilus) parasuis*
	Pasteurella multocida

South African piggeries do not experience problems from diseases in category one, except for the pneumonic form of circovirus-associated disease. Diseases in the second category are, however, a common occurrence depending on the health status of the farm. We differentiate between high-health farms (specific pathogen-free -SPF farms) and normal-health farms. High-health farms are free of diseases of which pigs are the only carriers. Examples of respiratory pathogens are *Mycoplasma hyopneumoniae* and *Actinobaccilus pleuropneumoniae*.

Anatomy of the porcine lung

Pigs (like cattle) have well-lobated, well-lobulated lungs. Each lung is subdivided into various numbers of pulmonary lobes. In the past, these were defined by anatomic fissures. However, in current anatomy, lobes are defined by the ramification of the bronchial tree. The right lung consists of the upper, middle, caudal, and accessory lobes, whereas the left lung consists of the upper and caudal lobes. The left lung is ventilated through the left bronchus. The right main bronchus ventilates the middle, lower, and accessory lobe, whereas the tracheal bronchus, which arises from the right side of the trachea, ventilates the upper right lobe.

The degree of lobulation determines the degree of air movement between the lobules. Pig lungs have thick connective tissue separating individual lobules, making air movement between lobules absent. Pigs do, therefore, not have collateral ventilation.

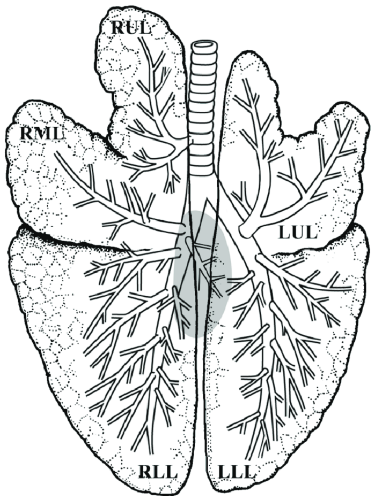


Figure 1: Schematic representation of porcine lung. LLL = left lower lobe; LUL = left upper lobe; RLL = right lower lobe; RML = right medium lobe; RUL = right upper lobe. (2)

Mycoplasma hyopneumoniae

Mycoplasmal pneumonia is a chronic, clinically mild, infectious pneumonia of pigs. It is also known as enzootic pneumonia, the most common cause of respiratory disease worldwide. It tends to become endemic in infected pig herds and negatively impacts growth and FCR.

M hyopneumoniae is a host-specific, pleomorphic agent that lacks a cell wall and is smaller than most bacteria. The organism grows extremely slowly, which makes it very difficult to isolate. The pathogen-host interactions are very complex, and the pathogenesis is not fully understood. The organism is mainly found on the trachea, bronchi and bronchioles' mucosal surface, which affects the mucosal clearance system by disrupting the cilia on the epithelial surface. It also modulates the immune system of the respiratory tract, making the animal more susceptible to other lung pathogens. Cell-mediated and likely also mucosal humoral responses are considered important for protection, but infected animals are not able to rapidly clear the pathogen from the respiratory tract. M hyopneumoniae is rapidly inactivated in the environment and by disinfectants, but it can survive longer in a colder environment. Pigs of all ages are susceptible to mycoplasmal pneumonia. Pigs are colonised within the first few weeks of life by their dam or other young pigs after mixing (at weaning). Sows of all parities can spread the disease to their offspring, but gilts litters are generally the most affected. It is usually introduced into a herd by an asymptomatic carrier pig. The incubation period can vary from 2 – 8 weeks before clinical signs are seen. The onset of the disease can occur throughout the grower phase and sometimes even during the late weaner phase.

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The incidence of lung lesions is highest in pigs 3–5 months old. Immunity develops slowly, followed by regression of the lung lesions. Older growing and mature pigs may recover from the disease completely. However, the persistence of the bacterium in the respiratory tract of infected animals has been confirmed for up to 7 months. Clinical signs are only seen in sows and piglets when the disease is introduced into a naïve herd.

When the disease first enters a herd, one will see the acute form of the disease. Typical clinical signs would be prolonged, non-productive coughing, respiratory distress, fever and high mortality rate across all ages. The chronic form of the disease is the more common form after the disease has been in the herd for a prolonged time. Often one will only hear a non-productive cough after the animals are roused. The morbidity is high, but the mortality is low, and other clinical signs can be minimal. Predisposing factors that cause flares of the disease include overcrowding, continuous flow production systems, concurrent diseases, < $3m^3$ air space per pig, poor air quality, temperature fluctuations, variable wind speeds and chilling, pig movement, stress and mixing.



Figure 2: Lungs with M hyopneumoniae lesions showing typical distribution of the lesions

The most common macroscopic lesion seen in mycoplasmal pneumonia is the consolidation of the cranioventral lobes of the lungs. The lobes are firm, red-purple in colour, and oedematous; the delimitation between healthy and affected tissue is clear. The alveoli can be swollen in complicated enzootic pneumonia cases, and mucus can block portions of the airways. Older lesions become clearly demarcated and eventually heal, leaving visible scars. The associated lymph nodes may be enlarged. Microscopically, cell congestion around the airways is often seen with enzootic pneumonia. These cuffs are typically made up of lymphocytes. The alveoli and airways are filled with fluids and macrophages.

Diagnosis is often based on clinical and histopathological findings and epidemiological indicators. In acute cases where pigs cough a lot, antemortem diagnosis can be made by taking oral-pharyngeal fluids and testing the samples via PCR.

Culture is seldom done because of the slow growth of the organism and the tendency for M hyopneumoniae to be overgrown by other mycoplasmas. In chronic cases, PCR testing can be done on oropharyngeal swabs. The lower down the sample is taken in the respiratory tract, the more sensitive the PCR becomes. Lung tissue samples (including airways) can be taken for histopathology. Commercially available ELISA tests are done on a herd basis, but results can be difficult to interpret because of the slow seroconversion of the disease. ELISA tests are more commonly used to confirm the negative status of SPF herds.

When a farm breaks with mycoplasma, the following classes of antibiotics are used to treat the pigs: macrolides, quinolones, tetracyclines and combination drugs. At the same time, the farm will start vaccinating the pigs. Once the disease is endemic in a unit, control is the main focus. It is done by vaccination. There are several vaccines on the market (single-shot and double-shot vaccines) and different vaccine protocols. A standard program would be to vaccinate piglets for the first time between 12 – 21 days and the second time 14 days later. Piglets are usually vaccinated at weaning when a single-shot vaccine is used. In some units, sows are vaccinated to prevent transmission to piglets. The whole sow herd can be vaccinated twice a year, or sows can be vaccinated before farrowing. Even if a farm is vaccinating, the disease sometimes flares. In these cases, antibiotics are used to bring it under control again. In some units, treating the pigs at strategic times is necessary to control mycoplasmal pneumonia. Buying gilts from a single source (preferably from an SPF unit) is also necessary for disease control. The only way to eliminate this disease is to depopulate and repopulate the unit.

Pleuropneumonia

Actinobacillus pleuropneumonia (APP) is the causative agent of porcine pleuropneumonia. It is a swine (host) specific respiratory pathogen that affects pigs of all ages, with many asymptomatic carriers. It is the cause of high economic losses in herds across the globe. It has a high morbidity and mortality rate, and losses can be significant. Losses result from increased mortality, reduced growth, veterinary costs and slaughter condemnations. APP is a gram-negative encapsulated coccobacillary rod. Depending on its nicotinamide adenine dinucleotide (NAD) requirements, it is classified into two biotypes, biotype one groups NAD-dependent strains and biotype two groups NAD-independent strains. Currently, 19 different serotypes are recognised. The biotypes and serotypes of *A. pleuropneumoniae* have genetic, structural, and virulence differences. Serotyping is mainly based on capsular antigens. The presence and prevalence of serotypes vary between countries. Although APP is a widely studied pathogen, its pathogenesis is not clear.

The virulence of the serotypes varies a lot, with some serotypes not virulent at all and other serotypes causing severe disease. App virulence is mainly determined by the production of pore-forming RTX toxins, namely ApxI, ApxII, and Apx III. These toxins have hemolytic and cytotoxic properties and are secreted by the different serotypes in various combinations. ApxI is expressed by serotypes 1, 5, 9, 10, 11, and 14. All serotypes produce ApxII except 10 and 14, and ApXIII is present in serotypes 2, 4, 6, 8, and 15. ApxIV is found in all serotypes and is specific to App; therefore highly used in diagnostics for species

identification. Many other virulence factors have also been described. APP survival in the environment is usually of short duration. However, it can survive for several days when protected by organic matter, such as faeces.

Transmission is primarily snout to snout and by air droplet transmission over short distances. In sudden outbreaks, sick animals will secrete large amounts of APP; therefore, APP has the potential to be transmitted several hundred meters by air. Other animal species have not been shown to carry or spread the bacteria to pigs. Recovered pigs may become chronic carriers and are a source of transmission within and between herds. Some exposed animals may become subclinical carriers. The spread between herds is related to the movement of animals as artificial insemination is an unlikely introduction source. Pleuropneumonia is more prevalent in units that get pigs from several sources regularly. Typically, in herds where APP is endemic, the piglets are infected in the farrowing pen, and a carrier sow is the source. All age groups are affected, and morbidity and mortality are linked to environmental quality, stress, and concurrent infection with other pathogens.

The clinical signs of APP can be categorised into peracute, acute, and chronic forms. The per-acute form is characterised by the rapid development of fever to 41.7°C, anorexia, and depression. There is an increased heart rate, and the skin becomes cyanotic, beginning at the extremities with terminal open-mouth breathing (thumping). Near death, there is foamy blood-tinged nasal/oral discharge. Animals may be found dead with no prior signs. In the acute form, pigs have fevers of 40.5–41.1 °C, depression, anorexia, reddening or congestion of the skin, severe dyspnea with a marked abdominal component, and sometimes death within 36 hours. The chronic form is characterised by variable cough, decreased rate of body-weight gain, and other complications (pleuritis, abortion, endocarditis, arthritis, abscesses).



Figure 3: Pig that died from APP. Note the severe fibrinous pleuritis and black areas of consolidation.

The gross findings in pigs with APP depend on the disease's time course. They include fibrinous pleuritis, pulmonary oedema, and the presence of bloody froth or clotted fibrin plugs in the trachea and bronchi. The lungs contain bilateral lesions that are dark red and firm, with a predominance of lesions in the dorsal aspects of the caudal lobes. There may also be a bloody nasal discharge. Lung lesions are well-defined abscesses and necrotic areas in the lung. Extra thoracic lesions are uncommon. Histopathologic lesions are necrotising, fibrinous, and hemorrhagic pneumonia that is predominantly lymphocytic and histiocytic, as well as vasculitis with thrombosis of vessels and lymphatics



Figure 4: Abscessation, usually caudo-dorsal

The golden standard for diagnosis is to culture the organism. Samples should include affected and healthy lung tissue, as the organism is best isolated from the edge of the lesion. Identification of the serotype is recommended when vaccination of the herd is considered. PCR is also an option to confirm recent infections. Chronic pleuropneumonia is more difficult to diagnose because it is difficult to culture the organism from chronic lesions. In these cases, the detection of antibodies is the best tool for confirming a suspicion of infection. Elisa test kits are available.

In very acute outbreaks, parenteral antimicrobials, including ceftiofur, penicillin, tetracyclines, and enrofloxacin, are necessary to reduce mortality Pigs are usually so ill that they do not eat or drink. A combination of parenteral and oral medication is generally the best method to get an acute outbreak under control.

In herds with APP, in-feed and in-water medication can be used strategically to prevent outbreaks. Vaccines are also available, but their ability to control the disease is variable. Neither antibiotics nor vaccines effectively eliminate APP; even pigs that survive the acute phase can reach subclinical infection and be persistent carriers, harbouring App in the tonsils and suffering from chronic lung lesions. The most satisfying control method is eliminating the disease from a herd (depop repop) and maintaining strict biosecurity protocols to prevent it from entering the herd again.

Glässer's disease

Glässers disease is an infection caused by *Glaesserella (Haemophilus) parasuis*. In 1910, Glässer observed a pig disease and associated it with a small, Gram-negative rod. In 1931 an organism, presumably the same, was isolated from pigs with influenza and named Haemophilus influenzae suis. By 1943 it was clear that the organism was a pathogen in its own right, not necessarily associated with swine influenza, and the name was shortened to *Haemophilus suis*. Then by 1976, definitive taxonomic studies resulted in the previous name, *Haemophilus parasuis*. The importance of Glasser's disease has increased with the expansion of the pig industry. In 2020, it had a name change (again) to *Glaesserella parasuis*,

G. parasuis is a small, pleomorphic, fastidious, gram-negative rod. It is confirmed as a member of the family *Pasteurellaceae*, but it is distinct from other genera in this family. It requires V factor (NAD) supplementation but not X factor (hemin) for growth. In the laboratory, *G parasuis* grows on enriched chocolate agar; it can also be cultured in blood agar with a staphylococcal nurse streak. It is not an easy organism to grow and often yields "no growth" on reports. At least 35+ serovars have been identified, with additional untypeable strains also common. Some are more commonly associated with disease than others, and groups of pigs often harbour several different serovars. The correlation between serovar and virulence is unclear, and strains belonging to the same serovar may vary in virulence. As the organism is delicate and does not survive drying well, indirect contact with clothing and pen furniture is less important. The infection (or a new strain of the organism) is usually introduced into a herd in carrier pigs. Infection may be present on a farm for some time without resulting in clinical disease.

Glässers disease has a worldwide occurrence, and the incidence of the disease seems to be increasing. It is a commensal organism of the upper respiratory tract and is transferred from sow to piglet before weaning. In most herds where the bacterium is endemic, sows produce a strong maternal immunity that normally persists in their offspring until 8 to 12 weeks of age. In such herds, pigs can be colonised by *Glaesserella parasuis* before weaning. Because of this strong maternal immunity, the effects of the infection in piglets and weaners are usually nil or very minimal. The pigs become sub-clinically infected when still protected, stimulating their immune response. If the maternal immunity wears off before they become infected, they may develop severe disease. It can also become a secondary organism with other major pathogens, such as enzootic pneumonia. Other than sow-to-piglet transmission, exposing pigs to other pigs or boots, clothing, or equipment that have been around other pigs greatly increases the risk for disease transmission. Pig-to-pig contact and aerosol are the primary modes of transmission.

Glaesserella parasuis has a short incubation period of around 12 hours. Within 36 hours, it progresses to a fibrinous polyserositis and polyarthritis, but septicemia with sudden death and bronchopneumonia also can occur. The disease has a sudden onset, short course, and high morbidity and mortality. Young animals (4–8 weeks old) are primarily affected. Animals of any age may be affected in non-immune herds. Coughing, bronchitis and meningitis may occur in gilts or older finishers (and sometimes even sows), and sudden death has also been recorded in this age group.

The onset of Glässers disease is usually sudden. The best pigs frequently are the first affected. They may be found dead or die after a disease course of fewer than two days. Typically, there will be red to purple discolouration of the ears, abdomen and occasionally legs. Where pigs are found alive, there will be a range of symptoms, depending on where the organism is localised. Severely affected pigs collapse and have laboured breathing associated with high temperatures (41°C or more), and there are often signs of the central nervous system disturbance. Signs may then include tremors, incoordination, posterior paresis or lateral recumbency. Arthritis often develops in several joints - affecting one or more legs with obvious joint swelling. It appears as if the pig is "walking on eggs". There is usually a lot of sneezing in the affected group of pigs, even if they are not disturbed. One can hear coughing. This is particularly evident in herds which are free of enzootic pneumonia.



Figure 5: Glässers disease

There is often a complete loss of appetite and severe listlessness. Survivors may develop chronic arthritis, pericarditis, heart failure, or intestinal obstruction once the fibrinous exudate becomes fibrous. Pigs with inguinal hernias are at particular risk of strangulation of the bowel. Chronically affected animals may have a reduced growth rate as a result of severe fibrosis in the thoracic and peritoneal cavities

Post-mortem findings usually present a serofibrinous or fibrino-purulent exudate on the mucosal surface, and characteristics of pleuritis, pericarditis, peritonitis, meningitis, and polyarthritis are observed. Microscopic lesions include inflammation of the serous membrane with neutrophils and macrophages. Sometimes, *G. parasuis* infection may result in acute septicemia, cyanosis, subcutaneous and pulmonary oedema, and death can occur without the typical serosal inflammation

A tentative diagnosis can often be made based on history, signs and typical lesions. The diagnosis should be confirmed by culture of *H. parasuis* from lesions affecting serous membranes, cerebrospinal fluid, joints, or other tissues, but this can be very frustrating. Brain, visceral pleura and other serosal exudates are preferred culture sites. PCR samples can also confirm the organism's presence, which, together with on-farm presentation, is often used to confirm suspicions.

The course of the disease is often short, and many sick animals die if untreated. Therefore, sick animals should be treated promptly and parenterally with an appropriate antimicrobial. *G. parasuis* is sensitive to many antibiotics and sulfonamides, but the sensitivity pattern of isolates from individual farms may vary over time, so periodic evaluation of antibiograms is warranted. If numerous pigs are affected, the severely sick pigs should be injected while the rest of the group receives in-feed or in-water medication.

Prevention of the disease involves management modifications, sound husbandry, and control of other respiratory pathogens, such as enzootic pneumonia. Glässers disease is very unpredictable. A standard prophylactic treatment plan is, therefore, difficult to prescribe. Recently vaccines have become available, but because there is not a lot of cross-protection between the different serovars, it is not guaranteed to work. Where vaccination is done, it is important to establish when the pigs become seropositive and adjust the timing of the first vaccination accordingly.

Pasteurella multocida

Pasteurella multocida is one of the leading causes of pneumonia in growing pigs. It is most commonly associated with enzootic pneumonia, playing an important role in the porcine respiratory disease complex, but it can also be the primary agent causing fibrinous pneumonia. Chronic thoracic lesions and polyarthritis tend to develop in both primary and secondary forms. It has a large economic impact as it results in decreased growth rate and feed efficiency and increases in premature death and condemnations at slaughter.

Pasteurella multocida is a facultatively anaerobic, fermentative Gram-negative coccobacillus found in the oropharynx of healthy animals, particularly cats, dogs, and pigs, as well as various wild animals. It is, therefore, present in almost every commercial herd, even if it is not causing disease. There are 4 Pasteurella species, but *P multocida* is the only one that routinely affects pigs. There are five serotypes, and type A is routinely isolated from affected lungs. It spreads via nose-to-nose contact and vertical transmission from sow to piglet.

In acute cases (generally when it acts as the primary agent), the pigs will present with severe, sudden pneumonia. Any age group can be affected. They will have high temperatures with discharges from the nose and breathe rapidly with open mouths (pumping). The skin on the extremities, especially the ears, will discolour blue. Mortalities can be very high. In sub-acute cases, the pneumonia is less severe but often complicated by heart sac inflammation and pleurisy. Pigs are usually between 10 and 18 weeks old. The pigs cough, and they have discharges from the nose. Pigs lose condition, and there is an increase in mortality.

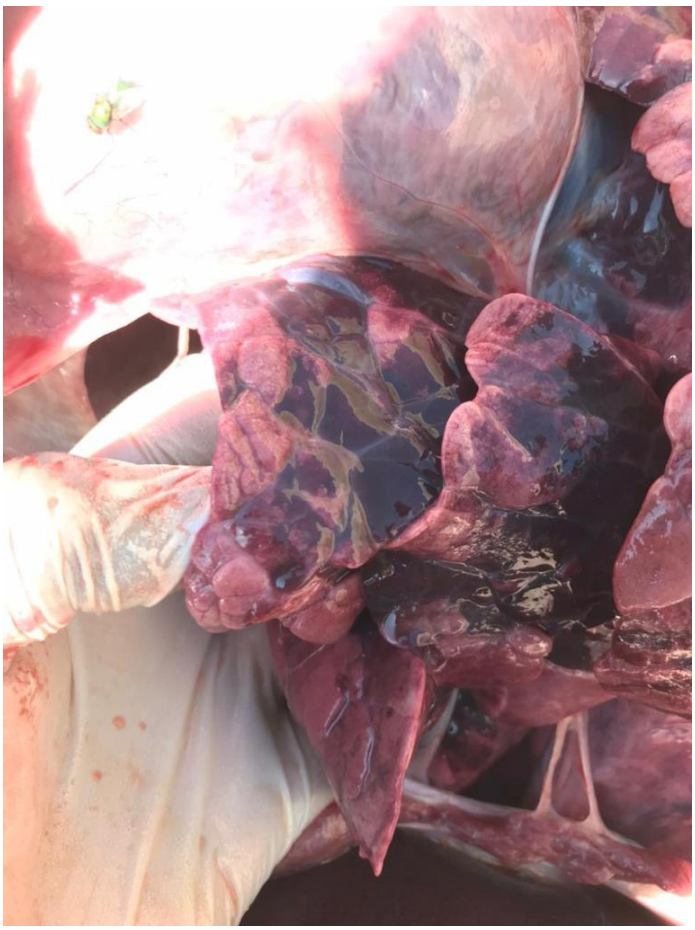


Figure 6: Primary pasteurellosis

Macroscopic lesions vary depending on the nature of other infectious agents. Consolidation of the cranio-ventral lobes is consistently seen and more severe than in the case of *M.hyopneumoniae* infections. Fibrin and adhesions between the lung and the pleura are common. Since *P.multocida* becomes systemic, pericarditis can also be seen. Lymph nodes near the affected areas are enlarged. Microscopically the alveoli present a large number of neutrophils, fibrin, and bacteria typical of a suppurative bronchopneumonia.

Because of the many other pathogens causing pneumonia, clinical signs and lesions can not be used for diagnosis.



Figure 7: Pasteurella with adhesions

The presence of other infectious agents can also make diagnosis more difficult. Swabbing the trachea and collecting lung tissue for culture and histopathology is the standard diagnostic method. Culture and isolation of *P.multocida* are relatively easy to do.

Because P multocida is a normal commensal of the upper respiratory tract, a positive sample from the upper respiratory tract (nasal swabs or oral fluids) is inconclusive. In acute outbreaks, parenteral antibiotics are better to use. Tetracyclines, cephalosporins and macrolides tend to be effective. In sub-acute cases, oral antibiotics can be used. Because this organism tends to develop resistance, it is best to do regular antibiograms. The disease is controlled by good husbandry practices and by controlling other respiratory pathogens.

Porcine respiratory disease complex

Porcine Respiratory Disease Complex (PRDC) is a multifactorial syndrome that causes health problems in growing pigs and economic losses to farmers. This complex syndrome is influenced by the presence of several types of pathogens (porcine reproductive and respiratory syndrome virus (PRRSV), porcine circovirus (PCV)-type 2, *Mycoplasma hyopneumoniae, Mycoplasma hyorhinis, Pasteurella multocida, Haemophilus parasuis*, etc.), along with environmental conditions, as temperature, dust, ammonia, carbon dioxide and airborne bacteria and farm management. The role and impact of different microorganisms in the development of the disease can be complex, and these are not fully understood. The severity of lesions is a consequence of synergism and a combination of different factors. On most farms, it is more than likely that several, and not only one, organisms are causing the problems. When deciding the cause of the problems, a systematic approach should be followed, looking at both organisms and the environment.

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MULTIPLE-CHOICE QUESTIONS

QUESTION 1

Which disease is not an example of persistent disease with ongoing economic losses?
a) Enzootic pneumonia
b) Pasteurellosis
c) Classical swine fever
d) Glässers disease
e) Porcine pleuropneumonia

QUESTION 2

Porcine lungs are:
a) Well-lobated, well-lobulated
b) Well-lobated, poorly lobulated
c) Poorly lobated, poorly lobulated
d) Poorly lobated, well lobulated
e) None of the above

QUESTION 3

The following class of antibiotics will be unsuccessful in the treatment of enzootic pneumoniae:
a) Macrolides
b) Quinolone
c) Tetracyclines
d) Penicillin
e) Combination drugs

QUESTION 4

Which statement is true regarding enzootic pneumonia:
a) The causative agent for enzootic pneumonia is a virus
b) The most common macroscopic lesions seen are the consolidation of the cranioventral lobes of the lungs
c) The organism survives for extended periods in the environment
d) The disease is usually confirmed by culturing the organism
e) It is not possible to eliminate the disease from a farm

QUESTION 5

How many serotypes of APP are currently recognised?
a) 19
b) 3
c) 18
d) 7
e) 35

QUESTION 6

The gross findings in pigs with APP depend on the disease's time course. Which of the following is not true?
a) Fibrinous pleuritis, pulmonary oedema, and the presence of bloody froth or clotted fibrin plugs in the trachea and bronchi.
b) Bilateral lesions on the lungs
c) Lungs appear dark red and firm, with a predominance of lesions in the dorsal aspects of the caudal lobes.
d) Lung lesions are well-defined abscesses and necrotic areas in the lung.
e) Extra thoracic lesions are a common finding

QUESTION 7

***Glaesserella parasuis* is part of which bacterial family?**
a) Corynebacteriaceae
b) Pasteurellaceae
c) Enterobacteriaceae
d) Staphylococcaceae
e) Pseudomonadaceae

QUESTION 8

Which of the following symptoms is not typical for Glässers disease?
a) Sneezing
b) Arthritis
c) Vomiting
d) Lateral recumbency
e) High fever

QUESTION 9

Which serotype of Pasteurella multocida is routinely isolated from pig lungs?
a) D
b) E
c) C
d) A
e) B

QUESTION 10

Porcine Respiratory Disease Complex is influenced by the presence of several types of pathogens. Which one of the pathogens below does not influence it?
a) Porcine circovirus (PCV)-type 2,
b) *Mycoplasma hyopneumoniae*
c) *Pasteurella multocida*
d) *Haemophilus parasuis*
e) *Brachyspira hyodysenteriae*

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