Tegumentary

Eyelid
Edema
Edema disease

Skin
Cyanosis (Congestion and vasculitis)
Porcine Reproductive and Respiratory Syndrome
Porcine arterivirus

Skin, planum nasale (snout)
Melanosis
(Not to be confused with cyanosis)

Skin, pinna
Otohematoma
Trauma
(Unilateral, bilateral and scar)

Skin
Wounds

Skin
Periarteritis nodosa
Also kidneys, heart, gastrointestinal tract, meninges, lungs and liver. Mainly in sows. Etiology unknown.

Skin, scrotum
Angiomatosis

Skin, pinnae
Gangrene, bilateral, apical, locally extensive
Sepsis. Various bacteria including Salmonella.

Piglet, Skin and limb extremities
Amputation
Cause unknown in this case.

Skin
Petechiae, diffuse
Bacteria (e.g. *Salmonella* spp. *Erysipelothrrix rhusiopathiae*); PCV2.

Skin
Infarcts, multifocal (with vasculitis)
Previously considered pathognomonic for *Erysipelothrrix rhusiopathiae*. Sepsis due to several bacterial infections including *Salmonella* and *Actinobacillus suis*.

Erysipelas still occurs in swine raised entirely in environmentally regulated buildings. *E. rhusiopathiae* causes disease in all ages. Mortality is highest and lesions are most extensive and severe in suckling and recently weaned pigs. In growing and finishing pigs, pigs may be found dead with few gross lesions - typically sparse renal cortical petechiae and a slightly enlarged spleen that is firm and red or lameness may predominate with proliferative synovitis and fibrous periartthritis. Outbreaks in sows are typically associated with pyrexia, anorexia, few cutaneous infarcts and occasional abortions. *Eryseipelothrix* has zoonotic potential and may cause endocarditis in humans. Erysipelas may have a potential involvement in urogenital disease of the sow.

Skin
Epidermitis, exudative
Greasy pig disease (syndrome)
*Staphylococcus hyicus*
Epidemics predisposed by PRRSV immunosuppression.

Skin, snout dorsum
Dermatitis (exfoliative)
Lincomycin

Skin, dorsum
Dermatitis, necrotizing
Sunburn

Skin, snout
Vesicular dermatitis
*Foot and mouth disease picorna-aphtovirus; vesicular stomatitis rhabdovirus; swine vesicular disease picorna-enterovirus; vesicular exanthema vesivirus-calicivius.*

Skin
Dermatitis, papular, pustular, ulcerative, necrotizing
Swinepox
Poxvirus
Due to changes in swine production systems and availability of parenteral ectoparasiticides, lice and thus swine pox are/is rare. However, infrequently congenital swine pox occur sporadically in some lice-free herds affecting few pigs in few litters with a high case fatality rate by 10-days-of age (Thibault et al., 1998, *Swine Health and Production*, 6: 276-278). This suggests that swine pox virus may be endemic in some swine herds.

Skin and subcutis
Dermatitis and cellulitis, gangrenous
Trauma, inoculation of bacteria or reactivation of *Clostridium* spp. spores
(Here 2 images of clostridiosis and 1 of a lesion resembling *Fusobacterium necrophorum*).

Subcutis
Abscess
*Streptococcus suis; Arcanobacterium pyogenes;* Others including *E. coli, Salmonella, Erysipelothrix.*

**Skin**
Porcine juvenile pustular psoriasiform dermatitis (*Pityriasis rosea*)
Idiopathic, resolves spontaneously. DD: dermatomycosis, exudative epidermitis, dermatosis vegetans, and swinepox.

**Skin**
Dermatitis, hyperplastic, crusting, chronic
Demodectic mange
*Demodex* spp.

**Skin**
Dermatitis, hyperplastic, crusting, chronic
Sarcoptic mange
*Sarcoptes* spp.

**Skin**
Parakeratosis
Zinc deficiency
DD: Chronic solar dermatitis (sunburn), zinc toxicity causes pancreatic necrosis (Gabrielson et al., 1996, Vet path 33: 692-696)

**Skin**
Melanoma
Duroc, usually young pigs, heavily pigmented, may be malignant. The local cellular immune response may play a crucial role in the regression of these tumors.

**Foot**
Pododermatitis, necrotizing
Penetrating nail
Common.

**Foot**
Pododermatitis (with abscess)
*Fusobacterium necrophorum*

**Foot**
Pododermatitis, coronary band, necrotizing and hemorrhagic (gangrenous)
Selenium toxicity
DD: vesicular diseases, sepsis, ergot.

**Foot**
Pododermatitis, solar, ulcerative, necrotizing
Cement chemical burns

**Foot**
Hoof cracks
Flooring inducing trauma. Fiberglass in this case.

**Foot**
Pododermatitis, chronic with hoof irregular hyperplasia

**Foot**
Pododermatitis, coronary, vesicular, ulcerative
The 4 viral vesicular diseases.
Foot and mouth disease picorna-aphtovirus; vesicular stomatitis rhabdovirus; swine vesicular disease picorna-enterovirus; vesicular exanthema vesivirus-calicivius.

Mammary skin
Dermatitis, papular
*Stomoxys calcitrans*, L.

Mammary skin
Dermatitis, locally extensive, ulcerative, (lichenoid)
Systemic Lupus Erythematosus-like ulcerative dermatitis in Belgian Landrace Sows
Lesions are located on ears, limbs, and in the mammary region and are resistant to treatment that included corticosteroid therapy.
Ulcerative dermatitis of sows is morphologically similar to pemphigus, pemphigoid, systemic lupus erythematosus, epidermolysis bullosa simplex, erythema multiforme, toxic epidermal necrolysis, and drug eruption. However, significant differences exist between ulcerative dermatitis and these conditions.

Mammary gland
Hamartoma

Skin of mammary gland
Dermatitis, ulcerative, chronic
Vesicular viral disease + bacteria

Mammary gland
Hamartoma

Skin
Dermatitis, ulcerative, granulomatous, necrotizing, with lymphocytes and plasma cells
Tuberculin reaction

Mammary gland
Mastitis, pyogranulomatous, ulcerative, chronic
Maduromycosis
Allesceria boydi
Limb, Subcutaneous bursa
Bursitis, serous
Trauma

Sternal bursa
Bursitis, serous
Trauma

**Lymphoid and Hematopoietic**

Spleen
Normal extruded red pulp

Spleen
Abscesses
Bacteria

Spleen
Congestion, severe.
Gastric torsion
Rare

Spleen
Torsion with severe passive hyperemia (infarction)

Spleen
Torsion with infarction, chronic with necrosis and capsular fibrosis

Spleen
Abscesses
*Arcanobacterium pyogenes*

Spleen
Infarcts
Classical Swine Fever (Hog Cholera)
*Pestivirus*

Spleen
Infarct, global
African Swine Fever
*ASF virus*


Classical Swine Fever (Hog Cholera, European Swine Fever). Acute virulent form: Pigs are pyretic with cutaneous cyanosis, conjunctivitis, anorexia, constipation followed by severe diarrhea (“cholera”), convulsions and death. Lesions include peripheral hemorrhage of lymph nodes, generalized vasculitis, tonsillar necrosis, splenic infarcts, serosal hemorrhages, button ulcers in colon. Subacute form: pyrexia, diarrhea, low mortality with few gross lesions. Reproductive form: mummified, stillborn and weakborn pigs, congenital tremors, cerebellar hypo- or aplasia, limb deformation, arthrogryposis (underlined are lesions that help differentiate from existing US diseases). Almost complete loss of lymphocytes associated with follicular necrosis of lymphoid tissues in pigs infected with highly virulent strains. B lymphocyte loss not prominent in pigs infected with less virulent strains. Increased activity of T lymphocytes with all strains. CSF antigen detected in tonsillar epithelial cells, macrophages, endothelial cells in lymphoid tissues Pulmonary intravascular macrophages have also been shown as target cells for CSFV infection and atypical cilia were observed in the bronchiolar epithelium. CSFV infects bone marrow haematopoietic cells, especially myelomonocytic precursors, and causes apoptosis. Thrombocytopenia is caused by massive
activation and subsequent phagocytosis of platelets secondary to the release of platelet-activating factors by activated macrophages infected with CSFV. Perivascular cuffing of mononuclear cells in the gray and white matter of the brain may be the most consistent microscopic lesion. Viral antigen has been detected consistently by IHC and in-situ in mononuclear cells of lymphoid tissues. Pigs infected with BVDV-2 might develop antibodies that cross-react in tests for antibodies against classical swine fever virus. In addition, pigs developed leucopenia and thrombocytopenia after infection with BVDV-2.

Spleen
Hyperplasia
_Eperythrozoon_ spp

Spleen
Mast cell tumor

Spleen
Lymphoma

Lymph node
Hemorrhage
Classical Swine Fever (Hog Cholera)
Pestivirus

Lymph node
Necrosis
_Salmonella_ spp.

Lymph node
Cavitations, multifocal
Autolysis or mesenteric emphysema at slaughterhouse

Lymph node, pharyngeal
Lymphadenitis with abscesses
_Streptococcus_ spp.

Lymph node
Lymphadenitis with abscess

Tonsils
Necrosis
African Swine Fever
ASF virus

Tonsils
Necrosis
Pseudorabies (Aujeszki disease, bulbar paralysis, mad itch)
_Suid herpesvirus 1_

Tonsils
Necrosis
Anthrax
_Bacillus anthracis_

Lymph nodes
Hemorrhagic necrosis
Anthrax
_Bacillus anthracis_
Tonsils
Necrosis
Lymphoma and *Salmonella* spp.

Lymph node, gastro-hepatic
Hemorrhage, severe
*African Swine Fever*
*ASF virus*
This lymph node is called “old reliable” since expresses changes even in mild forms of the disease.

Lymph node, renal
Hemorrhage, severe
*African Swine Fever*
*ASF virus*

Lymph node, renal
Hyperplasia
*African Swine Fever*
*ASF virus*

Lymph nodes, mesenteric
Lymphadenitis, caseous, granulomatous
*Tuberculosis*
*Mycobacterium avium*

Lymph node
Lymphadenitis, pyogranulomatous with hyperplasia
*Phycomycosis*
*Phycomycetes*

Lymph node
Lymphoma

**Digestive**

Piglet
*Atresia ani et recti*

Piglet
*Atresia coli*

Piglet
*Atresia coli cum megacolon*
*DD: bloat*

Rectum
Prolapse, partial with congestion
Dietary and any cause increasing abdominal pressure

Mouth
Cheiloschysis and palatoschysis

Tongue
Epitheliogenesis imperfecta
Tongue
Glossitis, vesicular, erosive and ulcerative
Viral vesicular diseases

Gums
Necrotizing (gangrenous) gingivitis and alveolitis
_Fusobacterium necrophorum_
Careless trimming of “milk” or “needle” teeth

Oral cavity
Erosive and ulcerative stomatitis with glossitis with parakeratosis
_Candida_ spp.
DD: Salmonellosis (without parakeratosis)

Esophagus (2)
Parakeratosis
_Candida_ spp.

Esophagus
Wire, foreign body

Stomach
Edema severe
Edema disease, emaciation, vasculitis, PCV-2.

Stomach
Gastric folds hyperplasia
Unknown cause

Stomach
Gastroesophageal ulcer, chronic
Acute hemorrhage, exsanguination; chronic hemorrhage and anemia. Costly problem in the swine industry. Risk factors include: gender (barrows), genotype, season (summer), particle size of feed, anorexia (concurrent disease). _Helicobacter heilmannii_ has been sporadically associated with ulcers, but the experimental infection failed. Lesions were reproduced using high card diets. _Helicobacter pylori_ causes lymphoplasmacytic gastritis in experimentally inoculated pigs similar to human disease.

Stomach
Ulcers, chronic
_Hyostrongylus rubidus_

Stomach
Ascaridiosis
_Ascaris suum_

Small intestine
Diverticuli

Small intestine
Diverticular rupture

Intestine
Petechiae, diffuse
DD: ASF, CSF, sepsis
Intestine
Displacements: volvulus, torsion, knotting, intussusception

Large intestine
Prominent lymphoid follicles (normal)

Piglet, small intestine
Rupture
Trauma, sow stepped on. Confirmed by focal hemorrhage on abdominal muscles.

DD: Diarrhea in swine:

<table>
<thead>
<tr>
<th>Without blood</th>
<th>With blood</th>
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<tbody>
<tr>
<td>Colibacillosis</td>
<td>Clostridium perfringens type C</td>
</tr>
<tr>
<td>Clostridium perfringens type A</td>
<td>Salmonellosis (dark digested blood)</td>
</tr>
<tr>
<td>Coccidiosis</td>
<td>Proliferative enteritis - PHE form</td>
</tr>
<tr>
<td>Viral enteritis</td>
<td>Swine dysentery</td>
</tr>
<tr>
<td>Proliferative enteritis (except PHE)</td>
<td>Whipworms</td>
</tr>
<tr>
<td>Whipworms</td>
<td></td>
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<tr>
<td>Intestinal spirochetosis</td>
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</tbody>
</table>

*Enterotoxigenic E. coli (ETEC).* Hemolytic or non-hemolytic, small intestine only, colonize via fimbria: sucking pigs - K88, K99, 987P, F41; weaned pigs - K88, F18ac (2134P), fluid accumulates due to enterotoxins (secretory diarrhea): LT (A and B units) activates adenylate cyclase $\uparrow$ cAMP > increased secretion of Cl, Na, HCO$_3$; water into lumen; STa & Stb activates guanylate cyclase $\uparrow$ GMP > inhibit Na/Cl cotransport system, Hpth.: Uniform colonization of the brush border.

*Attaching And Effacing E. coli (AAEC).* Uncommon in 1-6 week old pigs, “classic AE lesion”, small and large intestine, attach by eae gene product - 94 Kd protein “intimin”, verotoxin negative, Hpth.: Colonization and degeneration of villous enterocytes; “cobblestone” appearance of brush border.

Differential diagnoses for *atrophic enteritis* in pigs: TGE (Coronavirus), Rotavirus (A, C, B), Coccidiosis (*Isospora suis*). Less likely differentials: chlamydia, adenovirus, enteric calicivirus, astrovirus, parvovirus.

Small intestine
Catarrhal enteritis
DD: *Escherichia coli*; *Salmonella* spp; *Isospora suis*; rotavirus (combination of them, even all together).

Small intestine
Enteritis, fibrinous and necrotizing (diphtheritic)
*Salmonella* spp.

*Salmonella choleraesuis:* Causes severe septicemia +/- concurrent pneumonia or enterocolitis in weaned and grower pigs. Multifocal hepatic necrosis (paratyphoid nodules) is a fairly consistent lesion. *S. choleraesuis* replicates in macrophages as well as extracellularly in lymphoid tissues (causing necrosis) and elsewhere. Large amounts of systemic endotoxin activate cytokines and induce vascular damage (hemorrhage, interstitial pneumonia with edema, glomerulonephritis, gastric mucosal venous thrombosis and arterial thrombosis (skin of extremities and colon $\rightarrow$ ulcers).

Small intestine
Enteritis, necrohemorrhagic
*Clostridium perfringens*

Ileum
Enteritis (ileitis) regional (segmental), proliferative
*Lawsonia intracellularis*
Proliferative; proliferative and hemorrhagic; ulcerative.

Intestine
Serosal edema
Lymphadenomegaly due to histiocytic mesenteric lymphadenitis with lymphoid depletion
Enterocolitis, fibrinuous with Payer’s patch necrosis
Spiral colon mesenteric edema
PCV-2
DD: Salmonellosis, CSF.

Colon
Typhlocolitis, necrotizing and hemorrhagic ulcerative
*Brachyspira* spp.

There are at least 5 species of *Brachyspira* in swine: Strongly β hemolytic - *B. hyodysenteriae* (Swine Dysentery); Weakly β hemolytic – *B. pilosicoli* (Intestinal Spirochetosis), *B. innocens*, *S. intermedia* and *S. murdochii* (all 3 are nonpathogenic). They cannot be distinguished microscopically.

Colon
Edema, mesenteric, severe
Edema Disease
*Escherichia coli*
ETEEC - enterotoxemc *E. coli*: hemolytic colony types, somatic serotypes O139, O141 and O157, colonizes the S.I. via F18ab (F107) or K88 fimbria, secretes shiga-like toxin II variant (SLTIIvar) that induces characteristic systemic angiopathy. In the brain, lesions are most often in the brain stem. Hpth.: Vasculopathy; mural degeneration, mural and perivascular edema.
One study identified production of verotoxin SLTIIvar and expression of F18 and K88 colonization factors only in isolates of the serogroups O139, O141, and O157, respectively. The presence of the adhesin involved in diffuse adherence (AIDA) gene has been suggested as an important virulence factor.

Colon
Edema, mesenteric, severe
DD: Edema Disease, Clostridium difficile, PCV-2.

Rectum
Post-ulcerative fibrosis with stenosis
*Salmonella choleraesuis*
DD: congenital.
Megacolon is a possible complication.

Intestine
Granulomas
Acanthocephalisis
*Macracanthorhynchus hirudinaceus*

Cecum
Catarrhal typhlitis
*Trichuris suis*

Mesentery
Emphysema
Abattoir lesion. Gas bubbles in mesentery and mesenteric lymph nodes. Unknown cause.

Mesentery
Bony metaplasia
Common. Unknown cause.

Stomach
Lymphoma

Abdominal cavity
Lymphoma

Liver

Liver
Autolysis

Liver
Glisson capsule osseous metaplasia
Unknown cause. Sporadic.

Gall bladder
Cholelithiasis

Gall bladder
Cholecystitis, fibrinous
Salmonella spp.

Gall bladder
Edema
Systemic passive congestion, cardiogenic

Gall bladder
Edema, severe
African Swine Fever
ASF virus

Liver
Hemorrhagic necrosis
Hepatosis Dietetica
Vitamin E and Selenium responsive disease.
Associated with mulberry heart disease (cardiac hemorrhage), perhaps mycotoxins. Diets with cereals containing yeasts (Torula spp.) may predispose it.

Liver
Cholangiohepatitis, fibrinosuppurative with pseudomelanosis
Various bacteria

Liver
Icterus
PCV-2

Livers
Necrosis
PCV-2

Liver
Necrosis, multifocal (also enteric and pulmonary necrosis, multifocal)

**Pseudorabies**

*Suid herpesvirus 1*

Liver

**Hepatitis, necrosuppurative, multifocal (embolic)**

Listeriosis

*Listeria monocytogenes*

Liver

**Caseous, granulomatous hepatitis**

Tuberculosis

*Mycobacterium avium* complex

Liver

**Hepatitis, eosinophilic, fibrosing, chronic, multifocal**

*Ascaris suum* larval migration

Liver

**Ductus coledocus obstruction**

*Ascaris suum*

Liver

**Hydatidosis**

*Echinococcus granulosus*

Liver

**Fibrosis, multifocal with central fibrotic cores**

*Stephanurus dentatus* migration

Liver

**Hepatocellular carcinoma**

Liver

**B cell lymphoma**

Hepatic Lymph node

**B cell lymphoma**

Liver

**Lipidosis, cholestasis and necrosis**

Aflatoxins

Liver (cross section)

**Lipidosis, cholestasis and necrosis**

Aflatoxins

Liver

**Postnecrotic macronodular regeneration and diffuse fibrosis (cirrhosis)**

Carcinoma, presumptive

Aflatoxins

**Pancreas**

Necrosis and mineralization, multifocal

*Ascaris suum* migration
Urinary

Kidney
Renal aplasia

Kidney
Renal hypoplasia.
Related to one boar in this case.

Kidney
Hypoplasia and dysplasia, unilateral

Kidney
Cysts
Congenital

Kidneys
Hydrourether and hydrenephrosis, bilateral

Kidney
Pyelonephritis, hemorrhagic
Bacterial. In this case *Haemophilus* spp.

Kidney
Petechiae
CSF. DD: ASF, sepsis (e.g. *Salmonella* spp.)

Kidney
Petechiae (and tubular necrosis with histiocytic interstitial nephritis)
PCV-2 nephropathy
In this case only petechiae visible (DD as above).

Kidney
Perirenal hemorrhage
Vitamin K responsive disease
Heated and stored feed. DD: Amaranthus toxicity

Kidney
Infarcts
From valvular vegetative endocarditis

Kidney
Tubular necrosis (with numerous intranuclear lead inclusion bodies)
Lead toxicity
Ground up bedding material in this case

Kidney
Fibrosis, multifocal, linear (post necrotic)
Ochratoxicosis
Ochratoxins

Kidney
Papillary dehydration salts
Common finding in many species subjected to dehydration. Rehydration washes these out without residual effects.
Kidney
Edema and hemorrhage, bilateral, severe
*Amaranthus retroflexus* (pigweed) toxicity

Kidney
Hemorrhage and edema, bilateral, severe
*Athropa belladonna* (Nightshade, *Solanaceae*) toxicity, presumptive

Kidney
Mineralization
*Cestrum diurnum* toxicity
Certain plants contain glycosides of the active metabolite of vitamin D. The metabolite is called 1,25-dihydroxycholecalciferol or more simply 1,25-OHD3. Consumption of glycosides of 1,25-OHD3 by grazing animals leads to a vitamin D toxicity, which causes the deposition of excessive calcium in the soft tissues (calcinosis). Of the three rangeland plants, *Cestrum diurnum*, *Solanum malacoxylon*, and *Trisetum flavescens*, known to contain these glycosides only *Cestrum diurnum* is found in the U.S. primarily in Florida.

Kidney
Nephritis, uretheritis and cellulitis, chronic
*Stephanurus dentatus*

Kidney
Nephroblastoma
Young < 1 yr old; females > males, 4 types: nephroblastic and epithelial (most common), mesenchymal and mixed (less common)

Kidney
Mast cell tumor

Kidney
B cell lymphoma

Urinary bladder
Petecheia
Classical Swine Fever
Pestivirus

Urinary bladder
Hemorrhage, acute, multifocal, severe
CSF or ASF

Urinary bladder
Blood clot
Vitamin K responsive disease

Urinary bladder
Trigone area necrosis, rupture, edema and petechiae
Trauma in this case. Necrosis and rupture at the pole are more common and associated with lumbosacral myelopathies.

**Respiratory**

Nose
Osteonecrosis with bone loss and remodeling
Atrophic rhinitis
*Pasteurella multocida*
A cytotoxin produced by toxigenic strains of primarily capsular serotype D (rarely serotype A) of Pasteurella multocida is absorbed and causes bony hyperplasia in the nasal turbinates and physes of long bones by inhibiting osteoblasts, inhibiting chondrocyte proliferation and (most likely indirectly) stimulating osteoclasts.

Nasal cavity, turbinates
Rhinitis with severe edema (and megalocytes with intranuclear large herpetic inculsion bodies)
*Porcine cytomegalovirus*

Lung
Edema, severe
Moldy corn poisoning
Fumonisin B1, produced by *Fusarium moniliforme*
Fumonisin inhibits sphingosine- and sphinganine-N-acyltransferase causing elevated levels of sphingosine and sphinganine in serum and tissues. There are ultrastructural changes in endothelial cells.

Primary pulmonary pathogens:
- Bacterial: *M. hyopneumoniae, A. pleuropneumoniae, B. bronchiseptica, S. choleraesuis, A. suis*
- Viral: *PRV, SIV, PRCV, PRRSV, PCV2*

Secondary pulmonary pathogens:
- Bacterial: *P. multocida, S. suis, H. parasuis, A. pyogenes*, others…..

Lung
Lobular (bronchointerstitial) pneumonia and cranioventral bronchopneumonia
Influenza
*Orthomyxovirus* and *bacteria* (e.g. Mycoplasma)
Swine influenza: Detected mainly in the bronchial and bronchiolar epithelial cells by in-situ and IHC and a less intense signal was detected in the interstitial and alveolar macrophages.

DD: Interstitial pneumonia in swine:

Viral: Pseudorabies (PRV)  Septicemic: S. choleraesuis  Allergic: Ascarid larval migration
- Swine Influenza (SIV)  H. parasuis
- Porcine Respiratory Coronavirus (PRCV)  S. suis
- PRRS virus (PRRSV)  other
- Porcine circovirus type 2 (PCV2)

Paramyxovirus from pigs with interstitial pneumonia, necrotizing bronchiolitis and encephalitis.

Post-weaning Multisystemic Wasting Syndrome (PMWS): A wasting syndrome affecting 5-15% of weaned pigs associated with porcine circovirus type 2 (PCV2). The most characteristic lesion in PMWS is histiocytic (granulomatous) inflammation with or without unique globular intracytoplasmic viral inclusion bodies in macrophages. Less consistent lesions include interstitial pneumonia, interstitial nephritis, myocarditis, hepatitis (with hepatic atrophy and/or icterus) and perivasculitis in a number of. Liver lesions have been identified as a frequent finding and are the most likely cause of icterus and wasting. PMWS has been associated and reproduced with combined PCV2 and porcine parvovirus inoculation and combined PCV2 and PRRSV infection and M. hyopneumoniae. PMWS been reproduced in gnotobiotic pigs with PCV2 alone following administration of keyhole limpet hemocyanin in incomplete Freund’s adjuvant and has been reproduced with PCV2 alone in cd/cd pigs. Intramuscular injection of pigs with a vaccine against
Mycoplasma hyopneumoniae or a nonspecific immunomodulating drug (Baypamun) caused clinical signs, moderate to severe gross and histopathological lesions of PMWS. There are also breed-dependent differences to PCV2 associated disease and lesions. Vaccination with selective bacterins increased the severity of lesions in conventional pigs infected with PCV-2. PCV2 was also associated with transplacental infection of fetuses that were aborted and had myocarditis. Transplacental transmission of PCV2 has been shown experimentally and virus concentration were highest in the heart.

Pseudorabies (Aujeszky’s Disease): Clinical signs and lesions vary according to age. In suckling pigs, mortality is high and is associated with nervous clinical signs (nonsuppurative encephalomyelitis with a few neutrophils) and multifocal necrosis in the parenchyma of organs (tonsil, lung, liver, spleen). In nursery, growing, finishing and adult swine, mortality is lower and is associated with respiratory clinical disease and lesions (rhinitis, laryngotracheitis, interstitial pneumonia). CNS clinical signs are less common, although microscopic lesions in the CNS are common. Late-term abortions occasionally in epizootics. Lesions uncommon in fetuses – same as for neonate.

PRRS: Consistent gross lesions are in lungs and lymph nodes only. Microscopic lesions: interstitial pneumonia with aggregates of necrotic alveolar macrophages in alveoli, lymphoid necrosis followed by nodular lymphoid hyperplasia in lymphoid organs, lympho-plasmacytic myocarditis and mild nonsuppurative encephalitis. Syncytial cells are more likely the result of PCV2 infection. Differentials for lympho-plasmacytic myocarditis in aborted or suckling pigs include porcine parvovirus and porcine circovirus. A proliferative vasculopathy has been described in aborted piglets. PRRSV induces apoptosis in infected and bystander cells including macrophages (histiocytes, tingible body macrophages and pulmonary intravacular macrophages), alveolar pneumocytes and epithelial germ cells in the seminiferous tubules. PRRS viral contamination of semen is due to PRRSV- infected epithelial germ cells, spermatocytes, macrophages and PRRS viral contamination of the cell-free fraction. Tonsil biopsies can be used for detection of persistently PRRSV-infected breeding age gilts by PCR.

Lung, Kidney
Hemorrhage and necrosis, multifocal
Systemic salmonellosis
Salmonella choleraesuis

Lung
Hemorrhage, multifocal
Sepsis

Lung
Necrosis, multifocal and edema
Embolic pneumonia
Bacterial emboli from valvular vegetative endocarditis

Lung
Bronchopneumonia hemorrhagic, multifocal, with diffuse edema
Actinobacillus pleuropneumoniae
Areas of edema with underlying not easily seen bloody firm lung

Lungs
Bronchopneumonia hemorrhagic and fibrinous pleuritis (pleuropneumonia)
Actinobacillus pleuropneumoniae
Distribution varies: multifocal, cranioventral, dorsal, bilateral, unilateral
DD: Pasteurella spp., Bordetella spp.

Lung
Bronchopneumonia, cranioventral, unilateral
Bordetella spp.
Lung
Bronchopneumonia with abscesses
*Arcanobacterium pyogenes*
Common isolate from swine. Usually is an environmental contaminant of wounds, causing a localized purulent infection followed by bacteremia resulting in vegetative valvular endocarditis, purulent arthritis, embolic abscessing pneumonia, ascending urogenital infections or other localized pyogenic infection. It is also a common opportunistic secondary pulmonary pathogen.

Thoracic and abdominal cavities
Polyserositis, fibrinous
*Glasser Disease*
*Haemophilus parasuis*

Thoracic and abdominal cavities
Polyserositis, fibrinous and bronchopneumonia
*Glasser Disease*
*Haemophilus parasuis*

Fibrinous polyserositis: In suckling pigs: S. suis is the most common cause and E. coli is a sporadic cause when there is inadequate intake of colostrum. In weaned pigs: Differentials include H. parasuis (Glasser’s disease), S. suis and M. hyorhinis. Although all 3 can cause meningitis in weaned pigs, clinical CNS disease is usually a consistent feature in only S. suis infections.

H. parasuis can cause an acute septicemia that resembles septicemic Salmonellosis. H. parasuis more commonly causes polyserositis, polyarthritis and meningitis (Glasser’s disease) in weaned pigs.

Neurological clinical signs are uncommon in weaned pigs with Glasser’s disease. Occasionally, H. parasuis causes acute outbreaks of highly fatal fibrinosuppurative leptomenigitis in young adult replacement breeding stock shortly after entry into recipient herds. H. parasuis also causes eustachitis and temporary otitis media is suggested as predisposing to ascending secondary pyogenic bacterial otitis media.

*Streptococcus suis:* There are at least 35 capsular serotypes in pigs. Disease is most common in suckling and recently weaned pigs, but can occur in any age. Fibrinopurulent leptomenigitis causing CNS clinical signs and high mortality is common. Septicemia with or without fibrinous polyserositis or leptomenigitis is also common. When fibrinous polyserositis predominates, S. suis septicemia is difficult to differentiate from Glasser’s disease. In general, the amount of fibrin and the severity of peritonitis are greater with Glasser’s disease than with S. suis septicemia. Has assumed greater importance since PRRS has become ubiquitous in swine populations. Acute PRRS predisposes to S. suis induced septicemic diseases. Like H. parasuis, S. suis also may cause acute highly fatal outbreaks of leptomenigitis in young replacement breeding swine shortly after introduction into recipient herds. The tonsils as possible portals of entry for S. suis serotype 2. S. suis serotype 2 bacteria are frequently identified immunohistochemically in the regional lymph nodes of the upper respiratory tract, possibly reflecting primary lymphogenous spread from the tonsils.

*Actinobacillus suis* causes sporadic outbreaks of fulminant embolic septicemia in all ages of pigs. In suckling and recently weaned pigs, most present as acute death. Those with clinical signs have fever and multifocal cutaneous hemorrhages. Occasionally, pigs may be lame, exhibit dyspnea or have nervous signs. In finishing-age pigs, most pigs are also found dead; however, in sick pigs the primary clinical manifestation is respiratory disease characterized by pyrexia, dyspnea and cyanosis. In adults (and sometimes younger animals), disease is less often fatal and resembles erysipelas. Sick adults typically are pyretic, anorectic and depressed with raised red rhomboid skin lesions typical of erysipelas. Some adults are found dead and occasionally sows abort. Lesions in all ages are the consequence of septicemia with septic embolism. Petechial hemorrhages are diffusely distributed on serosal surfaces and a wide variety of organs including lungs, kidneys, spleen and skin. Common lesions also include necrohemorrhagic pneumonia and serofibrinous pericarditis, pleuritis and peritonitis. Less common lesions include fibrinous arthritis, rhomboid cutaneous infarcts, meningitis and myocarditis. In pneumatic lungs, affected areas of necrosis, hemorrhage and fibrin deposition are multifocal and randomly distributed, suggesting a
hematogenous origin. However, these pneumonic foci may coalesce until lung lesions are grossly indistinguishable from those caused by APP.

Diaphragm
Hypertrophy
Compensatory secondary to severe chronic pneumonia

Lung
Pneumonia, granulomatous
Tuberculosis
*Mycobacterium avium* complex

Lung
Necrosis, multifocal with edema
*Toxoplasma gondii*

Lung
Bronchopneumonia, lymphocytic and eosinophilic
*Metastrongylus elongatus* (syn. *M. apri*)

Lung
Edema, severe
*Ascaris suum* larval migration

Lung
B cell lymphoma

Lung
Melanosis maculosa

**Cardiovascular**

Heart
Subaortic stenosis

Heart
Dilatative cardiomyopathy with systemic passive congestion

Heart
 Multifocal to coalescing necrohemorrhagic myocarditis with mineralization
Mulberry Heart Disease
Vitamin E and selenium responsive disease
Pigs range in age from 3 to 7 weeks, concentrations of Vit. E below 2 ppm are deficient, often selenium concentrations are within limits. DD: EMCV; fetuses neonates (PPV, PRRSV, PCV2).
Other related conditions: Hepatosis dietetica; exudative diasthesis, hemolytic anemia, necrotizing steatitis

Heart
Necrosis and mineralization
Encephalomyocarditis Virus (EMCV); viral myocarditis (multifocal necrosis and mineralization, not hemorrhage), EMCV also causes necrotizing pancreatitis and tonsilitis.

Heart
Endocarditis, valvular, fibrinous, chronic
Bacterial infection seeding on a dysplastic or misshapen valve
*Streptococcus suis, Escherichia coli, Erysipelothrix rhusiopathiae, Arcanobacterium pyogenes*, others
Septic emboli to other organs including lung, spleen, kidneys.

Heart
Rupture of the base of the aorta
Copper deficiency
Piglets on a low copper milk diet (cow milk)

Heart
Cysticercosis
*Cysticercus cellulosae (Taenia solium)*

Heart
Sarcocystosis
*Sarcocystis suihominis*

Heart
Hamartoma, rhabdomatous

Heart
Choristoma, epithelial

**Muscular**

Skeletal muscle
Necrosis and mineralization
White Muscle Disease (Enzootic Myopathy)
Vitamin E and selenium responsive disease

Skeletal Muscle
Necrosis
Porcine Stress Syndrome
Group of conditions associated with a recessive gene. The group includes acute stress and sudden death (malignant hyperthermia), pale soft exudative muscle (PSE), dark firm dry meat, and back muscle necrosis. Heavy muscled pigs are more likely to carry the gene than leaner pigs. The gene is called the halothane gene because of the adverse effect halothane anesthetic has on pigs carrying it. Each pig is homozygous (i.e. possessing a pair of halothane genes), or heterozygous (i.e. possessing one normal gene and one halothane gene) or two normal genes. Homozygous pigs or their meat may show any of the four conditions. Homozygous (but not heterozygous) pigs can be identified by their response to the anesthetic with halothane. Recent developments have produced a gene probe that identifies both the homozygous and heterozygous carriers using only a drop of blood or a single hair. Back muscle necrosis is a more localised form of PSS. When the homozygous state is present and following a period of muscle activity, there is a change in muscle metabolism from aerobic to anaerobic and biochemical abnormalities develop. The body tissues become acid with a marked rise in temperature (42C).

Skeletal muscle
Hemorrhagic necrosis (gangrene)
Black leg
*Clostridium chauvoei*

Skeletal muscle
Cysticercosis
*Cysticercus cellulosae (Taenia solium)*
Adipous tissue

Body as whole
Emaciation

Adipous tissue
Steatitis
Yellow fat disease
Vitamin E responsive disease

Skeletal

Piglets, body as whole
Arthrogryposis and twisted and malaligned bones, scoliosis, rib cage deformity
Poison hemlock (Conium maculatum) – gamma-coniceine

Skeletal malformations were induced in newborn pigs from gilts fed C. maculatum seed or plant during gestation days 43 through 53 and 51 through 61. The teratogenic effects in groups dosed during gestation days 43 through 53 were more severe than those in groups dosed during the later period, with many newborn pigs showing arthrogryposis and twisted and malaligned bones in the limbs and with 1 pig showing scoliosis and deformity of the thoracic cage. The pigs born to gilts given C. maculatum during gestation days 51 through 61 had excessive flexure primarily in the carpal joints, without scoliosis or bone malalignment in the limbs.

Piglets, body as whole
Complete amelia
Cause unknown

Piglet, limbs
Polydactilia

Piglet
Forelimbs hyperostosis
Porcine hyperostosis
Rare, sire related. Atherosclerotic changes in blood vessels.

Body as whole
Hind limb paralysis
Spinal abscess at T9

Bones
Congenital porphyria (Gunther’s disease)
Extremely rare and autosomal recessive. The deficient enzyme is uroporphyrinogen III cosynthase (or uroporphyrinogen III synthase). Porphyrins are markedly increased in bone marrow, red blood cells, plasma, urine and feces. Porphyrins are also deposited in the teeth and bones.

Head, mandibles and nasal bones
Fibrous osteodystrophy
Abnormal calcium/phosphorus ratio in feedstuffs. Bran disease, Panicum spp.

Head
Osteopetrosis
Numerous functional defects have been observed in osteoclasts of osteopetrotic animals including: 1. Lack of hydrolytic enzymes in the bone-osteoclast interface or the inability of osteoclasts to discharge lysosomal and oxidative enzymes in the extracellular spaces. 2. Partial or complete absence of ruffled borders on the osteoclasts. The brush border is considered the cytoplasmic structure which allows bone resorption. 3. Reduction in the number of osteoclasts.
Head, frontal sinus
Improper use of captive bullet

**Articular**

Joint
Arthrosynovitis, chronic, villous
Bacterial: *Streptococcus suis, Mycoplasma, Erysipelothrix, E.coli, Salmonella.*

**Endocrine**

Pituitary gland
Abscess

**Genital**

Young sows
Vulvar edema
Zearalenone toxicity

Ovaries
Cysts
Rete ovarii

Reproductive tract
Pseudohermaphrodite

Reproductive tract
Hermaphrodite

Uterus
Horn duplication

Uterus
Horn segmental aplasia

Uterus
Leiomyoma
Very common in old sows

Piglet
Segmental hyperemia and hemorrhage
Dystocia

Allantois
Cysts
Normal

Fetus
Umbilical cord knotting with hemorrhagic necrosis
Fetus
Umbilical cord torsion with hemorrhagic necrosis

Aborted fetuses
Progressive fetal death with mummification
DD: any infectious disease, such as SMEDI picornavirus, Leptospira, Japanese encephalitis flavivirus, others.

Testis
Hypoplasia

Testicular damage can be caused by anabolic treatments with the beta2-adrenergic agonist clenbuterol. Clenbuterol treatment causes an increased volume fraction of the testicular interstitium especially in the Leydig cell population.

Testis
Orchitis, marked, unilateral, necrotizing and granulomatous
*Brucella suis*

Testis and tunicae
Periorchitis, chronic
Torsion

Preputial diverticulum
Diverticulitis, chronic, ulcerative
*Mycoplasma* spp.

Perineum
Abscess
Postcastration complication

Perineum
Hernia

**Neural**

Piglet, head
Exencephalocele

Piglet
Dicephalus

Fetus
Hydrocephalus and holoprosencephalic anomaly with cyclopia

Brain
Malacia with cavitations
Edema disease

Mid and inner ear
Otitis media and interna, suppurative, chronic
DD: *A. pyogenes, P. multocida, M. hyorhinis*

Brain
Meningitis, suppurative
*S. suis*; others
Brain
Meningitis, granulomatous and caseous
*Mycobacterium avium complex*
Spinal cord
Poliomyelitis

DD: Posterior Paralysis or Paresis:
Spinal cord: enteroviral poliomyelitis (histo.: lymphoplasmacytic poliomyelitis), selenium intoxication (histo.: Bilateral poliomyelomalacia, ventral horns) – poliomyelomalacia (also: anorexia, alopecia, separation of hoof and skin at coronary band, degenerative changes in liver and kidney), fibrocartilagenous emboli and infarction secondary to disk rupture, contusion secondary to spinal fracture, lymphosarcoma
Spinal column: vertebral osteomyelitis or osteomalacia with secondary spinal fracture, fracture of lumbar spinal cord secondary to lightning.
Bones and Muscles: Ischial epiphysiolysis, rupture of the “hamstring”, fractures +/- osteomyelitis or osteomalacia, arthritis
Peripheral nerves: organic arsenical intoxication, sciatic damage from injections
A vestibulocerebellar disorder has been described in pigs after consumption of broken rice contaminated with *Aeschynomene indica* seeds.

DDX: Frequent causes of CNS disease in swine:

<table>
<thead>
<tr>
<th>Suckling Pigs:</th>
<th>Weanling pigs:</th>
<th>Grower/Finisher &amp; Adult</th>
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<td>Hypoglycemia</td>
<td>Streptococcal meningitis</td>
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<tr>
<td>Streptococcal meningitis</td>
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<td>Pseudorabies</td>
<td>Pseudorabies</td>
<td>S. choleraesuis meningitis</td>
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<tr>
<td></td>
<td>Water deprivation</td>
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