EQUINE PROCEEDINGS

June 5–7, 2022

2022 ANNUAL CONFERENCE

College of Veterinary Medicine
Kansas State University

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EQUINE PROCEEDINGS

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Veterinarian's Role at Rodeo and Equine Events

Dr. Chris Blevins, Kansas State University

Roles of a Veterinarian at a Rodeo or Equine Event

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Horses are athletes and used all over the world for events. Veterinarians are used in multiple aspects for these events. Many equine veterinarians are used during the pre- and post- event time to optimize the equine performance with medical, surgical and rehabilitation patient needs. During the event, veterinarians are also used to advocate for the animals and aid in their specialized needs. In addition, they need to have an understanding of the events and how the animals perform. The events (i.e. rodeo or other equine show) have veterinarians help with a vast array of categories/responsibilities.

During a rodeo (Professional Rodeo Cowboys Association, Women Professional Rodeo Association), the veterinarian's responsibility is to advocate for animal welfare at the event, and aid in treating any animal injuries during the rodoe. The rodeo associations have implemented multiple rules that govern the treatment and use of the animals at rodeos, and can be found on their website for the rodeo veterinarian to become familiar with. In addition, during and after the event the veterinarian could be the spokesperson for the public and media in discussion of animal safety and welfare.

The importance of animal welfare and the intense flow of events aid to develop a standard operating procedure (SOP) for the event. This allows for a better understand and benchmark for large number of staff/volunteers at the rodeo.

Rough stock bucking horses and bulls are treated and managed different than rough stock steers and calves. Equine performance horses at the rodeo are managed and treated different than the rough stock. Safety and equipment/medications are different for each these different species. As a veterinarian works on these animals, he or she must know these differences and how to manage each one. The event and animal used will have areas of focus for injury or medical concerns that can be outlined in the SOP. A crucial role for each patient seen is the communication with the owner, rodeo contractor and staff.

During horse shows and other equine events, a veterinarian will be used in similar fashions as rodeos. However, drug use in the patient must be understood and communicated with the show office. Multiple drugs have withdrawal times or banned from giving to a horse performing at a

show. An additional job and separate entity at shows are veterinarians that test animals for banned substances. The drug and medication group at the equine event can test animals before and after an event.

Emergency care and first aid treatment at an equine event/rodeo are also requested by the veterinarians. To be ready for these scenarios a veterinarian will be stationed in an office, trailer and/or veterinary vehicle at the event. Being prepared for critical patients in the arena or stall side is important. Emergency bags and kits for the event/show are important for time sensitive treatment.

Disease control and outbreaks are also handled by the rodeo and show veterinarian. In addition to treating a sick animal, the veterinarians need to determine the cause and if the pathogen is contagious to other horses (or people). Biosecurity measures include testing, treatment and quarantine. These measures are also implemented by the show/rodeo veterinarian. A biosecurity plan also can be discussed and implemented with the show/rodeo staff to help make a quarantine area. In addition, roadblocks for traffic used by state or federal authorities during outbreaks or high suspect foreign animal diseases can be implemented.

In high traffic and publicized events, the veterinarians will also need to have good communication skills and possibly media training. A lot of things are asked of the veterinarian and multiple eyes are watching every move of the event. Being able to communicate different scenarios to the staff and public aid in the outcome of the patient and the event.

It is very important for veterinarians to be a part of equine sporting events. They advocate for animal welfare and treatment of the sick or injured. Just as veterinarians are leaders within a community they also can be great leader for these animal event regionally, nationally and worldwide.

EQUINE PROCEEDINGS

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Endocrinology of Donkeys and Mules

Dr. Ramiro Toribio, The Ohio State University

Donkey Endocrinology

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Summary

The evolution of the donkey occurred under harsh environmental conditions, leading to adaptations to handle water deprivation better than horses, digestion of poor-quality feedstuff, a good capacity to accumulate adipose tissue, and efficient fat mobilization under increased energy demands or food scarcity. These features also predispose donkeys to gastrointestinal disorders, obesity, dyslipidemias, insulin dysregulation, metabolic syndrome, pituitary pars intermedia dysfunction, and endocrinopathic laminitis. Endocrine and metabolic disorders are common but underdiagnosed in donkeys. Their stoic nature also complicates early detection of clinical abnormalities. Donkeys have different hormone dynamics and reference values than horses, and some testing protocols may not apply to donkeys. This could influence diagnosis and treatment. Therefore, an overview on donkey endocrinology for which information is scarce will enhance our diagnostic, therapeutic and prognostic options.

Introduction

The donkey plays important economic, social, and cultural roles throughout the world, particularly in developing countries. In these countries, donkeys are valuable assets, central to the livelihood of families and local economies. In contrast, in developed countries, these animals are mainly used for recreational activities, ecotourism, hippotherapy/onotherapy, and as companion.[1] Donkeys are also used as a source of food (meat, milk), byproducts for medical conditions, and for the cosmetic industry. The high demand for donkey hide from China to produce "Ejiao" and other products has led to a decline in the worldwide donkey population.[2,3]

The number of donkeys and mules being admitted to veterinary hospitals or receiving specialized care has been increasing in recent years. Thus, there is a need to have better donkey and mule-specific veterinary knowledge. Practitioners should be familiar with anatomical, physiological, endocrine and pharmacological differences between donkeys and horses because extrapolating from horses can result in misdiagnosis, inadequate treatment, complications, unnecessary expenses, even death.[1,4-6] Due to their unique features and longevity, endocrine and metabolic disorders are common, but underdiagnosed in donkeys.

Donkey Metabolic Syndrome

Donkey metabolic syndrome (DMS) or asinine metabolic syndrome (AMS) is recently recognized condition that appears to be highly prevalent in donkeys, in particular in developed countries,

where food is readily available, or physical activity minimal.[1] DMS shares key features with equine metabolic syndrome (EMS), including obesity, insulin dysregulation (ID), and endocrinopathic laminitis.[7] Like horses and ponies, not every obese donkey has DMS and lean animals can be affected. The incidence of DMS is higher in jennies and middle to old age donkeys (>8 years of age);[1] however, it can affect younger donkeys.

A multitude of factors participate in the pathogenesis of ID and endocrinopathic laminitis in donkeys. Their energy efficiency combined with reduced physical activity promote obesity, which is a major complicating factor for endocrine disorders. Adipose tissue produces hormones and cytokines that interfere with insulin signaling and promote a systemic pro-inflammatory state.[8] Prolonged hyperinsulinemia and inflammatory factors disrupt lamellar integrity, which combined with excessive body weight contribute to endocrinopathic laminitis. Insulin concentrations are often elevated in obese donkeys.[9] Reduced insulin sensitivity interferes with glucose uptake, promotes fat mobilization (lipolysis), and alters endothelial integrity. Inflammatory cytokines also reduce insulin signaling in the liver, worsening hepatic fatty infiltration. Adipocyte endocrine factors such as adiponectin and leptin may be involved in the pathogenesis of ID and endocrinopathic laminitis, although information is lacking.

Diagnosis

The diagnostic principles of DMS are similar to EMS. Donkey-specific body condition scores (BCS) have been developed,[10,11] ranging from 1 (very thin) and 5 (obese)[10] or from 1 (emaciated) to 9 (obese).[11] A neck score system was also developed (0 = thin neck, no palpable crest; 4 = thick neck, rounded, gross cresty neck).[12] In addition to phenotype and clinical examination, fasting glucose and insulin concentrations, as well as dynamic tests are the methods used to diagnose DMS. A number factors could hamper the diagnosis of ID including stress, physical activity, pain, transport, fasting time, carbohydrate rich diets, endocrinopathies (PPID), concomitant diseases, and α2-adrenoreceptor agonists (e.g. xylazine).[7,13-15]

Fasting insulin concentration with a cut-off value of >50 µIU/mL is suggestive of ID.[13] Insulin concentrations differ between assays and extrapolation can be misleading.

Dynamic tests can be used when baseline insulin results are equivocal (20-50 µIU/mL). These protocols differ between donkeys and horse.[16]

Intravenous glucose tolerance test (IVGTT): glucose (300 mg/kg, 50% dextrose solution) is administered IV as a bolus and glucose and insulin concentrations measured at 0 (baseline), 5, 15, 30, 45, 60, 75, 90, 105, 120, 150, 180, 210, 240 and 300 min. In insulin sensitive donkeys, glucose should be back to the normal range by 180 minutes. This protocol takes time, defeating practicality.

Combined glucose-insulin test (CGIT): glucose (150 mg/kg, IV) is administered as a bolus and immediately followed by regular human insulin (0.1 IU/kg, IV). Glucose and insulin concentrations are determined at the same time points as the IVGTT. There is a practical version of the test, where donkeys are considered ID if glucose concentrations are at or above baseline values at 60 minutes (for horses this value is 45 minutes). The test can be simplified by collecting

blood samples at baseline and at 60 minutes. A serum insulin concentration of >100 µIU/mL at 60 minutes supports ID.

The intravenous insulin tolerance test (IVITT) and frequently sampled intravenous glucose tolerance test (FSIGTT) are mainly used for research.[1,17]

Oral carbohydrate tests: The oral sugar test (OST) and oral glucose test were recently evaluated in donkeys demonstrating differences with horses.[18] These tests need additional evaluations, but are promising. The OST is very easy to perform, where corn syrup (Karo®) is administered and insulin measured around 60 minutes later.

Leptin and adiponectin cut-off values for donkeys with evidence of ID and endocrinopathic laminitis have not been reported.

Treatment

Management of DMS is based on principles established for horses, focusing on weight loss, improving insulin sensitivity, and controlling endocrinopathic laminitis. Caloric restriction and increased physical activity are central for weight loss. Due to their energy efficiency, dietary management should be based on low quality hay, no grain and no access to carbohydrate-rich pastures. Commercial diets to manage ID and obesity in horses may not be indicated for donkeys because of their energy efficiency. Weight loss must be very slow due to risk of hyperlipemia. Levothyroxine sodium can be used at equine doses (0.1 mg/kg, q 24 h, PO). Metformin (15-30 mg/kg, q 12 h, PO) can be administered to donkeys with severe metabolic and endocrine disturbances (hyperglycemia, hypertriglyceridemia, hyperinsulinemia) or that are not responding to standard treatment.

Pituitary Pars Intermedia Dysfunction

Pituitary pars intermedia dysfunction (PPID) is common in geriatric donkeys, partly due to their longevity. PPID is underdiagnosed and epidemiological information is lacking.[1] Gender and breed do not appear to be risk factors.[1] The pathogenesis of PPID in donkeys is similar to horses and ponies,[1,9,19] result of hypothalamic damage that results in excessive proliferation of cells (melanotropes) in the pars intermedia.[20] These cells produce excessive amounts of pro-opiomelanocortin peptides, including adrenocorticotropic hormone (ACTH) and melanocyte-stimulating hormone (α-MSH). These imbalances alter the function of other organs resulting in a multitude of clinical abnormalities.

Clinical signs of PPID are similar between donkeys, ponies and horses. These include hypertrichosis, weight loss, muscle wasting, fat redistribution, laminitis, reproductive problems, secondary infections, endoparasitism, and orthopedic problems. Some donkey breeds have long haircoats, which can create confusion. Laminitis is a consistent finding.[19,21]Lethargy could be hard to assess due to their nature.[1] Polyuria is infrequent in donkeys.

Diagnosis

Plasma basal ACTH concentrations and the thyrotropin-releasing hormone (TRH) stimulation test

are the main diagnostic tests for the diagnosis of PPID in donkeys.[1,9]

Baseline ACTH concentrations in healthy donkeys are higher than in healthy horses and mules.[9] Like horses, donkeys have seasonal variations in ACTH concentrations that must be considered when measuring baseline ACTH or performing the TRH-stimulation test.[22] Seasonal ACTH values (donkeys: August-October = 19.5-143 pg/ml; November-July = 5.0–55.4 pg/ml; mules: August-October = 9.8–68.7 pg/ml; November-July = 5-37.1 pg/ml).[22] Plasma ACTH values in the fall higher in donkeys compared to horses.[23] However, basal cortisol concentrations are similar between healthy donkeys and horses. High cortisol is rare in donkeys with PPID and has minimal diagnostic value. Blood samples for ACTH should be collected in EDTA tubes and shipped on ice. Stressful conditions and drugs (e.g., α2-adrenoreceptor agonists, glucocorticoids) could alter ACTH concentrations. The dexamethasone-suppression test is not reliable and should be avoided in donkeys.[9,24,25]

TRH-stimulation test: collect baseline sample, inject 1 mg of TRH intravenously, and a second sample at 10 minutes. A donkey is considered PPID positive if plasma ACTH concentrations are > 110 pg/mL at 10 minutes in non-fall months.[9]

Treatment

Pergolide (dopamine D2 receptor agonist) is the drug of choice to treat PPID in donkeys.[26] A dose of 0.25-0.5 mg/250 kg, q24 h, PO often results in clinical improvement. In animals that develop anorexia, reduce dose or discontinue temporarily. Repeated basal ACTH measurement to assess response to treatment and adjust dosing.

Hyperlipemia

Hyperlipemia results from increased triglyceride levels (hyperlipidemia).[1,27] It is more frequent in donkeys than other equids due to their efficiency to store lipids and rapid ability to mobilize fat stores.[1] Hyperlipemia affects donkeys regardless of body condition or age, but it is more frequent in older donkeys, jennies and small donkey breeds.[1,27,28]

Predisposing factors: a negative energy balance (anorexia, fasting), increased energy demands (pregnancy, lactation), and diseases (stress, diarrhea, colic, endotoxemia, parasites, liver disease, laminitis).[1,27,28] Stress, obesity, and pregnancy are major risk factors. Mortality can be up to 80%.[1,27,28]

Conditions that increase energy demands activate hormone-sensitive lipase in adipocytes to induce lipolysis and the release of free fatty acids (FFA) into circulation.[1] In the liver, FFA are re-esterified into triglycerides to be released as very-low-density lipoprotein (VLDL) into systemic circulation. If VLDL production exceeds tissue uptake of triglycerides, hyperlipidemia (hypertriglyceridemia) ensues, with hepatic fatty infiltration, liver dysfunction, and risk of liver rupture. Other organs (kidneys, pancreas, heart, intestine, skeletal muscle) can be infiltrated by triglycerides, altering their function.[1,27,29]

Glucocorticoids, catecholamines, ACTH, growth hormone, glucagon, and cytokines (IL-6, TNF-

α) increase lipolysis, while insulin inhibits lipolysis and promotes lipogenesis. Increased levels of lipolytic factors (cytokines, hormones), decreased lipogenic hormones (insulin), and insulin insensitivity combined with decreased VLDL removal will lead to hyperlipemia. Because insulin is the main lipogenic hormone, impaired signaling (resistance) facilitates hyperlipemia, in particular in donkeys with DMS and obesity.

Hyperlipemia is secondary to other disorders and clinical signs are the result of the primary problem.[1,27-29] Hyperlipemia is often not diagnosed. Lethargy and anorexia are common findings of hyperlipemia. Fatty infiltration of various organs can alter their function, exacerbate disease progression, additional clinical signs (e.g., diarrhea, dysrhythmias) and laboratory abnormalities (e.g., increased liver enzymes, azotemia, acidemia, hyperbilirubinemia).[1]

Diagnosis

Hyperlipemia is diagnosed by measuring serum triglyceride concentrations. Gross lipemia can be noted with triglyceride concentrations >500 mg/dL in sitting EDTA and serum tubes. Severe hyperlipemia is evident when triglyceride concentrations are >1000 mg/dL.

Treatment

Therapeutic principles are aimed at controlling primary disease, reducing fat mobilization, halting hepatic triglyceride synthesis, avoiding stress, and restoring a positive energy balance.[1] In pregnant jennies, provide a good caloric intake, although this can also predispose to other disorders (ID). In extreme cases, pregnancy termination should be considered. In lactating jennies, early weaning is recommended. Hyperlipemic donkeys should be encouraged to eat by offering a variety of palatable foodstuff that may stimulate hunger (e.g., honey, apples, carrots). Placing a feeding tube should be considered if gastrointestinal function is normal. Intravenous dextrose may be required to decrease lipolysis. Parenteral nutrition without lipids may be indicated.[1,30] When hyperglycemia persists or triglyceride levels continue to increase over 24 h, regular insulin (0.05-0.1 IU/kg/h, IV) or slow release insulin (0.10-0.15 IU/kg, q 12-24 h, SQ) should be considered.[1,30] An insulin continuous rate infusion (CRI; 0.05 IU/kg/h starting rate) combined with intravenous dextrose can be implemented. Glucose monitoring is important.

There is no evidence that heparin is beneficial, and in fact, it can increase the risk of bleeding in animals with liver dysfunction.

Parathyroid Gland

Serum total calcium, total magnesium, and phosphorus concentrations in donkeys are within the reference range of values reported for horses. Nutritional secondary hyperparathyroidism may be seen in donkeys consuming diets with low calcium or high phosphorus content,[31] but also occurs with the ingestion of oxalate-rich plants.[32] Clinical signs (facial swelling, lameness, upper airway stridor and neurological signs), diagnosis, and treatment are similar to horses.

Thyroid gland

Donkeys have higher plasma free and total triiodothyronine (fT3, tT3), free and total thyroxine (fT4, tT4), and reverse T3 (rT3) concentrations than horses.[33-35] Young donkeys have higher fT4, tT4, and rT3 concentrations.[35] No gender differences for thyroid hormones have been documented in donkeys. Drugs such as phenylbutazone and dexamethasone reduce TH concentrations in horses;[36,37] but, information in donkeys is lacking.

References available upon request

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Pharmacology of Donkeys and Mules

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Donkey Pharmacology

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Summary

Most drugs used in equine practice work well and are safe for donkeys and mules, however, there are differences that could lead to suboptimal treatment, overdosing, toxicities, even death. Donkeys metabolize most non-steroidal anti-inflammatory drugs (NSAIDs) and antimicrobials faster than horses, but they also process other drugs slower or are more sensitive to their actions. For other drugs there is no information. Therefore, an overview on the pharmacology of drugs used in horses, donkeys and mules is clinically relevant.

Introduction

The donkey plays important economic, social, and cultural roles throughout the world, in particular in developing countries. In these countries, donkeys are valuable assets, central to the livelihood of families and local economies. In contrast, in developed countries, these animals are mainly used for recreational activities, ecotourism, hippotherapy/onotherapy, and as companion.[1] Donkeys are also used as a source of food (meat, milk), byproducts for medical conditions, and for the cosmetic industry. The high demand for donkey hide from China to produce "Ejiao" and other products has led to a decline in the worldwide donkey population.[2,3]

The number of donkeys and mules being admitted to veterinary hospitals or receiving specialized care has been increasing in recent years. Thus, there is a need to have better donkey and mule-specific veterinary knowledge. Practitioners should be familiar with anatomical, physiological, endocrine and pharmacological differences between donkeys and horses because extrapolating from horses can result in misdiagnosis, inadequate treatments, complications, unnecessary expenses, even death.[1,4-6]

Non-steroidal anti-inflammatory drugs (NSAIDs)

Most NSAIDs used in horses are metabolized faster by donkeys.[7-19] Therefore, for many of these drugs to reach therapeutic levels either the dose or dosing frequency will have to be increased.[7] Phenylbutazone clearance is faster and mean residence time (MRT) is shorter in donkeys compared to horses.[12,14] Increasing dosing could potentially result in higher risk of renal and gastrointestinal injury. Flunixin meglumine clearance is also higher and MRT lower in donkeys than in horses,[9,15] thus, the same principles of dosing and dosing frequency apply. One advantage is that flunixin

meglumine has less side effects than phenylbutazone.[15] Selective COX-2 inhibitors such as meloxicam, firocoxib and cimicoxib have faster clearances, lower MRTs, and shorter half-lives in donkeys compared to horses.[7,11,20,21] Therefore, more frequent dosing will be required to achieve adequate analgesia. Meloxicam is as effective as flunixin meglumine at reducing the systemic effects from endotoxemia in donkeys.[22] Unfortunately, meloxicam is not approved in the US and its use is extralabel. In contrast to most NSAIDs, carprofen has a slower clearance and larger AUC in donkeys.[13] This means, less frequent dosing is required.

Sedatives, analgesics and anesthetics

The response of donkeys and mules to pain can be subtle or go unnoticed due to their stoic nature, which could be detrimental (e.g., hyperlipemia, laminitis) because when signs are noted the disease process could be advanced. Analgesia in these instances can be valuable to ameliorate the pain-stress cycle. The dose of α2-adrenoreceptor agonists (e.g., xylazine) often needs to be increased by 50% the equine dose in donkeys and mules.[7] Analgesia from xylazine is similar between donkeys and horses,[23] but less effective than detomidine and romifidine.[24] Acepromazine provides good tranquilization in donkeys,[25] but they may require twice the acepromazine dose of horses. Butorphanol enhances the effects of α2-adrenoreceptor agonists in donkeys.[26] Tramadol is an effective analgesic in donkeys.[27] Fentanyl patches provide good pain relieve in donkeys.[28] Dipyrone has a shorter half-life in donkeys than horses.[8]

Ketamine has a faster clearance and shorter half-life in donkeys compared to mules and horses.[29] Therefore, higher or more frequent doses may be required for proper anesthesia. Guaifenesin (glyceryl guaiacolate) half-life is longer in donkeys than horses.[30] Propofol can be a safe option for anesthesia induction, but could be expensive.[30] Propofol combined with ketamine produces longer anesthesia and better than ketamine alone.[31] The combination xylazine, diazepam and ketamine is effective and safe in donkeys and mules.[31]

Inhaled anesthetics such as isoflurane and sevoflurane have similar properties in donkeys and horses.[7] Local anesthetics are used in similar manner and appear to be equally effective in donkeys, mules, horses.

Antimicrobials

Many antimicrobials used in equine practice, similar to most NSAIDs, have faster clearances, lower MRTs and shorter half-lives in donkeys compared to horses.[7,19] This indicates that for many of these drugs, donkeys and mules may require higher doses or shorter dosing intervals.[7,19] Aminoglycosides (amikacin and gentamicin) have similar pharmacology between horses and donkeys.[10] Donkeys eliminate fluoroquinolones (enrofloxacin and marbofloxacin) slower and once a day dosing interval is preferred.[17] Donkeys eliminate oxytetracycline faster than horses and ponies and twice daily dosing is recommended.[32] Doxycycline also reaches lower serum and tissue levels in donkeys compared to horses, which has been suggested to be result of increased hepatic

clearance.[33] Sulfonamides and trimethoprim are eliminated faster in donkeys compared to horses and mules,[34] but current dosing at 2-3 times daily is recommended. For many other antimicrobial drugs used in equine practice information in donkeys and mules is scarce. As important as antimicrobial use is the implementation of antimicrobial stewardship practices. Similar to other equids, donkeys are susceptible to the toxic effects of ionophores.

Anti-parasitic drugs

Macrocyclic lactones (ivermectin, moxidectin) can be neurotoxic to young donkeys or those in poor body condition.[7] Imidocarb dipropionate, which is the preferred treatment for piroplasmosis can cause hepatic side effects and neurologic signs in donkeys and mules.[35] Doses should be reduced for these animals. Premedication with anticholinergic drugs such as glycopyrrolate (0.0025 mg/kg/IV) or N-butylscopolammonium bromide (Buscopan®; 01-0.2 mg/kg/IV) 5-10 minutes prior to imidocarb administration is recommended.[7] Principles for anthelmintic in horses apply to donkeys. Fenbendazole is not very effective at eliminating lungworms in donkeys and horses. [40] Moxidectin (400 µg/kg, PO) is the drug of choice for lungworms in donkeys.[7]

Other drugs

Drugs such as lidocaine and metoclopramide are occasionally used in donkeys under protocols similar to horses, but pharmacological and efficacy information are lacking.

References available upon request

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Endocrinology of Geriatric and Obese Horses

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Endocrinology of Geriatric and Obese Horses

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Summary

Horses are living longer, which requires veterinarians to have a better understanding on equine geriatric medicine. This increase in aging requires better knowledge on equine health, preventative medicine, nutrition, but also endocrine disorders of aged animals. The best example of this is pituitary pars intermedia dysfunction (PPID), a condition almost exclusive of geriatric horses. Similarly, changes in feeding practices associated with reduced physical activity have made horses, in particular in developed countries, more prone to obesity which carries other disturbances such as insulin dysregulation (equine metabolic syndrome), but also complications, including endocrinopathic laminitis. Client education is as important as weight loss and physical activity to prevent and control EMS. Other endocrine disorders (e.g. hypothyroidism) are rare, but often misdiagnosed. In this presentation we will provide an overview on key pathophysiology, diagnosis and treatment of these conditions.

Introduction

Endocrine systems are essential for organ development and tissue differentiation. They are central for activity of all body systems. Therefore, a balance on their activity and interactions is required for proper organ function. Endocrine disorders are common with different diseases and contribute to dysfunction, in many instances associated with disease severity and death. Disorders related to energy metabolism are consequence of excessive carbohydrate intake (obesity, hyperinsulinemia), reduced physical activity, but can also be triggered by other endocrinopathies. In some instances, endocrinopathies are result of aging and environmental factors. Examples of these conditions include insulin dysregulation (ID) and pituitary pars intermedia dysfunction (PPID). The goal of this presentation is to revise current concepts on endocrinopathies of obese and geriatric horses.

Equine Metabolic Syndrome

Equine metabolic syndrome (EMS) is a syndrome characterized by obesity (regional adiposity), insulin dysregulation (ID) and laminitis. Risk factors alter energy homeostasis, promote obesity, and increase insulin concentrations, which in turn increases the risk of laminitis (endocrinopathic laminitis; hyperinsulinemia-associated laminitis [HAL]).[1-4] These horses also have increased concentrations of endocrine factors produced by adipocytes (adipokines), elevated intestinal hormones (incretins) that promote insulin secretion, and increased triglycerides. Hypertension has been reported.

Insulin dysregulation is a collective term that considers different factors that alter normal insulin dynamics. This include abnormally high baseline insulin concentrations, post-prandial

hyperinsulinemia (exacerbated insulin response to oral carbohydrates), reduced insulin clearance (liver clears insulin) and insulin resistance (IR).

Risk factors include excessive caloric intake, obesity, genetics, lack of physical activity, and other diseases. Environmental factors likely play a role but are poorly defined. Diets rich in non-structural carbohydrates (NSC) are the major risk factor for ID and EMS. Therefore, for most horses, management is at the center of ID. The condition is more common in Arabians, Saddlebreds, Morgan, Andalusians, Paso Finos, Tennessee Walking Horses, Warmbloods, ponies, and donkeys. These breeds are considered "easy keepers" which is a term suggesting that these animals are more efficient at handling energy, which translates in fat accumulation and hyperinsulinemia. Most animals are between 5 to 12 years of age, with no gender difference. EMS is rarely diagnosed in Thoroughbreds and Standardbreds. In addition, to obesity and ID, genetic and environmental factors contribute to the development of laminitis.

Adipose tissue releases adipokines and inflammatory cytokines. Thus, EMS horses are in a pro-inflammatory state. Adipokines likely interfere with insulin signaling, reducing its actions, which is translated as IR. Therefore, to achieve its effects insulin concentrations must increase (hyperinsulinemia).

Not all horses with ID are obese, supporting that other factors are equally important. For example, the liver is the main site for insulin clearance and reduced hepatic activity to remove insulin could result in hyperinsulinemia. Liver dysfunction is evident in many of these horses that have mild elevations in liver enzymes and hypertriglyceridemia.

There are other endocrine factors yet to be elucidated that contribute to hyperinsulinemia. For example, many horses with PPID also have ID. It has been proposed that local production of cortisol which is known to cause IR is at play. Cytokines (IL1-β, IL-6) can interfere with insulin signaling.

Incretins are hormones produced by intestinal cells to enhance insulin secretion after a meal. They are more important than glucose in stimulating insulin release. Therefore, if incretin levels are elevated it will lead to higher insulin levels in circulation, in particular after a meal (post-prandial hyperinsulinemia). There is evidence that this occurs in some horses with EMS.[5]

Diagnosis: The initial diagnosis is based on clinical signs (obesity, regional adiposity, laminitis). A number of diagnostic methods are available, from resting insulin concentrations to dynamic tests.

Resting insulin: Do not feed the horse grain overnight. Offer one flake of hay. Collect blood sample in serum or EDTA tube. Use assay-specific reference values. For the Immulite 1000, which is the most commonly used system, insulin <20 μIU/ml suggest the horse is insulin sensitive (normal). Concentrations of 20-50 μIU/ml are equivocal, and >50 μIU/ml indicates ID. Values are slightly higher for the Immulite 2000.[1]

Dynamic tests: the oral sugar test (OST) is the most commonly used and practical test. It should

be used in animals with equivocal insulin values or to confirm the diagnosis of ID. The horse is fasted overnight (at least 4 hours). Karo corn syrup is administered via dosing syringe – there are two versions of this test (0.15 ml/kg or 0.45 ml/kg). There are other tests such as the in-feed oral glucose tolerance test (OGTT), combined glucose-insulin test (CGIT), and twos-step insulin-response test.

Low dose OST: Collect baseline blood sample and administer syrup (0.15 ml/kg) – Collect sample at 60 minutes. If insulin >45 µIU/ml is considered positive for ID.[1] Collection at 90 minutes has also been proposed. Baseline blood sample is optional.

High dose OST: Collect baseline sample and administer syrup (0.45 ml/kg) – Collect sample at 60 minutes. If insulin >65 µIU/ml is considered positive for ID.[1] Collection at 90 minutes has also been proposed.

Baseline samples can be used to measure resting glucose and insulin concentrations and assess the response to treatment. Hyperglycemia at baseline further supports ID.

Leptin can be useful and currently measured in some labs as part of the EMS panel. High leptin has been associated with EMS and laminitis. Triglyceride concentrations are often elevated in horses with ID. Many horses have increase GGT activity consistent with liver involvement.

Management: Caloric restriction and physical activity are at the center of the management of these horses.[1-3] Do not feed grain. Reduce or eliminate grazing depending on grass quality. Feed hay low in NSC (<10%) and soak hay for >1 hour. A number of rations based on low NSCs (low starch diets) and high fat content are available. The rationale is that sugars, not fat) are the main trigger for insulin release, while fat provide an alternative source of energy. Promote physical activity in animals with mild laminitis. Monitor weight and body condition score (BCS). The goal is a BCS of 5-6. Repeated testing is indicated. Medical therapy should be considered in some of these animals to either increase metabolic activity (levothyroxine), insulin sensitivity (metformin) or glucose urine waste (gliflozins).[1-3] Some horses in addition to EMS could have PPID and will benefit from adding pergolide to the treatment. Other medications will depend on the clinical presentation (e.g. non-steroidal anti-inflammatory drugs [NSAIDs]; gabapentin, etc)

Prevention: Client education is important in preventing and controlling EMS. The major challenge is client compliance.

For additional details on the diagnosis and management of EMS, the reader is advised to visit the endocrinology group website: https://sites.tufts.edu/equineendogroup/

Pituitary Pars Intermedia Dysfunction

Pituitary pars intermedia dysfunction (PPID) is a slowly progressive condition of geriatric horses.[4,6,7] It is characterized by delayed seasonal coat shedding, hypertrichosis, weight loss (loss of topline muscles), and laminitis.[4,6,7] Other signs include lethargy, secondary infections, abnormal sweating, tendon laxity, infertility, and regional adiposity.[4,6,7] It results from damage of hypothalamic dopaminergic neurons that physiologically inhibit cells of the pars intermedia

(melanotropes), which results in excessive melanotrope proliferation.[8] These cells produce excessive amounts of pro-opiomelanocortin peptides, including adrenocorticotropic hormone (ACTH) and melanocyte-stimulating hormone (α-MSH). PPID is common in equids (horse, ponies, donkeys) over 15 years of age.[7] Hypertrichosis is considered pathognomonic of PPID. PPID could contribute to disorders in other body systems. Immune suppression is responsible for secondary infections and parasitism.

Diagnosis: The diagnosis is based on clinical signs that can be highly suggestive. Baseline ACTH concentration is the most practical method to confirm the diagnosis PPID. Plasma ACTH concentrations increase in fall in healthy and PPID horses. Therefore, results should be adjusted to season. Values also differ between horses, ponies and donkeys. In animals with equivocal results ACTH can be measured at a different season or use a dynamic test such as the TRH-stimulation test. Some animals also have ID and measurement of baseline insulin concentrations can provide a better assessment of the endocrine status of the animal. High insulin as described for EMS could contribute to regional adiposity and laminitis.

Most healthy horses have resting ACTH values <20 pg/ml from November to August.[4,6] Values between 20-45 pg/ml can be equivocal. Resting ACTH values >100 pg/ml from August to November are suggestive of PPID. Values >45 pg/ml can be diagnostic in the fall.[4,6] For confirmation, a dynamic test (TRH-stimulation test) is recommended.

TRH-stimulation test: Collect baseline blood sample in EDTA tube. Administer 1 mg of TRH intravenously. For small equids and donkeys administer 0.5 mg. Collect second sample at 10 minutes. Potential side effects include yawning, coughing and flehmen response. Interpretation also depends on the season. From January to June, ACTH <100 pg/ml is considered negative, 100-200 pg/ml is equivocal, and >200 pg/ml is positive. From July to December, ACTH <100 pg/ml PPID is unlikely, but values >100 pg/ml are hard to interpret due to major variations between horses. In other words, in this time of the year the TRH-stimulation test is good rule out PPID.[4,6]

Treatment: Therapy is oriented at reducing ACTH secretion and controlling complications from this condition. Pergolide (Prascend®; Boehringer-Ingelheim) at 1 mg (1 tablet per horse) is recommended. Lower doses can be used in donkeys and ponies. Some horses require higher doses (up to 2 mg/animal). Some animals may become inappetant and dosing should be adjusted. In some cases, it may to be transiently discontinued. Analgesics may be necessary to control foot pain. In animals with ID, metformin could be beneficial.

For additional details on the diagnosis and management of PPID, the reader is advised to visit the endocrinology group website: https://sites.tufts.edu/equineendogroup/

Endocrinopathic Laminitis

Laminitis is a consistent finding in horses with EMS and PPID.[1,3,4] In the case of EMS, hyperinsulinemia-associated laminitis (HAL) develops slowly to become a chronic condition with intermittent periods of mild to severe pain. This has been attributed to insulin altering the integrity of the dermal and epidermal lamellar cells, with subsequent detachment.[1,3] The role of leptin and

inflammatory cytokines in the development of HAL remains to be determined. However, high leptin has been associated with laminitis.[9] There are many aspects of this condition that remain unknown. In PPID, hyperinsulinemia could play a role, however, other factors such as increased ACTH and cortisol concentrations are at play. Either form of endocrinopathic laminitis differ from the sepsis-associated and supporting-limb laminitis regarding their pathophysiology, progression and prognosis.

Diagnosis: Diagnosing endocrinopathic laminitis is simple, evident by hoof changes and lameness. Radiographs are valuable to confirm the diagnosis, severity, prognosis and interventions (corrective trimming and shoeing).

Management: Depends on severity. Ranges from trimming to corrective shoeing, as well as use of different medications. Foot abscesses are common. Medical treatment is oriented at controlling pain, occasionally infections. Pain is usually managed with NSAIDs, but other type of drugs (gabapentin, opioids) are also used.

Prevention: Measures should be aimed at preventing or controlling signs of EMS, including caloric restriction, weight loss, and increasing insulin sensitivity. In animals with evident laminitis, the goal is to prevent progression.

Other Endocrine Systems

Disorders of the thyroid gland are uncommon in old horses. Some may have thyroid gland enlargement that in most instances is an esthetic issue. Functional thyroid adenomas are rare in horses. Hypothyroidism is often misdiagnosed in horses. Horses with various diseases can have low thyroid hormone concentrations, but this does not mean hypothyroidism but a biological response to disease (euthyroid sick syndrome). Malignancies such as squamous cell carcinoma and lymphoma can produce parathyroid hormone (PTH)-related protein (PTHrP), which resembles the actions of PTH. These horses develop hypercalcemia and excessive bone loss. This should be suspected in animals with hypercalcemia with normal values of renal function

References available upon request

EQUINE PROCEEDINGS

June 5–7, 2022

Equine Disorders of Calcium, Phosphorus, and Magnesium Regulation

Dr. Ramiro Toribio, The Ohio State University

Disorders of Calcium, Phosphorus and Magnesium in Horses

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Summary

Calcium (Ca), phosphorus (PO_4) and magnesium (Mg) have structural (mechanical) and non-structural (ionic) functions, and their concentrations in the extracellular and intracellular compartments are influenced by physiological and pathological conditions, acute and chronic. Disorders of these minerals are frequent in horses and foals with various diseases, but often go unnoticed. Abnormal blood concentrations have been associated with disease severity and outcome. These minerals interact with other ions (Mg^{2+}, Na^{+}, K^{+}) and analytes (glucose, lipids), and imbalances should be evaluated in the context of laboratory and clinical findings. Clinicians should be aware of these disorders in other to establish a proper diagnosis, treatment and prognosis.

Calcium

Calcium is essential for physiological processes such as muscle contraction, neuromuscular excitability, blood coagulation, enzyme activation, hormone secretion, cell division, and cell membrane stability.[1] It participates in cell injury and cell death. Maintenance of steady blood calcium concentrations is essential. Calcium is found in three main compartments: *the skeleton, soft tissues, and the extracellular fluid*. The skeleton contains 99% of the total body calcium. The remaining 1% is present within the cell (0.9%) and in the extracellular fluid (0.1%).

In blood, total calcium exists as a free (or ionized) form (55%), bound to proteins (40%), and complexed to anions such as citrate, bicarbonate, phosphate, and lactate (5-10%).[1] Free or ionized calcium (Ca^{2+}) is the biologically active form of calcium. Most protein-bound calcium is associated with albumin. During acidosis, Ca^{2+} binding to albumin is reduced, increasing Ca^{2+} concentrations; during alkalosis, Ca^{2+} concentrations are lower. This is relevant to conditions such as proximal enteritis and excessive sweating that result in alkalosis and signs of hypocalcemia. Horses have high blood calcium concentrations, high intestinal absorption of calcium, high urinary fractional excretion of calcium, and low serum vitamin D concentrations.

Phosphorus

Phosphorus is essential for energy homeostasis, intermediary metabolism of carbohydrates, fats, and proteins, oxidative phosphorylation, enzyme activity, electrolyte transport, oxygen transport, cell membrane stability, neuromuscular excitability, muscle contraction, nucleic acid metabolism, gene transcription, and cell proliferation. Phosphorus (phosphate; PO_4) represents ~1% of the body weight, with most (85%) located in the bone matrix (hydroxyapatite), 15% in blood and soft

tissues, and less than 0.1% in the extracellular fluid.[1] Serum PO_4 concentrations are higher in foals than horses result of increased intestinal absorption and renal reabsorption of PO_4 to supply the growing skeleton and other tissues. Alkaline phosphatase activity is also higher in foals from increased osteoblastic activity and bone formation.

Magnesium

Magnesium (Mg) is an essential macroelement involved in physiological processes such as enzymatic activation, intermediary metabolism of carbohydrates, fats, and proteins, nucleic acid metabolism, regulation of membrane function, nerve function, muscle contraction, and cell proliferation.[2,3] Similar to total calcium and ionized calcium (Ca^{2+}), total magnesium (tMg) in biological fluids exists bound to proteins, chelated to organic anions, and ionized/active/free Mg (Mg^{2+}). The body of domestic animals contains 0.05% Mg by weight of which 60% is in the skeleton (0.5-1% bone ash), 38% in soft tissues, and 1-2% in the extracellular fluid.[4]

Regulation

Extracellular calcium concentrations are regulated by three hormones: parathyroid hormone (PTH), calcitonin (CT), and 1,25-dihydroxyvitamin D [$1,25(OH)_2D$, or calcitriol).[5-7] Parathyroid hormone-related protein (PTHrP) shares homology with PTH, activates the PTH receptor, and is important for calcium homeostasis in the fetus.[8,9] PTH increases during hypocalcemia while CT increases during hypercalcemia. These hormones also regulate PO_4 concentrations. Endocrine regulation of Mg is minimal.

Parathyroid hormone: PTH is secreted by the chief cells of the parathyroid gland in response to hypocalcemia. In the kidney PTH increases Ca^{2+} and Mg^{2+} reabsorption, reduces PO_4 reabsorption, and promotes $1,25(OH)_2D$ synthesis. In bone, PTH promotes osteoclast-mediated resorption to release calcium and phosphorus into circulation. Excessive PTH secretion leads to bone loss.

Vitamin D: Vitamin D has endogenous (vitamin D_2) and exogenous (vitamin D_3) sources. It promotes intestinal absorption and renal reabsorption of calcium and phosphorus. It also suppresses PTH secretion. Vitamin D also modulates immune function as well as epithelial and endothelial integrity. Reduced vitamin D concentrations have been associated with a multitude of human pathologies,[10-15] and more recently were linked to sepsis and mortality in critically ill newborn foals.[16]

Calcitonin: Calcitonin (CT) is secreted by the parafollicular cells (C cells) of the thyroid gland in response to hypercalcemia. CT decreases plasma Ca^{2+} and PO_4 concentrations by suppressing osteoclastic bone resorption and increasing urinary Ca^{2+} and PO_4 excretion. Disorders of CT dysregulation are uncommon in horses.

Parathyroid hormone-related protein (PTHrP): Parathyroid hormone-related protein (PTHrP) is essential in embryogenesis and has important physiological functions in skeletal development. High PTHrP is measured with some malignancies in people, small animals and horses (humoral hypercalcemia of malignancy; HHM). This is a paraneoplastic syndrome reported in horses with lymphoma, gastric and preputial squamous cell carcinoma, ameloblastoma, and multiple myeloma.[17-21] Because PTHrP binds the PTH receptor, high PTHrP causes hypercalcemia.

CALCIUM DISORDERS

Hypocalcemia

Different pathologies are associated with hypocalcemia in horses **(Table 1).** Clinical signs of acute hypocalcemia result from increased neuromuscular excitability and decreased smooth muscle cell contractility**.** Clinical signs include anxiety, depression, SDF, hyperexcitability, ataxia, stiff gait, tetany, muscle fasciculations and tremors, tachypnea with flared nostrils, upper airway stridor, dyspnea, dysphagia, hypersalivation, hyperhidrosis, ileus, colic, seizures, hypotension, recumbency/ collapse, and death.

Hypocalcemic disorders

Hypoparathyroidism: This condition is characterized by hypocalcemia, hyperphosphatemia, and low serum PTH concentrations. Hypomagnesemia may be present. It can be primary or secondary hypoparathyroidism, and both occur in horses.

Primary hypoparathyroidism: Primary hypoparathyroidism results from decreased synthesis and secretion of PTH. The problem is in the parathyroid gland. Horses present with signs of hypocalcemia, as described above. The diagnosis is based on serum concentrations of Ca^{2+}, Mg^{2+}, phosphorus, and PTH. Hypocalcemia, hyperphosphatemia, hypomagnesemia, and low serum PTH concentrations are the typical features.[22,23] Some horses with primary hypoparathyroidism may benefit from $MgSO_4$ or $MgCl_2$ administration.

Secondary hypoparathyroidism*:* In secondary hypoparathyroidism reduced PTH secretion is consequence of another abnormality, such as low magnesium or sepsis (cytokines).

Sepsis and systemic inflammation: Hypocalcemia is frequent in critically ill horses and foals,[16,24] in particular those with gastrointestinal disease.[7,25-28] Hypocalcemia has been associated with mortality in horses[28] and foals.[16,24]

Hypocalcemia in foals: Sepsis, hypomagnesemia, acute renal injury, pancreatitis, and acute muscle injury have been linked to hypocalcemia in foals. Idiopathic hypocalcemia represents multiple disorders (increased cytokines, hypomagnesemia, abnormal parathyroid function, congenital disorder) that interfere with calcium homeostasis and PTH secretion. Recently, a mutation resulting in idiopathic hypocalcemia (e*quine familial isolated hypocalcemia*) was identified in Thoroughbred foals.[29]

Exercise-induced hypocalcemia: Electrolyte and acid-base abnormalities are common in exercising horses. These horses develop hypocalcemia from calcium loss in sweat and alkalosis.[30]

Cantharidiasis: Cantharidiasis (blister beetle toxicosis) occurs from ingestion of alfalfa contaminated with blister beetles (*Epicauta* spp.), which produce cantharidin (cantharidic acid), can cause severe hypocalcemia and hypomagnesemia in horses.[31-33] These horses have hypocalcemia and hypomagnesemia. Clinical signs include SDF, muscle fasciculations, ataxia, dyspnea, laryngeal spasm, cardiac dysrhythmias, colic, diarrhea, endotoxemia, dehydration, hypotension, hematuria, stranguria, pollakiuria, and sudden death. [1,154,183,186]

Acute renal failure: Hypocalcemia and hypomagnesemia are common findings in horses with

acute renal injury. Damage to tubular epithelial cells leads to reduced absorptive capacity for Ca^{2+} and Mg^{2+}.

Exertional rhabdomyolysis: Often tying up horses develop hypocalcemia due to calcium sequestration in the muscle fibers.

Pancreatitis: This is rare condition in horses where animals develop severe abdominal pain but also hypocalcemia.

Oxalate toxicity: Ingestion of oxalate-containing plants can cause signs of hypocalcemia. Chronic oxalate toxicity from prolonged consumption of these plants result in orthopedic problems (lameness, fractures) from secondary hyperparathyroidism and reduced bone mass.

Treatment of hypocalcemia: Treatment is based on enteral and parenteral administration of calcium salts. Calcium gluconate 23% is readily available and safe for intravenous administration in horses. For most horses, 50 ml of calcium gluconate per 5-liter bag of fluids suffices. Some horses require higher doses or direct calcium gluconate administration. Calcium chloride may be another option to treat hypocalcemia; however, is not available in large volumes, it is more expensive, and it may cause irritation at the administration site. Ideally, blood calcium concentrations should be monitored. Some horses with hypocalcemia also require intravenous magnesium (magnesium sulfate) to restore normocalcemia. Oral calcium salts can be used in horses with chronic hypocalcemia. Dicalcium phosphate and calcium carbonate (limestone) can be used safely.

Hypercalcemic disorders

Hypercalcemia from parathyroid gland dysfunction (parathyroid-dependent) include primary hyperparathyroidism, secondary hyperparathyroidism.

Hypercalcemia independent of the parathyroid gland dysfunction include vitamin D toxicity, humoral hypercalcemia of malignancy, and chronic renal failure (CRF).

Primary hyperparathyroidism (PHPT): PHPT develops from parathyroid adenomas that secrete excessive amounts of PTH and do not respond to negative feedback of Ca^{2+}.[34-36] There is hypercalcemia, hypophosphatemia, and increased PTH concentrations. Osteodystrophia fibrosa from excessive bone loss may ensue. Radiographic findings include reduced bone density, in particular in flat bones.[37] Diagnosis is based on laboratory tests, measuring PTH concentrations, ultrasonography, radiography and scintigraphy.

Secondary hyperparathyroidism: There is excessive PTH secretion consequence of hypocalcemia and/or hyperphosphatemia, and/or hypovitaminosis D, from CRF or from nutritional imbalances. Renal secondary hyperparathyroidism is poorly documented in horses.

Nutritional secondary hyperparathyroidism (NSHPT): Reduced intestinal calcium absorption results in hypocalcemia, which triggers the production of PTH to restore normocalcemia. PTH promotes renal reabsorption of calcium and bone resorption. Diets low in calcium, high in phosphorus or high oxalates that reduce calcium absorption lead to hypocalcemia, hyperphosphatemia, and NSHPT.[194] This condition is also known as ‘bran disease’, ‘miller’s disease’, ‘big head’, osteodystrophia fibrosa, osteitis fibrosa, and equine osteoporosis. Balanced

equine diets should have a calcium:phosphorus ratio of >1.5:1 to meet daily requirements. Diets with a phosphorus:calcium ratio > 3:1 (low calcium:phosphorus ratio) predispose horses to NSHPT.[38,39] NSHPT is uncommon in developed countries, but is occasionally seen, in particular in young horses. PTH will increase osteoclastic activity, bone resorption, and bone loss.[40] Facial bone loss and excessive accumulation of unmineralized bone matrix (osteodystrophia fibrosa) results in facial enlargement (big head). Clinical signs include enlargement of facial bones, upper respiratory noise, shifting lameness, and a stiff gait.[41,42] Young animals may develop physeal enlargement and limb deformities.[38] Vertebral abnormalities and compressive neurological signs may develop. Laboratory findings may include mild hypocalcemia and hyperphosphatemia; however, these values are often in the normal range.[43,44] Serum PTH concentrations may be increased.[41,45] Urinary excretion of calcium is low (normal 3-10%) and of phosphorus (normal < 0.5%) is high.[45] On radiographs there could be decreased bone density.[41,44] Dietary supplementation with calcium carbonate (limestone; $CaCO_3$) or dicalcium phosphate (dibasic calcium phosphate; $CaHPO_4$) is recommended.[39] Severely affected horses should be confined. Use of NSAIDs may be indicated in horses with severe pain.

Hypervitaminosis D: Ingestion or administration of ergocalciferol (vitamin D_2) or cholecalciferol (vitamin D_3) causes disturbances of calcium and phosphorus homeostasis. Ingestion of plants with vitamin-like compounds results in clinical signs of vitamin D toxicity.[46-50] *Solanum glaucophyllum (S. malacoxylon)* causes a condition known as "enteque seco" in Argentina and "espichamento" in Brazil.[49-51] Jessamine (*Cestrum diurnum*), a shrub distributed in the southern United States may cause hypervitaminosis D.[46,50] Hyperphosphatemia is the most consistent finding in horses with vitamin D intoxication.[52] Serum calcium concentrations may be in the normal range or increased.[48,52-54] PTH is often low.[55] Clinical signs include anorexia, weight loss, polyuria, polydipsia, lameness, and reluctance to move.[52,54,56] Renal mineral may lead to renal failure.[53] The prognosis is poor. Treatment may include reducing dietary calcium intake. Postmortem examination reveals mineralization of soft tissues.

Hypercalcemia of Malignancy (HHM): This is a paraneoplastic condition in which tumors secrete PTHrP leading to hypercalcemia. In horses, HHM has been described with squamous cell carcinomas, lymphosarcoma, multiple myeloma, and ameloblastoma. These animals have hypercalcemia, hypophosphatemia, low to normal PTH concentrations, reduced urinary excretion of calcium, increased urinary excretion of PO_4, and over time, reduced bone density. Most clinical signs are the result of the primary problem rather than hypercalcemia.

Chronic renal failure: This is the most common cause of hypercalcemia in horses. Healthy horses excrete large amounts of calcium through the kidneys and CRF results in calcium retention and hypercalcemia. In addition to hypercalcemia there is hypophosphatemia and azotemia. Most of these animals have a history of weight loss. Prognosis is guarded to poor.

Treatment of hypercalcemia: Hypercalcemia is rarely an equine emergency. Mild to moderate hypercalcemia in general is not life-threatening, and treatment should be directed at the primary cause. Hypervitaminosis D has a poor prognosis regardless of treatment. Surgical removal of epithelial tumors could be a successful treatment in some patients with carcinomas or

hyperparathyroidism. Furosemide can increase urinary excretion of calcium but is a short-term solution. Glucocorticoid administration should be considered.

PHOSPHORUS DISORDERS

Disorders of phosphorus homeostasis can be acute or chronic, resulting in hypophosphatemia or hyperphosphatemia. In critically ill horses hypophosphatemia is more frequent while in foals hyperphosphatemia is more common.[16] Genetic conditions leading to hypo or hyperphosphatemia remain to be documented in horses.

Hypophosphatemia

Acute hypophosphatemia usually reflects PO_4 redistribution between intra and extracellular compartments. However, in horses that have been sick for many days PO_4 depletion should be suspected. Hypophosphatemia occurs more often in miniature horses, ponies and donkeys than in horses, usually associated with anorexia, hyperlipemia and parenteral nutrition.

Clinical signs of hypophosphatemia: The clinical signs of acute hypophosphatemia are consequence of skeletal and smooth muscle as well as neurological dysfunction, while signs of chronic hypophosphatemia are mainly linked to the skeleton. Signs of hypophosphatemia often go unnoticed, and when present, serum PO_4 concentrations is usually very low. These include muscle weakness, fasciculations, dysrhythmias, neuromuscular excitability, ileus, and cell membrane fragility and lysis (hemolysis, rhabdomyolysis). Chronic hypophosphatemia is rare in horses and manifests as weakness, weight loss, depraved appetite (pica), reduced bone mass, orthopedic diseases, lameness, rickets, and hemolysis.[57] Rickets from PO_4 deficiency or hypovitaminosis D remains to be documented in equids.

Laboratory findings: hypophosphatemia, hyperglycemia, hyperinsulinemia, hypokalemia, and hypomagnesemia. Hemolysis has been reported in small animals, ruminants, and humans.[248,253,255-257] Muscle enzyme activity may be increased.

Treatment of hypophosphatemia: Critically ill equids are rarely treated for hypophosphatemia. Hypokalemia and hypomagnesemia are frequent in horses and foals with hypophosphatemia. Injectable and oral products formulated for other species can be used in horses. Potassium phosphate or sodium phosphate are available. Animals can be supplemented via nasogastric intubation with potassium or sodium phosphate salts. Sodium phosphate enemas may be useful. During rapid PO_4 replacement therapy it is important to monitor serum calcium concentrations as high PO_4 doses could cause hypocalcemia.

Hyperphosphatemia

Hyperphosphatemia results from increased PO_4 absorption, renal injury, hypoparathyroidism, vitamin D toxicity, metabolic acidosis, cell lysis (hemolysis, rhabdomyolysis, tumor necrosis), or iatrogenic causes.[243,263] In newborn foals, overuse of phosphate-based enemas can cause hyperphosphatemia. Hyperphosphatemia is common in critically ill foals.[16,24] There are reports of humans developing hyperphosphatemia from bisphosphonate therapy and bloodwork should be

monitored for in horses undergoing similar treatment.[58]

Clinical signs of hyperphosphatemia: The signs of acute hyperphosphatemia are those of acute hypocalcemia and include tetany, hyperexcitability, muscle fasciculation, colic, and dysrhythmias. Signs of chronic hyperphosphatemia are those of calcium deficiency including lameness, orthopedic pathologies, fractures, and osteodystrophia fibrosa (nutritional secondary hyperparathyroidism).[252] Developmental orthopedic diseases can be a problem in growing animals. Soft tissue mineralization (calcinosis) can be present, although it is rare.

Treatment of hyperphosphatemia: Based on primary pathology and duration, therapy to reduce serum PO_4 concentrations may not be necessary. For example, if acute hyperphosphatemia is result of acute cell lysis (e.g. rhabdomyolysis) or iatrogenic (e.g. phosphate enemas), fluid therapy and diuretics are the treatment of choice.

MAGNESIUM DISORDERS

Hypomagnesemia

Hypomagnesemia is a common finding in critically ill horses and foals,[7,59-63] although, rarely diagnosed because Mg values are not reported in most chemistry profiles. Causes of hypomagnesemia include sepsis, gastrointestinal disease, acute renal injury, tissue sequestration, shift to the intracellular compartment, and endocrine abnormalities.[4] Mg plays essential roles in inflammation, protection against free radical injury, and neurotoxicity.[64,65] Horses and foals with gastrointestinal disease often have endotoxemia and hypomagnesemia.[7,24,66-68] Equine cantharidiasis (blister beetle toxicosis) is a common cause of hypomagnesemia and hypocalcemia in horses. Clinical signs of hypomagnesemia include synchronous diaphragmatic flutter (SDF; thumps), muscle fasciculation, ataxia, dyspnea, laryngeal spasm, cardiac dysrhythmias, colic, diarrhea, endotoxemia, dehydration, hypotension, hematuria, stranguria, pollakiuria, and sudden death.[31,32,69] Many signs from hypomagnesemia are related to hypocalcemia because calcium homeostasis depends on Mg. Hypomagnesemia exacerbates the signs of hypocalcemia. Hypomagnesemia could contribute to the pro-inflammatory state observed in critically ill equine patients.

Treatment of hypomagnesemia: Treatment is based on intravenous administration of $MgSO_4$. Some animals with hypocalcemia could benefit from magnesium therapy. Magnesium therapy is also promoted for foals with neurological disorders (maladjustment syndrome seizures, cerebral and spinal trauma).[4,70] Oral administration of Mg (sulfate, oxide, citrate) should be considered for horses with prolonged hypomagnesemia or refractory hypocalcemia.

Hypermagnesemia

Hypermagnesemia is rare in horses and foals, may occur from cell lysis (e.g. rhabdomyolysis, hemolysis), but often is iatrogenic (e.g. Epson salt administration). High Mg concentrations may be seen in horses with chronic renal failure. Clinical signs of hypermagnesemia are not specific and, at least in horses, it may reduce neuromuscular activity, has calming effects, reduces physical activity, and decreases blood pressure through its Ca^{2+} and Na^{+} channel and receptor blocking properties.[71,72] Treatment depends on the clinical condition. Some animals may need diuresis.

Key points:

- Hypocalcemia and hypomagnesemia are frequent in critically ill horses and foals, in particular with sepsis and gastrointestinal disease.
- Animals with acute hypocalcemia should be treated with intravenous calcium, in particular those showing signs (e.g. SDF, fasciculations, ileus).
- Hypercalcemia is diagnosed in horses with hyperparathyroidism, CRF and malignancies. CRF is the most common cause of hypercalcemia in horses.
- To narrow cause of hypercalcemia, start by running a chemistry profile. If there is azotemia and hypercalcemia, likely is CRF. If there is no azotemia, consider measuring PTH. If PTH is normal, likely that the cause of hypercalcemia is a malignancy. Measure PTHrP. Vitamin D toxicity is infrequent, often iatrogenic.
- Hypophosphatemia is common in animals with energy dysregulation, in particular ponies and donkeys with anorexia, hyperlipemia or parenteral nutrition. This is often overlooked.
- Hyperphosphatemia is infrequent in horses, but common in septic foals. Also occurs in condition associated with cell lysis (hemolysis, rhabdomyolysis), which are usually transient. It can be iatrogenic in foals (enemas). Hyperphosphatemia is a consistent finding with vitamin D toxicity.
- Magnesium therapy should be considered in animals with hypomagnesemia, but also in some with hypocalcemia as calcium homeostasis depends on Mg.
- Hypermagnesemia is rare in equine patients, usually with cell lysis or iatrogenic. It can develop from administration of $MgSO_4$ (Epson salt). Horses with CRF can have hypermagnesemia.

Table 1. Conditions in which hypocalcemia has been reported in horses and foals

Colic	Oxalate ingestion
Colitis	Hypomagnesemia
Sepsis/septicemia	Cantharidin toxicosis
Endotoxemia	Pancreatitis
Endurance exercise	Retained placenta
Late pregnancy	Furosemide administration
Lactation (lactation tetany)	Excessive administration of $NaHCO_3$
Transport (transit tetany)	Primary hypoparathyroidism
Acute renal failure	Liver disease
Chronic renal failure	Dystocia
Rhabdomyolysis	Malignant hyperthermia
Pleuropneumonia	Magnesium toxicosis
Heat stroke	Postoperative myopathy

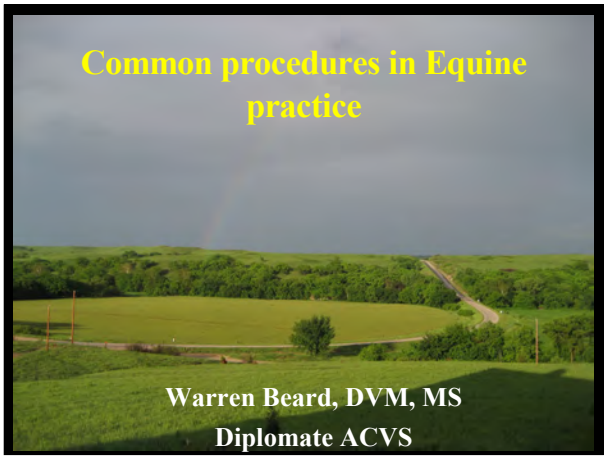
References available upon request

EQUINE PROCEEDINGS

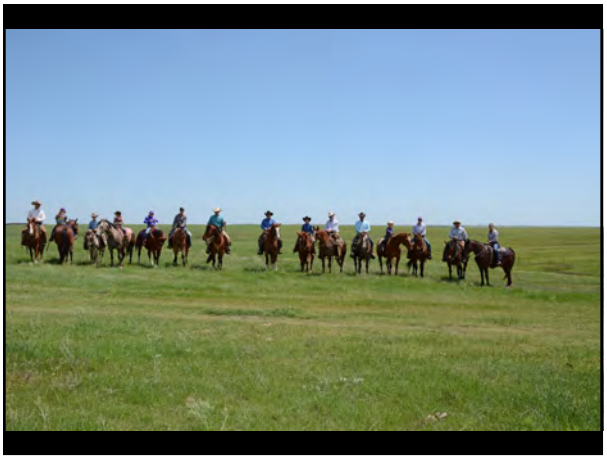
June 5–7, 2022

Common Procedures in Equine Practice

Dr. Warren Beard, Kansas State University


Common procedures in Equine practice
Warren Beard, DVM, MS
Diplomate ACVS

1



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Tenuous repair / extensor tendon laceration

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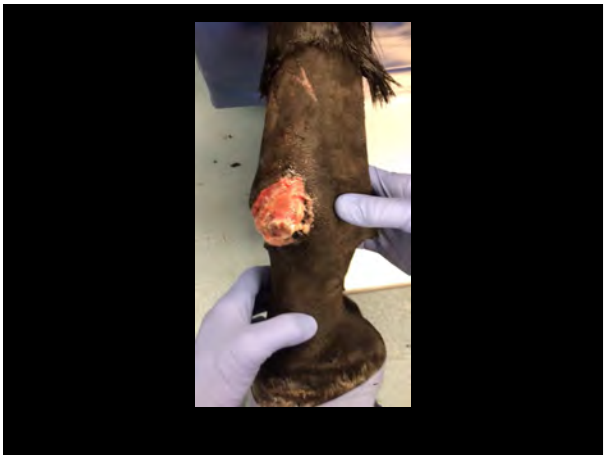

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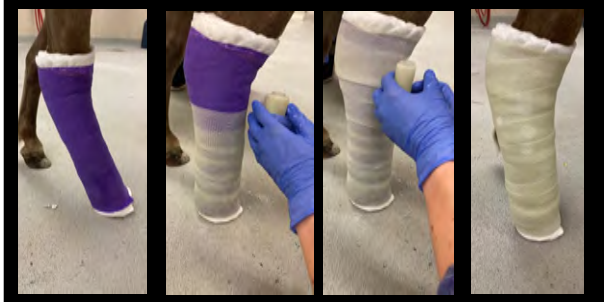
Cast sores

5


Splinting is also an option

6

Cast bandage



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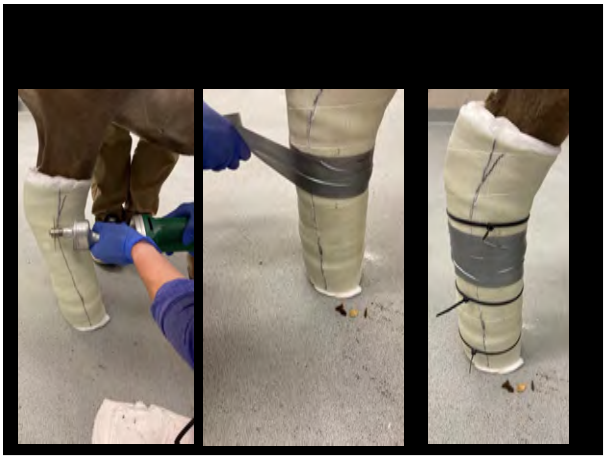

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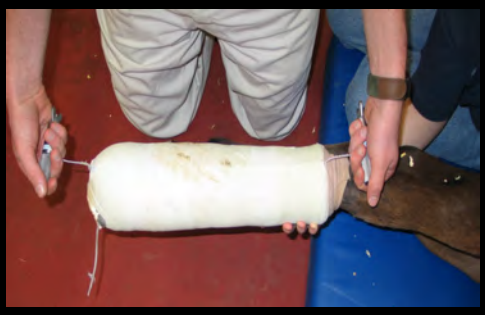
Embedded wires for cast removal



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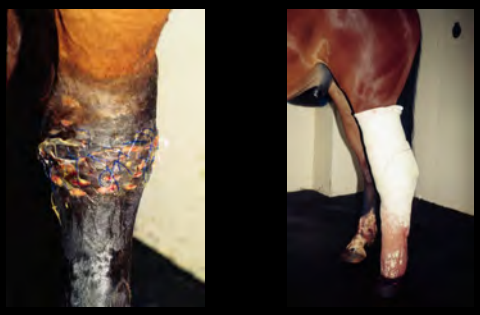

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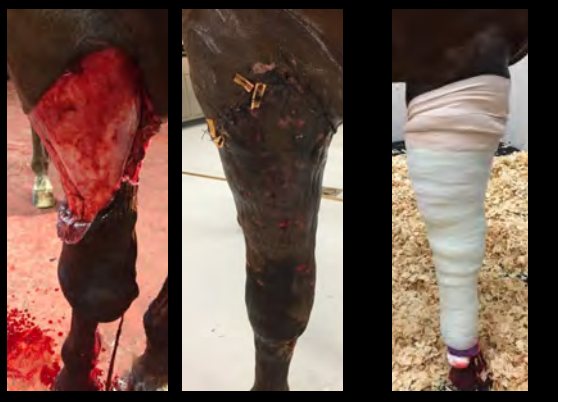

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Advantages of the cast bandage

- Can be performed standing
- Can evaluate the wound as often as desired
- No cast sores
- Easier than splinting
- Owners can manage at home unlike splints
- When less rigidity is required you can throw away the front half and use the back as a perfectly fitted splint

12

Disadvantages of a cast bandage

- Not as rigid of immobilization as a cast
- Exuberant granulation tissue can be more of a problem than with a cast
  - But you can debride it easily

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Clamp herniorrhaphy

15

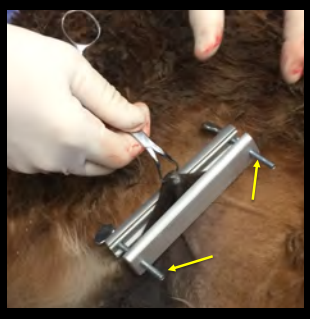
Comparison of herniorrhaphy versus clamping of umbilical hernias in horses: A retrospective study of 93 cases (1982–1994)

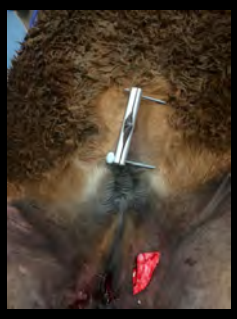
Christopher B. Riley, Antonio M. Cruz, Jeremy V. Bailey, Spencer M. Barber, Peter B. Fretz

Similar complication rate between clams and surgery

16

Use bolt cutters to cut these screws short





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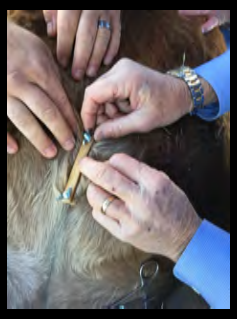

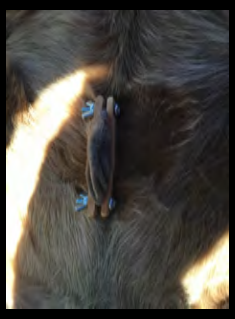

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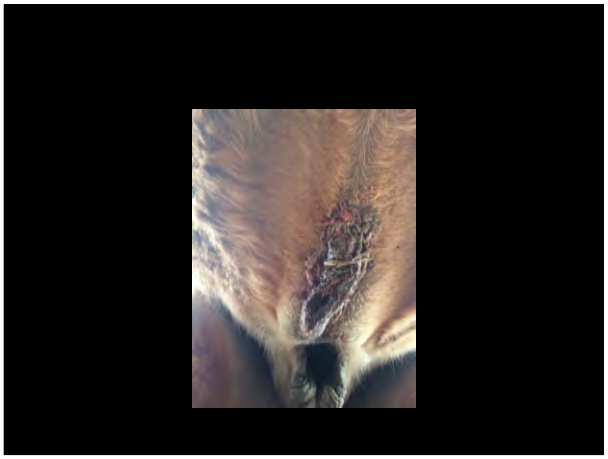

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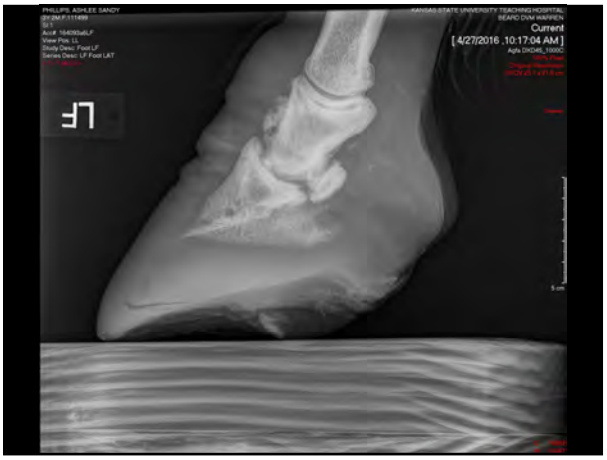

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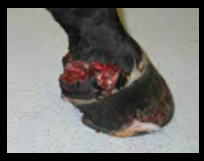
Standing joint lavage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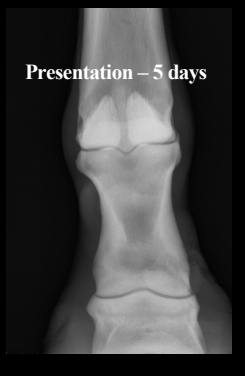

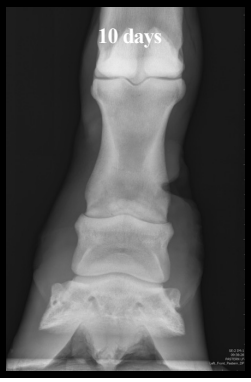

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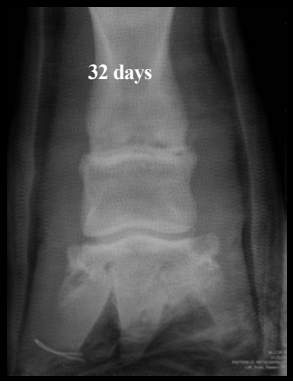

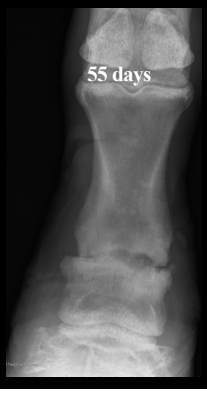

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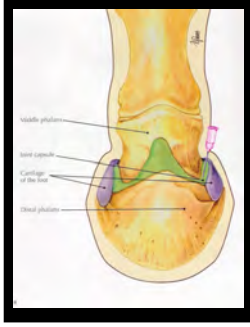
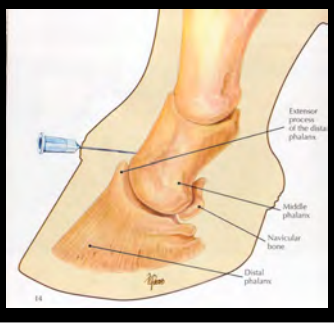
Dorsal midline
1 finger width proximal to the coronary band
perpendicular to the skin
advance needle to you hit P2

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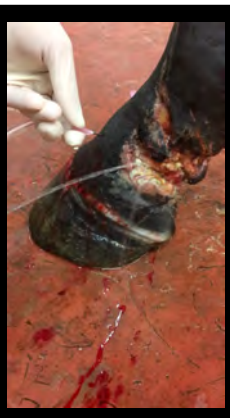

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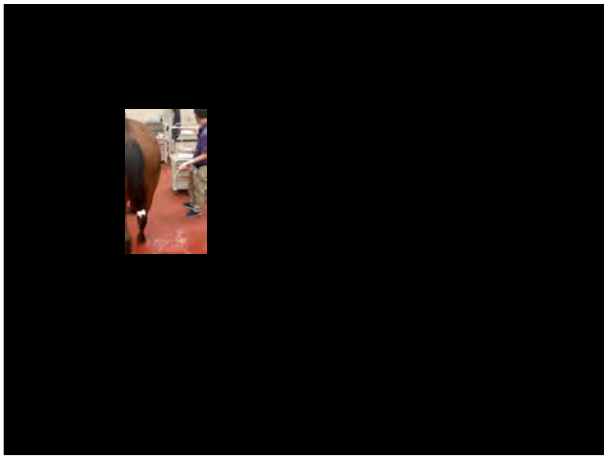

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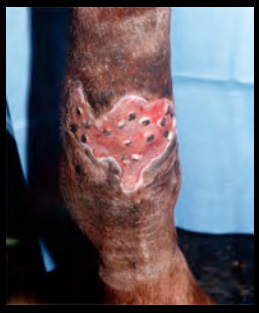
Punch grafting



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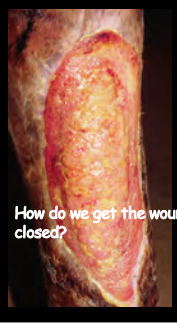
Proliferative Phase



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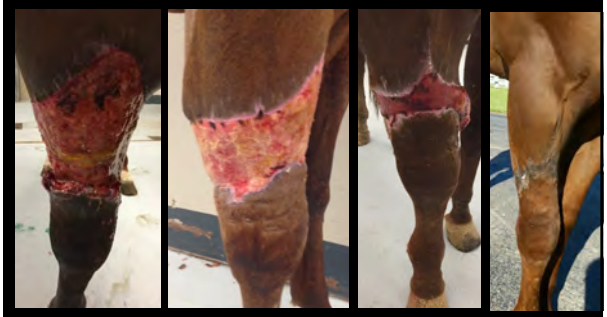
Wound contraction



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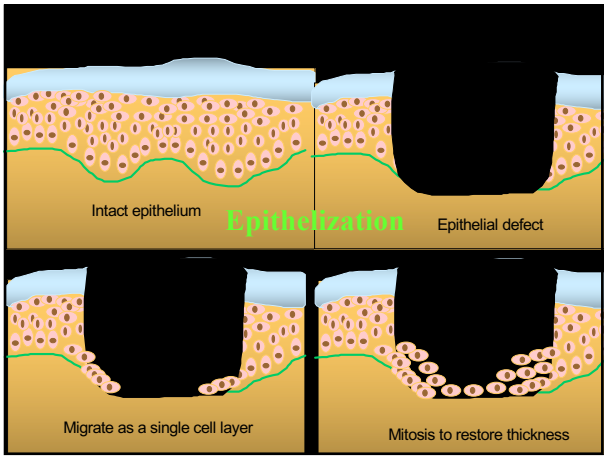

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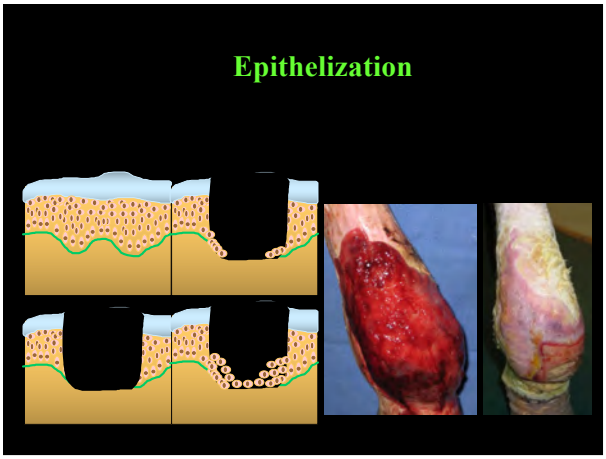

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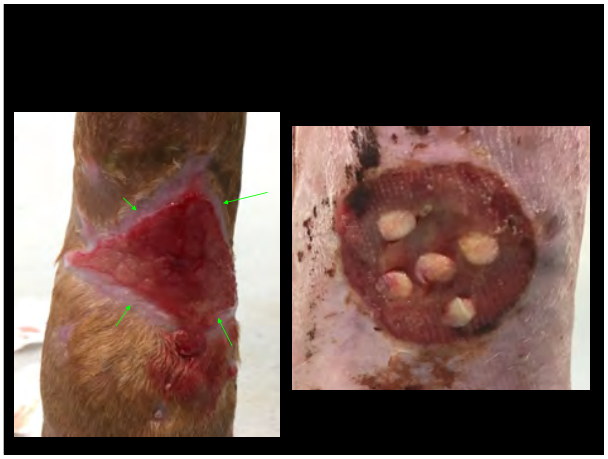

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Two things that make any skin grafting fail

- Infection
- Motion

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Case selection is important

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The day before

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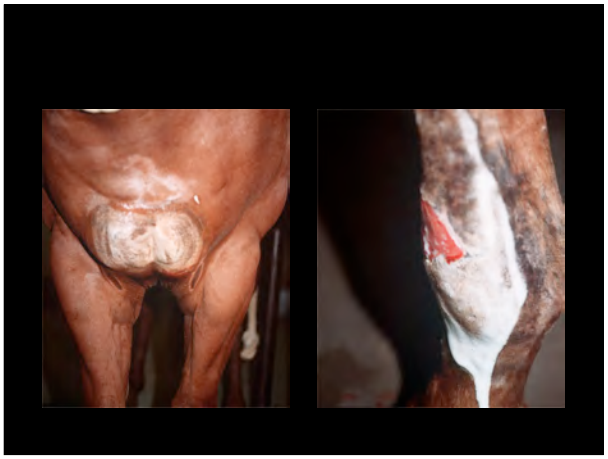

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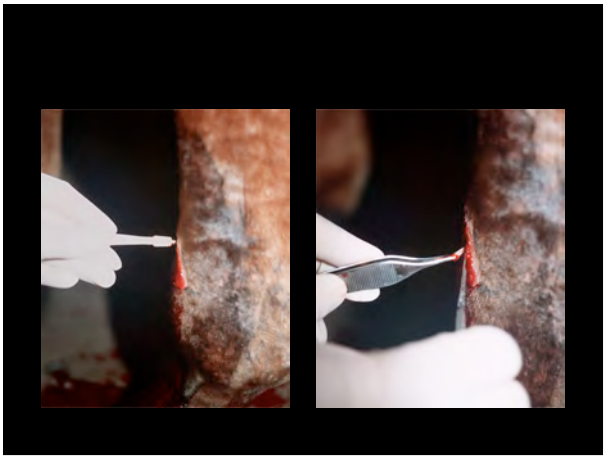

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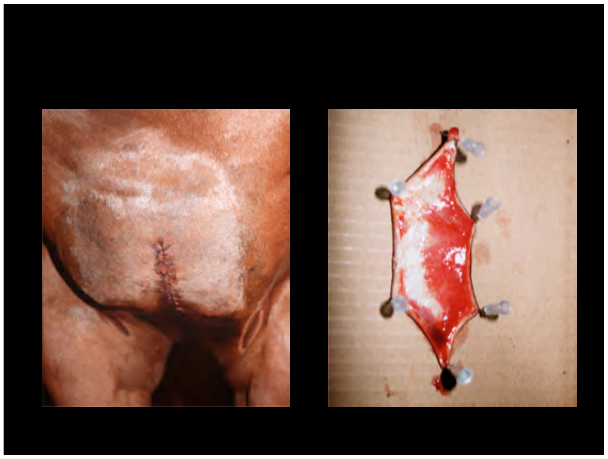

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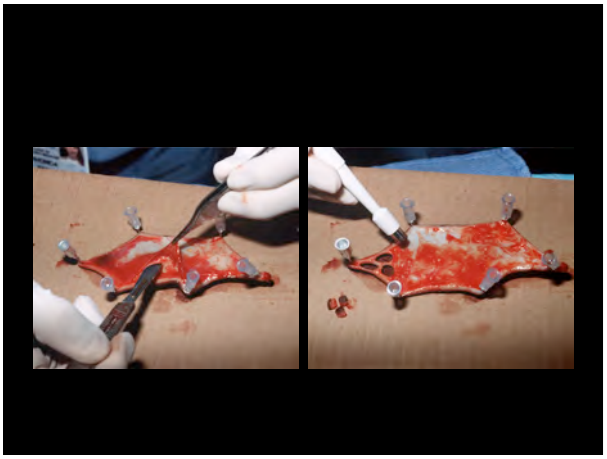

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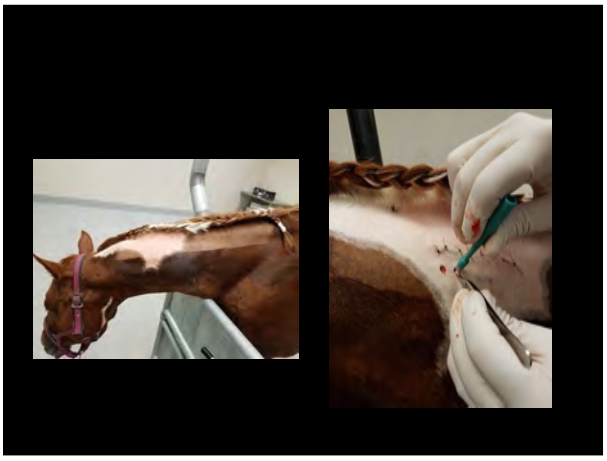

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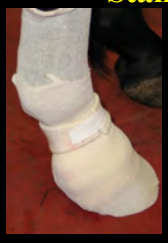
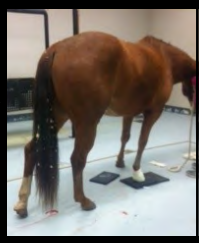
Standing cast application



- Perform in a quiet setting
- Have all equipment ready at hand
- Place stockinette and orthopedic felt
- Sedate horse and make sure that horse is standing squarely
- Work quickly



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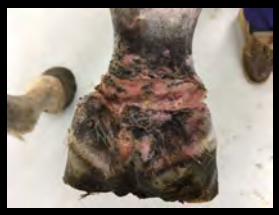
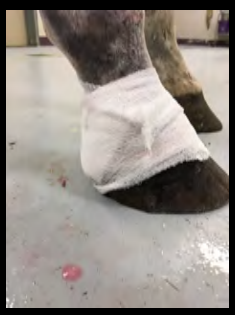
Foot cast





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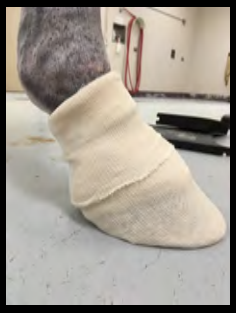

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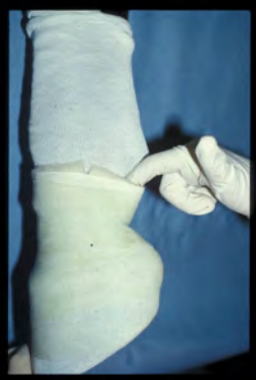

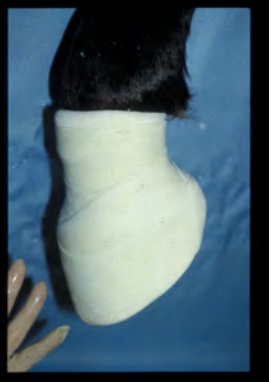

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EQUINE PROCEEDINGS

June 5–7, 2022

Evaluation and First Aid of Equine Musculoskeletal Injuries

Dr. Haleigh Avellar, Kansas State University

Evaluation and First Aid of Equine Musculoskeletal Injuries

Haileigh Avellar, DVM, MS, DACVS(LA)
Clinical Assistant Professor
Large Animal Emergency

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1

Outline

- Patient assessment and stabilization
- Goals
- First aid
- Splinting techniques
  - Fractures, luxations, and traumatic soft tissue injuries
- Transport

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Important considerations

- Temperament
- Age
- Size
- Use
- Prognosis
- Transport
- Referral center
- Euthanasia

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Goals

- Stabilize the patient
- Provide initial wound management if appropriate
- MINIMIZE FURTHER TRAUMA!
  - Assess extent and location of fracture
  - Prevent further damage to fractured bone ends, neurovascular structures, and soft tissues at the fracture site
- Advise the client
  - Cost
  - Transport
  - Prognosis

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Patient stabilization

- Patient triage
- Sedation and analgesia
- Anti-inflammatory medications
- Antibiotics and tetanus prophylaxis
- Fluid therapy
- Hemorrhage
- Prevention of thrombosis

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Patient assessment

- Hemorrhage
  - Shock
  - Estimate blood and fluid loss
    - PCV/TP- do NOT change immediately with acute hemorrhage
- Mentation/attitude
- Heart rate
- Mucous membranes and CRT
- Systemic lactate

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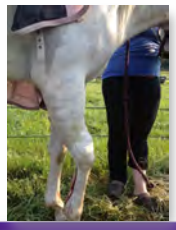
Patient assessment

- "Fracture lame"
  - Do not perform nerve blocks!
- Limb palpation, assess stability
- Diagnosis
  - Imaging- radiographs, ultrasound, CT, MRI
  - Nuclear scintigraphy
- Location
- Open vs. closed
  - Contamination
- Degree of soft tissue damage
- Patent vascular supply distal to fracture
  - Skin temperature
  - Pulse quality



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Patient assessment

- Sedation
  - Allows for more thorough evaluation
  - Safer for you and the horse
  - Be cautious of over-sedating

Do NOT do this!

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Patient assessment

- Sedation and analgesia
  - Alpha2-agonists
    - Xylazine, Detomidine, Romifidine
      - Sedative effects with minimal side effects
      - Short duration of action
  - Phenothiazine tranquilizers
    - Acepromazine
    - AVOID
      - Hypotension in presence of catecholamines
  - Opioids
    - Butorphanol

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Anti-inflammatory medications

- NSAIDs
  - Phenylbutazone, Flunixin meglumine, Ketoprofen, Firocoxib
- Control inflammation
  - Limit risk of thrombosis
  - Maximize perfusion to the leg
  - Provide after adequate stabilization has been achieved
- Dimethyl sulfoxide (DMSO)
  - Reduce edema
  - Scavenge free radicals
- Mannitol
  - Reduce edema in patients with skull fractures

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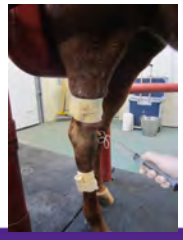
Antibiotics and Tetanus Prophylaxis

- Broad spectrum systemic antimicrobials
  - Penicillin
  - Gentamicin
- Regional limb perfusion
  - Amikacin
- Tetanus toxoid and/or antitoxin



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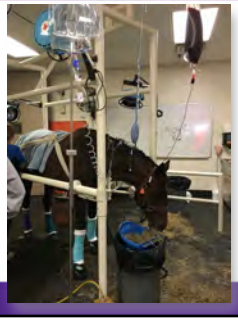
Fluid therapy

- Intravenous crystalloids for fluid volume replacement
  - Controlled vs bolus administration
  - Patients with profuse sweating, pain, or substantial blood loss from hemorrhage
- Volume depends on many factors
  - Minimum dose of 10L (20mL/kg), difficult to overhydrate an adult
  - Increase for patients showing signs of shock
- Neonates
  - Limited fluid and energy reserves
  - Add dextrose if unable to nurse



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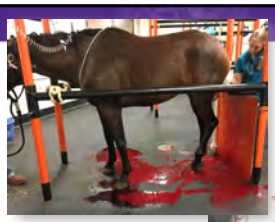
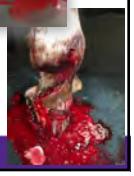
Hemorrhage

- Ligate if possible
- Pack the wound/ apply pressure
- If significant bleeding cannot be controlled:
  - Aminocarproic acid
    - 10-20mg/kg IV diluted in 0.9% saline (1-3L)
    - Inhibit fibrinolysis
    - Stabilize the clot
  - 10% Formalin
    - 50ml in 1L Saline or LRS





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Prevention of thrombosis

- Especially important in certain fracture types that disable the suspensory apparatus or those with severe soft tissue damage
- Any injury causing severe stretch of the palmar or plantar vessels at increased risk
- Aspirin
  - 10-20 mg/kg EOD
- Heparin
- Doppler ultrasonography

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Initial wound care

- Prevent the progression of contamination to infection
- Sterile lube in the wound, clip surrounding hair
- Lavage with surgical antiseptic solutions and sterile saline
- Evaluate for synovial or bone involvement
- Apply sterile dressings
- Open fractures
  - Immediate administration of IV broad spectrum antimicrobials
    - Pen, Gent

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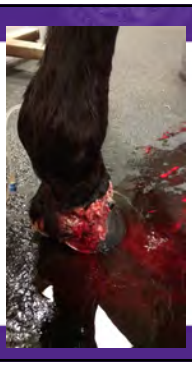
Synovial involvement



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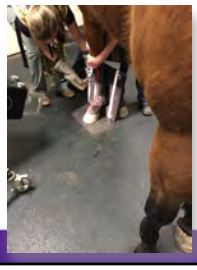
Initial fracture assessment

- Radiographs
  - Unstable distal limb fractures should be stabilized first!
  - Stressed radiographs
  - Soft tissue injuries
- Prognosis



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Principles/Goals of Fracture Stabilization

- Prevent further injury
- Assist with weight bearing
- Relieve anxiety and pain

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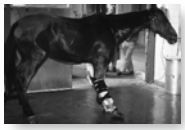
Fracture splinting guidelines

- Immobilize joints proximal and distal to the fracture
- Never end a cast or splint in the mid-diaphysis of a long bone
- Never end a cast or splint near the fracture line to be stabilized
  - Fulcrum effect
- When possible, include the foot in the coaptation
- Use adequate padding to protect skin from injury
  - Tight bandage– no motion
- Excessive padding will provide inferior stabilization



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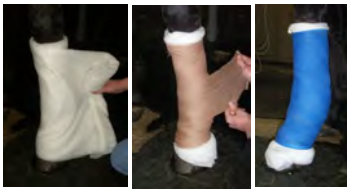
Bandaging

- Apply sterile dressing to any wounds
  - Non-adherent pad and kling gauze
- Cotton combine
- Brown gauze
- Vet rap
- Tight and even pressure with all layers



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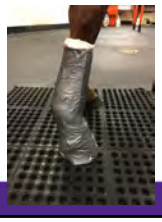
Splints

- PVC, wood, broom handle, cast material
- Apply using non-elastic tape
  - Duct tape!



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Other External Coaptation to Consider

- Bandage Cast
- Cast

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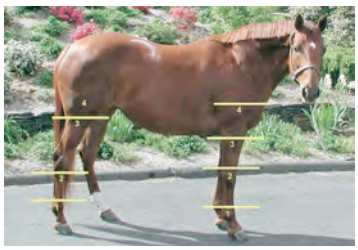
Guidelines for External Stabilization



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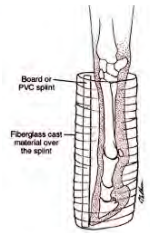
Level 1 – forelimb

- Fractures
  - Middle phalanx
  - Proximal phalanx
  - Proximal sesamoid bones
  - Distal MC3
- Luxations
  - PIP
  - MCP
- Goal
  - Align dorsal cortices
  - Neutralize bending forces
- **Dorsal splint from ground to carpus**



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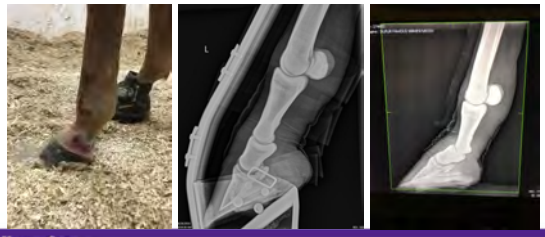
Level 1 – forelimb



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Level 2 – forelimb

- Fractures
  - Mid MC
  - Proximal MC
  - Carpus
  - Distal radius
- Goal
  - Maintain alignment of bony column
- Robert Jones bandage
  - 3 times diameter of limb
- **Caudal and lateral splints both from elbow to ground**
- **Cast from hoof to elbow**

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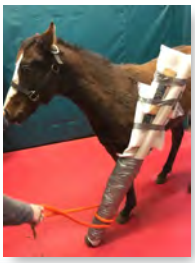
Level 3A – forelimb

- Fractures
  - Mid radius
  - Proximal radius
- Goal
  - Maintain alignment of bony column
  - Immobilize distal limb
  - Prevent abduction of limb
- **Caudal splint from ground to elbow, lateral splint from ground to shoulder**



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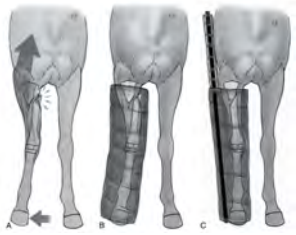
Level 3A – forelimb



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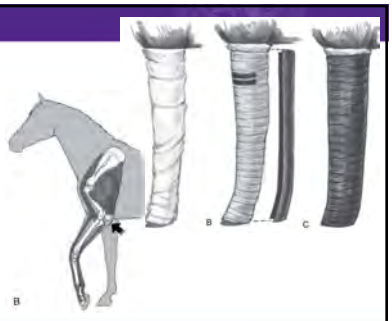
Level 3B – forelimb

- Fractures
  - Ulna
- Goal
  - Maintain alignment of bony column
  - Maintain passive stay of forelimb (triceps)
- **Caudal splint from ground to elbow**



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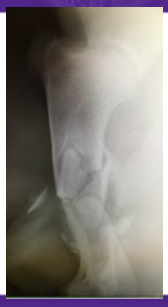
Level 4 -- forelimb

- Fractures
  - Humerus
  - Scapula
- **No stabilization required**
- Goal
  - Minimize stress and pain
  - Usually severe neurovascular compromise and swelling due to hemorrhage
  - Discuss prognosis prior to transportation



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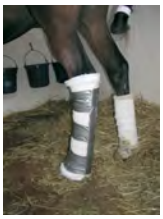
Level 1 – hind limb

- Fractures
  - Middle phalanx
  - Proximal phalanx
  - Proximal sesamoid bones
  - Distal MT3
- Luxations
  - PIP
  - MCP
- Goal
  - Align dorsal cortices
  - Neutralize bending forces
- **Caudal splint from ground to tarsus**



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Level 2 – hind limb

- Fracture
  - Mid MT
  - Proximal MT
- Goal
  - Align bony column
  - Immobilize distal limb
- **Caudal splint from ground to calcaneus, lateral splint from ground to stifle**

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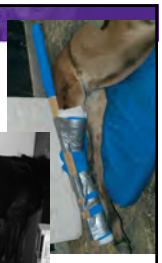
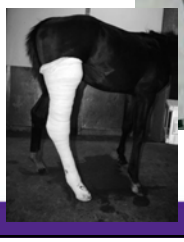
Level 3 – hind limb

- Fractures
  - Tarsus
  - Tibia
- Goal
  - Maintain alignment of bony column
  - Immobilize distal limb
  - Prevent abduction of limb
- **Wide lateral splint from the ground to the widest part of the hip**
- **Foals: cast foot to above stifle**





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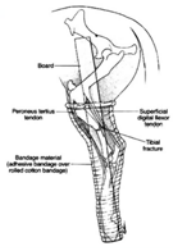
Level 3 – hind limb



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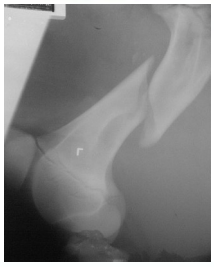
Level 4 – hind limb

- Fractures
  - Femur
  - Pelvis
- **Generally do NOT require splinting**
  - Fulcrum effect



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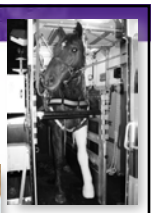
Transportation

- Minimize distance to the trailer
- Ramps
- Forelimb injury
  - Load & transport backwards
- Hind limb injury
  - Load and transport forwards
- Head and neck free for balance



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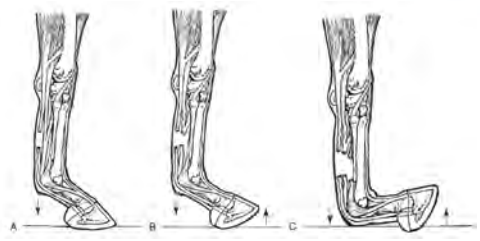
Distal limb lacerations



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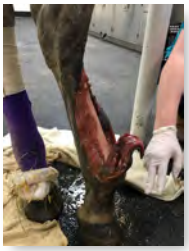
Extensor vs flexor tendon lacerations



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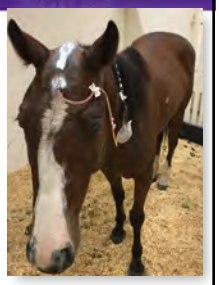
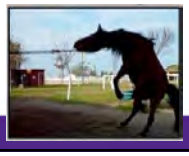
Head trauma

- Stabilize the patient
- Examine for neurological deficits
- Anti-inflammatories
  - NSAIDS, Steroids, DMSO, etc!





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Questions

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EQUINE
PROCEEDINGS

June 5–7, 2022

Feeding the Equine Athlete

Dr. Randy Raub, Kent Nutrition Group

Notes

EQUINE PROCEEDINGS

June 5–7, 2022

How Much Foo is Required in Foo Foo Dust?

Dr. Randy Raub, Kent Nutrition Group

Notes

EQUINE PROCEEDINGS

June 5–7, 2022

Is This Stud a Dud? Case Series of Managing Stallions for Breeding

Dr. Jason Grady, Kansas State University

Is This Stud a Dud? Case Series of Managing Stallions for Breeding

Jason Grady, DVM, MS, DACVIM
Kansas State University

Throughout a breeding season, pressures are put on clinicians that are managing brood mares to maximize fertility. In the mare, breeding soundness examinations allow us, to the best of our ability, the opportunity to ensure mare breeding soundness through the use of thorough physical examinations, rectal palpation, trans-rectal ultrasound, uterine culture, uterine cytology, and uterine biopsy. However, fertility in stallions is often overlooked. Although complete infertility is rare, reproductive problems do exist in stallions quite frequently. For clinicians that are actively involved in breeding management of mares and stallions it is critical that they be aware of common causes of decreased fertility such as: sperm accumulation/spermiostasis, testicular degeneration, partial ejaculation, failure to ejaculate, poor intrinsic fertility, sexually transmitted diseases, hemospermia/urospermia, and trauma.

Prior to breeding season, it is recommended to perform a thorough stallion breeding soundness examination including a thorough physical examination, lameness/neurological evaluation, evaluation of the external genitalia, internal accessory sex glands, observation of a semen collection, semen analysis, as well as bacterial culture of shaft of the penis, pre and post-ejaculatory urethra, and urethral fossa. Serological testing for Equine Arteritis Virus is also recommended. Following semen collection, evaluation of the ejaculate should include: volume, concentration, motility, morphology, and viability. There are a number of other parameters that can be evaluated, but those that have been mentioned may be easily be performed in a clinic with a microscope, hemocytometer, and eosin-nigrosin stain.

As stallions are managed for artificial insemination with fresh, cooled or frozen semen, we evaluate every ejaculate for those parameters that were previously mentioned. Furthermore, it is advised to evaluate every sample of fresh, chilled semen and frozen semen that gets shipped in to inseminate mares via artificial insemination. This provides the clinician the direct knowledge of what the mare has been inseminated with. There are a variety of breeding management numbers that can be reviewed to assess a stallion's reproductive efficiency such as per-cycle pregnancy rate and season ending pregnancy rate.

The Society for Theriogenology (SFT) has set minimum standards when evaluating a stallion for breeding soundness. To be considered a satisfactory breeder a stallion must have: normal libido and gait, two descended testicles, no scrotal or penile abnormalities, a total scrotal width of $\geq$8 cm, normal bacterial culture, $\geq$ 1 billion progressively motile, morphologically normal sperm in the second ejaculate one hour after the first ejaculate, free of venereal diseases, and free of heritable defects. The stallion is also expected to impregnate 75% of a full book of mares in $\leq$ 2 normal estrous cycles. A full book is considered 40 mares by natural service or 120 mares by artificial insemination. It is expected that stallions have at least 60% progressively motile and morphologically normal sperm. Stallions are considered questionable or unsatisfactory breeders if they fail to pass one or two of the parameters previously mentioned. In cases with an abnormal spermiogram, stallions that are questionable or unsatisfactory may be reevaluated 60 days from the initial evaluation to assess if the abnormalities are temporary or permanent. Stallions may not be considered sexually mature until they are four years of age. Younger stallions should

definitely be allowed to mature and be reevaluated before considering them unsatisfactory. Unlike some other species, stallions that are classified as questionable or unsatisfactory breeders are rarely retired. Instead, we are often asked to help stallion owners develop management practices to maximize the stallion's reproductive efficiency.

EQUINE PROCEEDINGS

June 5–7, 2022

Clinical Management of Endometritis

Dr. Jason Grady, Kansas State University

Clinical Management of Endometritis

Jason Grady, DVM, MS, DACVIM
Kansas State University

Horses are seasonal breeders and their natural breeding season coincides with long photoperiod (Aurich, 2011). The endometrium, the lining of uterus changes with stage of the estrous cycle, season, and age (Britton, 1982; Kozdrowski et al., 2015). The endometrium plays an important role in regulating estrous cycles as it produces prostaglandin F2α (PGF2α), a major luteolysin in mares (Kozai et al., 2016). Chronic bacterial infection of the uterus causes inflammation of the endometrium, a condition commonly referred to as bacterial endometritis. It is the leading cause of subfertility in broodmares (Dimock and Edwards, 1928; Traub-Dargatz et al., 1991). Non-infectious causes of inflammation/endometritis commonly occur post-mating. A presumptive diagnosis of post-mating induced endometritis may be made if more than two centimeters of uterine fluid is observed via trans-rectal ultrasound during estrus or present 24-36 hours post-breeding. Normally, the defense mechanisms consisting of physical (vulva, vestibular-vaginal sphincter, and cervix) and mechanical (uterine clearance of bacterial and inflammatory products) barriers and the innate immune system prevent bacterial infection in mares (Liu IK, 1988; Ferris, 2014). Properly functioning physical barriers prevent feces, air, and environmental pathogens from reaching the uterus. It has recently been described that a normal bacterial flora exists within the mare's uterus. Any abnormality or decreased function of the described physical barriers increases the probability of bacteria gaining access to the uterus and increasing the likelihood of bacterial infection. The presence of bacteria within the uterus subsequently activates the mare's innate immune system through the rapid influx of neutrophils, immunoglobulins and serum proteins (Ferris, 2014). Mares are commonly referred to as resistant when they are capable of clearing the bacterial infection through their innate immune function and through mechanical clearance (Hughes and Loy, 1969). However, some mares are susceptible to bacterial infection due to their inability to clear infection. Susceptible mares have 1) decreased ability to phagocytize bacteria (Cheung et al., 1985), 2) decreased uterine clearance through decreased uterine contractions, 3) ineffective uterine contractions, and/or 4) decreased lymphatic clearance when compared to resistant mares

(LeBlanc and Causey, 2009). Failure to clear bacteria or inflammatory products from the uterine lumen results in the continued activation of the innate immune system and continuous production and accumulation of inflammatory products (Ferris, 2014).

Obligate bacteria normally inhabit the external genital surfaces of mares and stallions and are introduced into the uterus during mating or if a mare has compromised function of the vagina or cervical region (Ricketts and Mackintosh, 1987). However mechanisms such as mating-induced transient uterine inflammation and activation of the innate immune response clear the bacteria from the uterus along with excessive spermatozoa and uterine fluid/debris within 24-36 hours post-mating (Christoffersen and Troedsson, 2017). Susceptible mares remain inflamed beyond 48-72 hours with retention of intrauterine fluid (Troedsson, 1999) and are therefore unable to clear bacteria post-mating. If present, opportunistic pathogens such as *Streptococcus equi* subsp. *zooepidemicus*, *E. coli, Pseudomonas aeruginosa,* and *Klebsiella pneumonia* are able to survive in the uterine fluid and proliferate in uterus of susceptible mares (LeBlanc and Causey, 2009). Cell surface proteins and proteins in the uterine fluid act as a conditioning film or coat to which bacterial cilia or flagella initially attach. The initial attachment of bacteria to conditioning film is not strong but with time, the attachment becomes stronger as pathogens begin to secrete polysaccharides. Polysaccharides on the bacterial surface act as ligands and irreversibly bind to surface proteins on uterine epithelium (i.e. endometrium) and serum proteins in the uterine lumen thus forming a thin film called biofilm. The biofilm matures as pathogens continue to grow forming a multilayer and eventually take the form of microcolonies in the biofilm. During the growth phase, the primary bacteria may attract other microbes (called secondary colonizers). Bacterial growth may be focal or uniformly spread within the uterine cavity. Bacteria integrated into the biofilm are resistant to the host's immune system and to antibiotics because polysaccharides act as a diffusion barrier allowing chronic infections to develop (Ferris, 2014; Beehan et al., 2015). Bacteria residing deep within the biofilm have a decreased metabolic rate and are often referred to as "persister cells" as these bacteria are metabolically inactive. In order for most antibiotics to be effective, bacteria must be metabolically active and/or rapidly multiplying (Morris et al, 2020). Often, persister cells are multi-drug resistant thus making it hard to treat the chronic infection (Anderson and O'Toole, 2008). In summary, loss of uterine

defense mechanisms (such as inability to uterine clearance, cervical competence, age, and perineal conformation etc.) and microbial flora and their secretions contribute to the pathogenesis of endometritis, often referred as *bacterial endometritis*. Endometritis is the major cause of reproductive failure including recurrent miscarriage in mares (Traub-Dargatz et al., 1991; Kitaya, 2011).

When addressing a mare with endometritis, a thorough history and review of breeding management should be performed. Is the problem a management problem, a problem with the mare, or problem with the semen/stallion? Has the mare been bred at the proper time with good quality semen to provide an adequate opportunity to become pregnant? Adequate history to ensure that no issues such as post-mating fluid accumulation, no evidence of vaginal or vulvar discharge were present, and that edema patterns are consistent with the stage of the estrous cycle. In addition to a review of the breeding records and history, a routine physical examination and complete breeding soundness examination should be performed.

Diagnosis of endometritis may be diagnosed in mares by a combination of diagnostics such as rectal palpation, transrectal ultrasound, and recognition of short inter-estrous intervals, along with vaginal examination, uterine cytology, uterine culture, and uterine biopsy. **Uterine culture taken in the absence of a uterine cytology only provides partial information.** Additional diagnostics to consider with the subfertile mare include:

1) Separate cultures of the clitoral fossa, vagina, cervix and uterus to look for potential reservoirs of infection
2) Low volume uterine lavage for culture and cytology
3) Endoscopy (hysteroscopy) of endometrium to look for focal infections or other pathologies
4) Culture through sterile speculum or “double sterile glove” with culture swab between gloves to avoid vaginal/perineal contamination
5) Ultrasound 12 to 24 hours post-insemination to diagnose delayed uterine clearance/post-mating endometritis

6) Biopsy following treatment/resolution may provide better information for prognosis for pregnancy. However, culture of uterine biopsy or special stains of biopsy may be utilized as more advanced diagnostics.

Use of antibiotics should be based on clinical signs, cytological evidence of inflammation, uterine culture and sensitivity. A combination of uterine irrigation and oxytocin injection (to stimulate uterine muscular contraction for uterine drainage) is helpful in clearing an intrauterine inoculum.

Biofilm producing bacteria make treatment challenging. Directing the treatment at disrupting the biofilm and eliminating the biofilm-associated bacteria is essential. (Morris, 2020). Therapeutics for the mare with endometritis that are currently employed in various combinations are judicious use of antibiotics, flushing uterus with saline, intrauterine antibiotics and chelators (EDTA–Tris), mucolytics (DMSO, kerosene, N-acetylcysteine), corticosteroids (prednisolone, dexamethasone), immunomodulators (cell wall extracts of *Mycobacterium phlei* and *Propionibacterium acnes*), ceragyn, and bActivate.

Minimizing post-mating induced endometritis can be achieved by attempts to minimize the number of times the mare is inseminated/mated to only once during an estrous cycle. This is will likely improve efficiency by inseminating the mare close to ovulation, but minimize the normal post-mating inflammatory response without the repeated inflammatory stimulation due to multiple doses of semen that is acting as an inflammatory stimuli. Deep horn insemination may result in less inflammation than conventional uterine body insemination (Morris, 2020). This is best achieved by managing the mare closely and inducing ovulation. Reducing the prolonged inflammatory effects of post-mating induced endometritis are also achieved by implementing uterine lavage with sterile saline or Lactated Ringers Solution in combination with ecbolic agents such as oxytocin or PGF2α within 48 hours of breeding. These are commonly implemented starting four hours post-insemination. Administration of intravenous dexamethasone (0.1 mg/kg) at the time of insemination has been shown to suppress the inflammatory response. Further considerations should include: choosing fertile stallions, along with the ideal breeding

method to maximize longevity of semen and minimize contamination during the breeding process.

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EQUINE PROCEEDINGS

June 5–7, 2022

Accreditation Module 18 – Avian Influenza & Newcastle Disease

USDA–APHIS Veterinary Medical Officers

Module 18: Avian Influenza & Newcastle Disease

USDA-APHIS National Veterinary Accreditation Program (NVAP)

Required

- Sign-in
  - iPad or paper
- Retain certificate of completion
- Complete 2-step process to renew accreditation
  - Complete modules
  - Submit an application

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- Tap each field to enter data*
- Select Submit
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  - If not accurate, see me after presentation
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- Your name must match what is in our records.

  e.g. “Jim” not “James”, “McDonald” vs “Mc Donald”
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Supplemental Training

- Familiarize accredited veterinarians with animal health regulatory concepts and activities
  - Does not supersede regulations
- For the most up-to-date regulations and standards, please refer to:
  - Code of Federal Regulations
  - OSHA
  - Occupational health specialist
  - Local VS District Office

Supplemental Training

- All APHIS Approved Supplemental Training (AAST) modules are also available on our Website with interactive features and links to additional Web resources.
- Type “NVAP” into your search engine, e.g., Bing, Google, Yahoo.

Overview

- Economic and public health impacts of disease outbreaks
- Clinical signs of avian influenza (AI) and Newcastle disease (ND)
- Concerns associated with H5/H7 LPAI viruses
- Roles of National Poultry Improvement Plan (NPIP) and Live Bird Marketing System (LBMS) programs
- Collection and submission of samples
- Reporting positive results
- Biosecurity measures

Introduction

- AI, ND similar, economically important
- Causative viruses
  - Mildly pathogenic to highly virulent
  - Mild forms in United States
  - Severe forms reportable to State Animal Health Official (SAHO) and Assistant Director (AD)

Avian Influenza

Avian Influenza: History

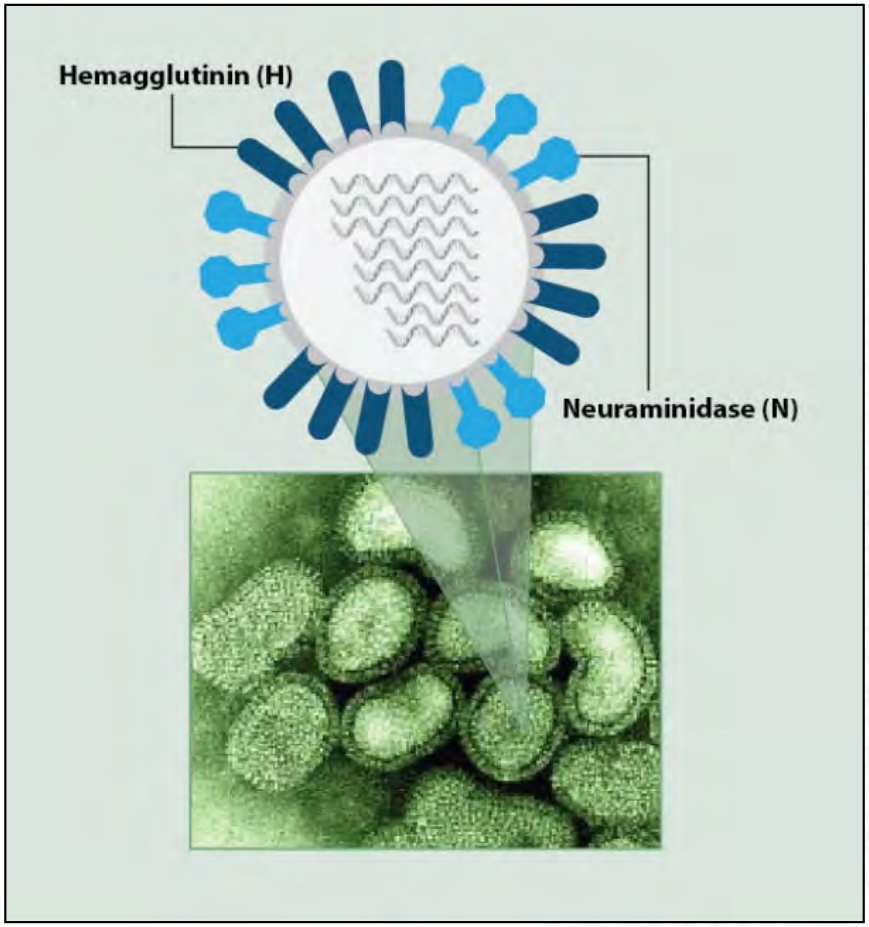
- Once called "fowl plague"
- 1878: Discovered in Italy
  - Endemic for 50 years, spread
- 1901–1930: Europe, North and South America, Egypt, China, Japan
  - Self-limiting outbreaks
  - Depopulations—mandatory, voluntary
  - Disease control mistakes spread AI
  - Germany: "Brunswick disease"

First AI Outbreak: United States

- 1924–1925
- Live bird markets, railways spread disease
- Restrict imports from affected States
- Federal programs
  - Depopulating infected flocks
  - Bury/burn carcasses
  - Clean, disinfect

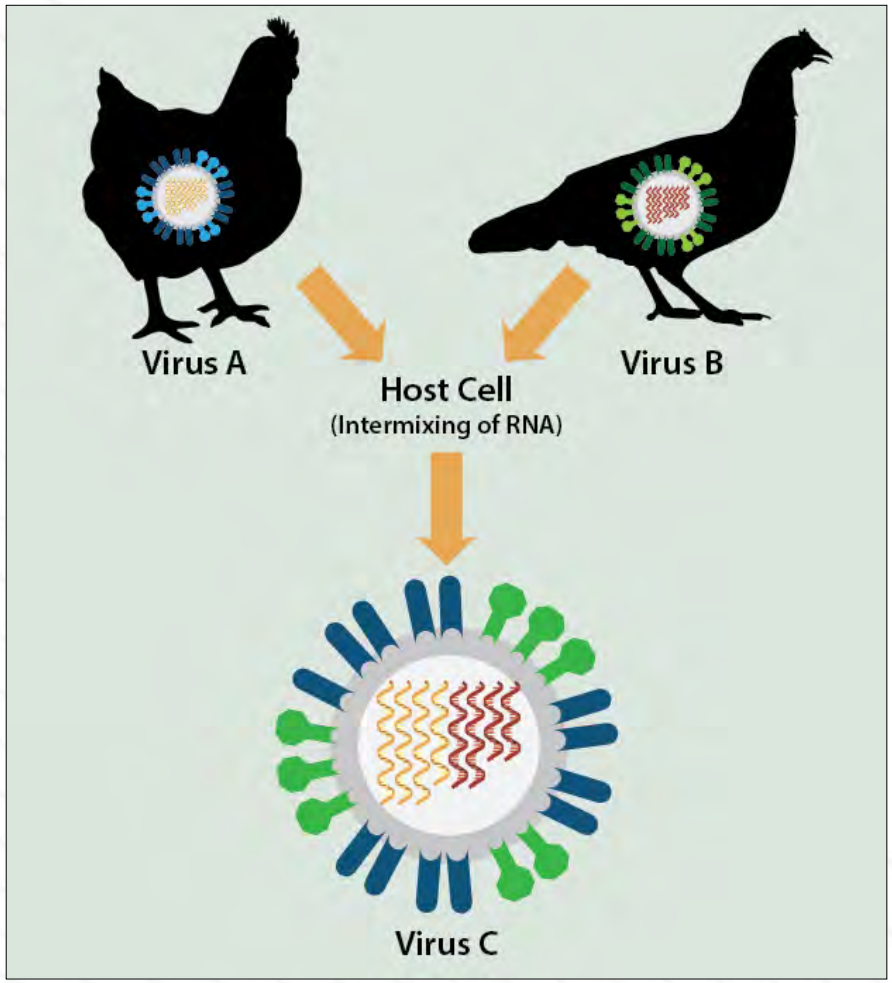
AI Viruses: Genetic Variability

- Genus — *Influenzavirus* A
- Family — *Orthomyxoviridae*
- Subtypes classified by 2 surface proteins
  - Hemagglutinin (H1–H16)
  - Neuraminidase (N1–N9)
  - 144 potential combinations

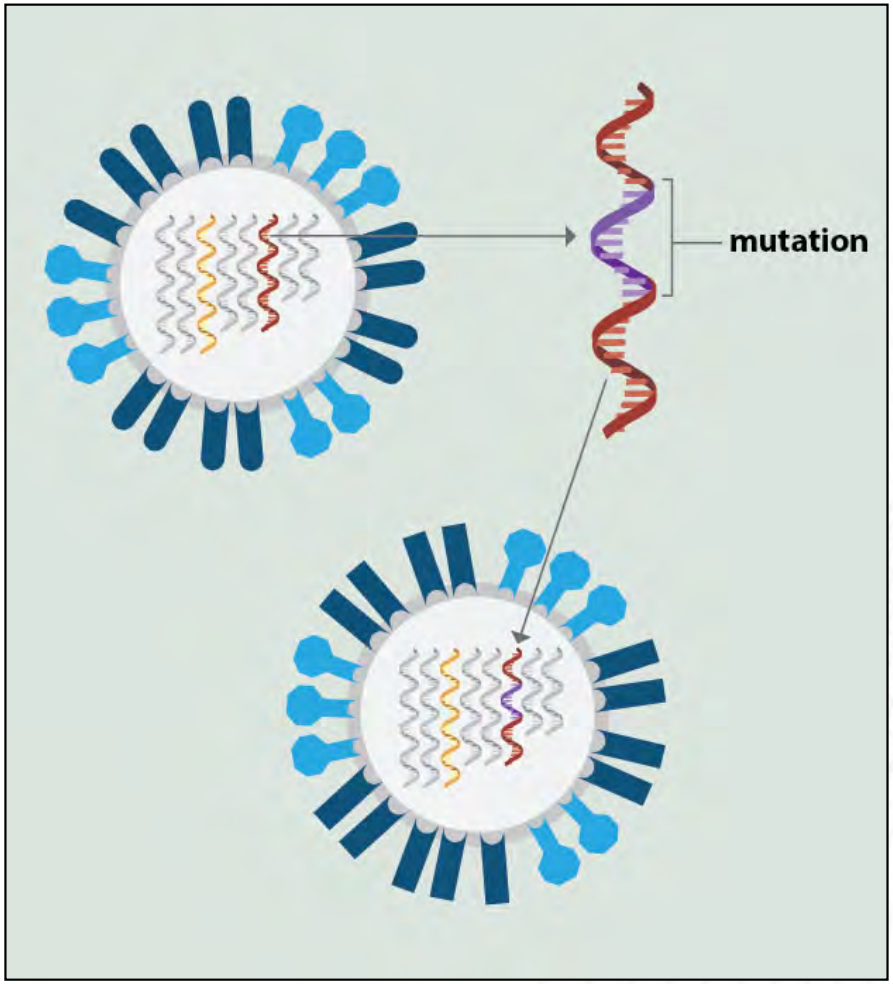


AI Viruses: Genetic Variability

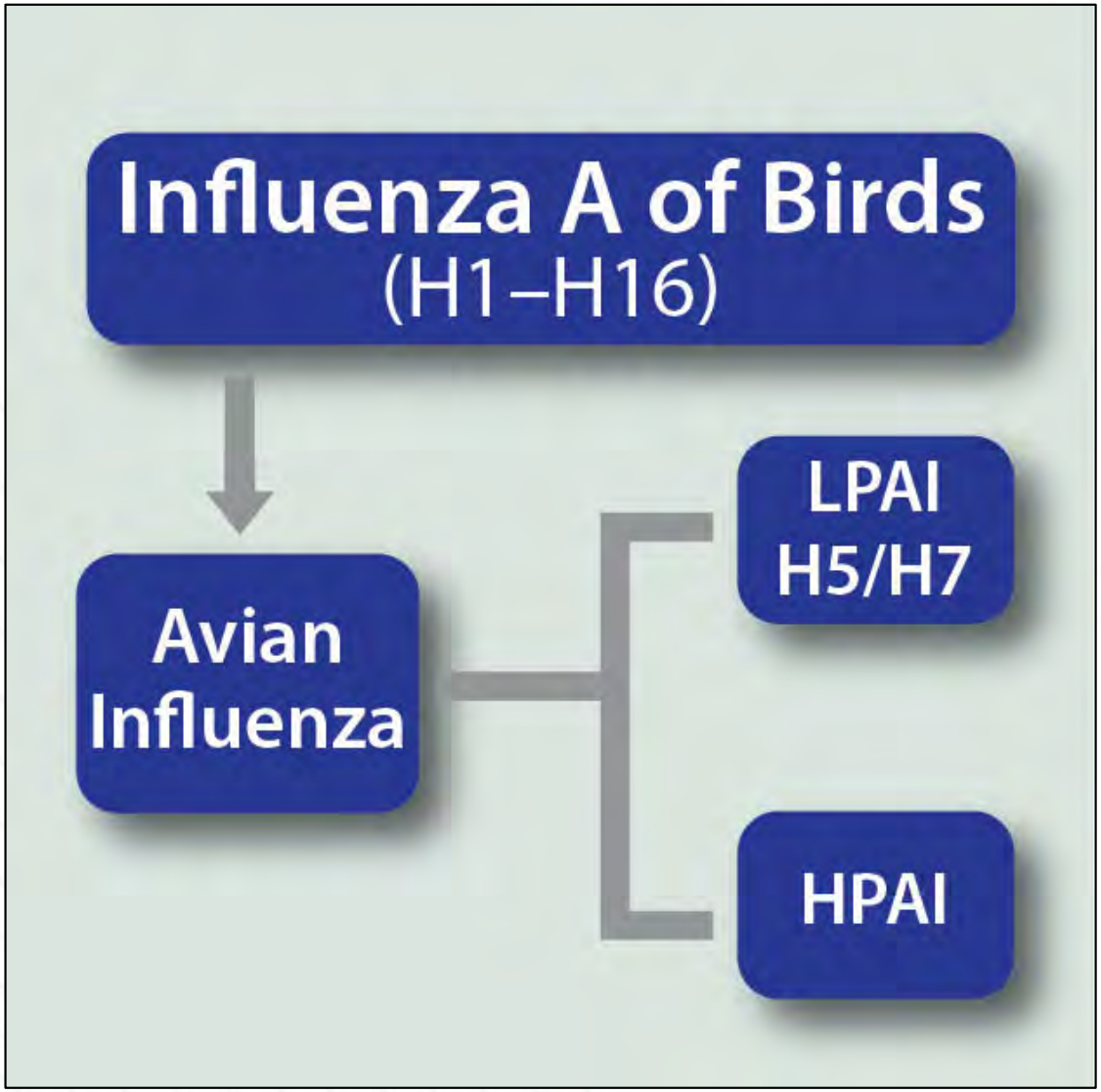
Antigenic Shift



Antigenic Drift



Influenza A of Birds



LPAI

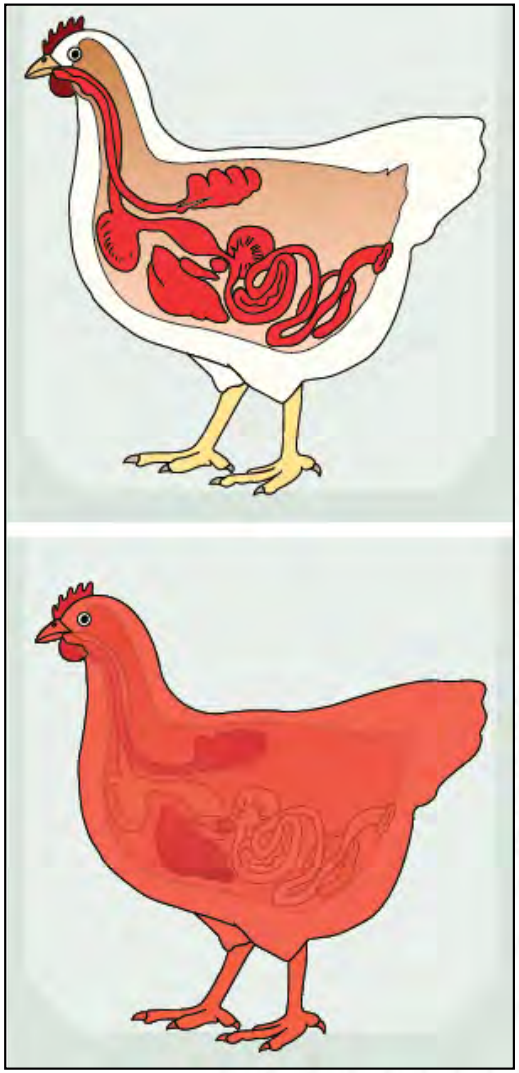
- Only H5 and H7 subtypes
- Limited replication, primarily in the respiratory and GI tracts
- Mild clinical signs
- H5/H7 viruses may mutate to HPAI viruses in non-host species
- Early detection and response crucial

HPAI

- LPAI H5/H7 can become HPAI
- Among poultry
  - Cause severe systemic disease
  - Morbidity and mortality near 100%
- Must meet specific virulence criteria
- Must contain certain genetic motifs at cleavage site of hemagglutinin
- Results in international trade restrictions

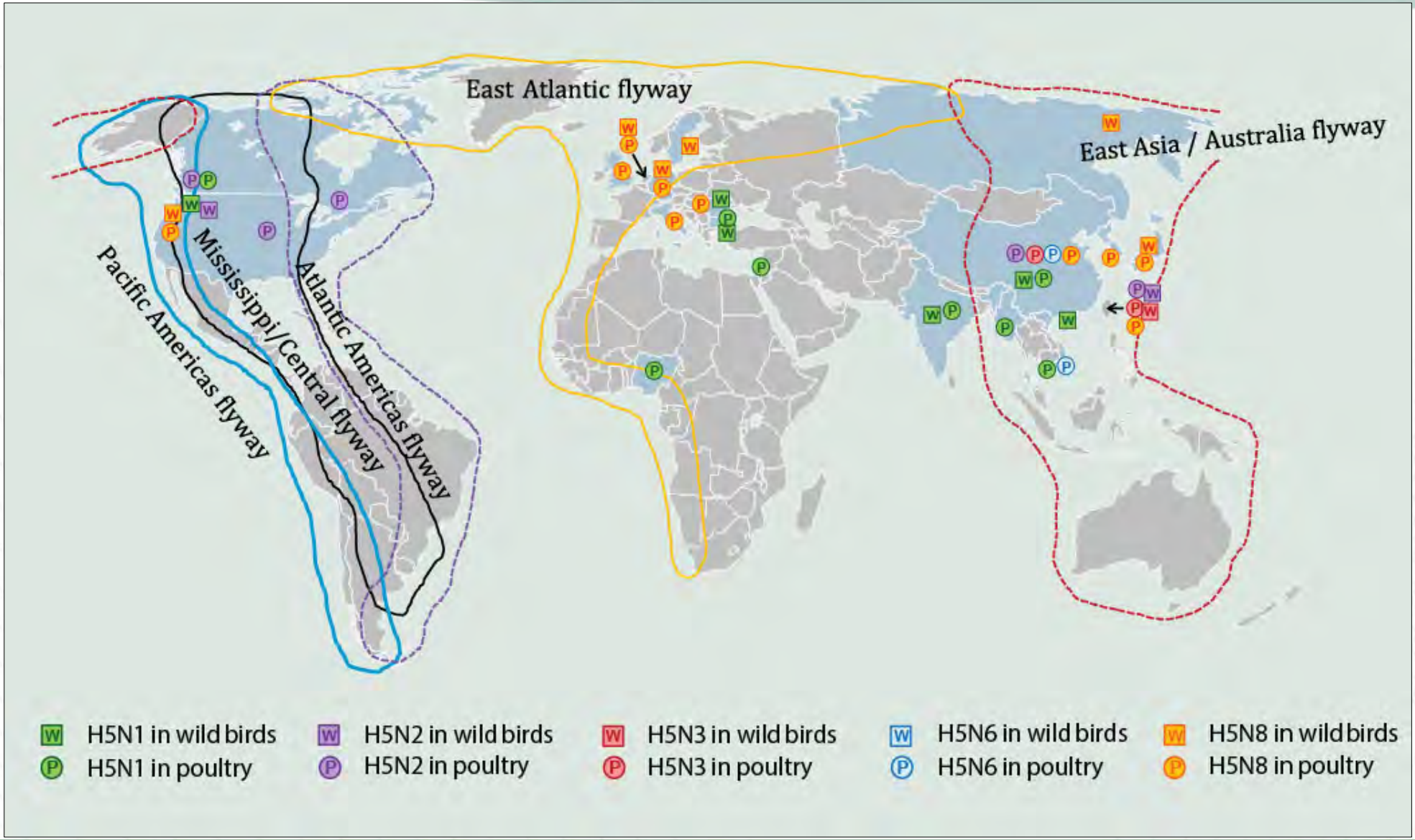
HPAI vs. LPAI Viruses

- LPAI
  - Localized disease
    - Respiratory, GI tracts
  - Enzymes cleave HA in limited locations
- HPAI
  - Systemic disease
    - Severe damage; death
  - Enzymes cleave HA throughout body



Recent Introduction of HPAI Viruses into the United States

HPAI in the United States, 2014



HPAI in the United States, 2014

- H5N8 first Eurasian lineage virus in North America
- Reassorted in wild birds with North American AI viruses
- Created H5N2 and H5N1 reassortants
- H5 gene distantly related to Asian lineage H5N1 HPAI

HPAI in the United States, 2015

- Reassortant H5N2 HPAI spread to upper Midwest via migratory birds
  - Adapted to commercial poultry and spread rapidly
  - 50 million laying hens, turkeys, pullets
- Costs to Iowa
  - 31.5 million poultry depopulated or died
- USDA spent >$900 million
  - Indemnity, disposal, C&D, preparation

HPAI in the United States, 2016

- Increased mortality in one turkey flock in Indiana—H7N8 HPAI
- H7N8 LPAI on eight farms
- LPAI mutation to HPAI on a single farm
- 258,000 turkeys depopulated
- 156,000 layer chickens depopulated

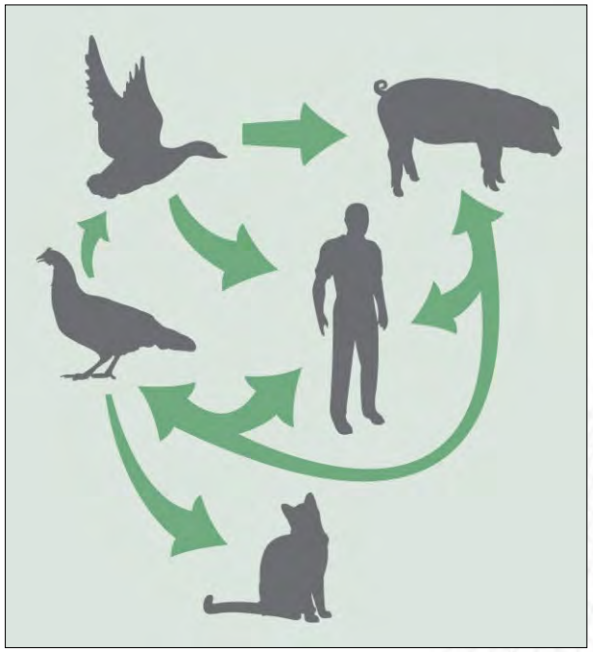
Reportable Avian Influenza and International Trade

- All avian influenza in poultry reportable to OIE
  - HPAI
  - All H5/H7
- U.S. poultry free of avian influenza
  - Vaccination not allowed for avian influenza

Public Health Impact: AI Viruses

Public Health Impact

- Indirect impacts
  - Reduce food supply
  - Change food costs
- Some AI viruses are zoonotic
  - Conjunctivitis, mild influenza-like illness
  - H5N1 HPAI serious, fatal



H5N1 Infections

- H5N1 outbreak—Hong Kong, 1997
  - Severe infections acquired from poultry
- H5N1 in poultry—Asia, 2003
  - Only HPAI virus known to circulate among migratory waterfowl
- 859 human cases resulting from direct contact with poultry; 53% fatal
- Asian lineage H5N1 wide host range
  - Poultry, humans, felines, dogs, stone martens, palm civets, wild birds
- Pandemic if H5N1 adapt to humans

H7 Infections

- Mild H7N7 influenza—Netherlands, 2003
  - 89 confirmed, 258 suspect human infections
- LPAI Asian Lineage H7N9—China, 2013–present
  - Severe respiratory disease among humans
  - Cases: 1557 Deaths: 605
  - Limited person-to-person transmission in China

Public Health: AI Prevention

- Generate new viruses
  - Other species: Humans
- Humans with seasonal influenza avoid contact with AI-infected poultry
  - Prevent cross-infection or combined infection
- Annual flu shots
  - Poultry veterinarians, workers, first responders

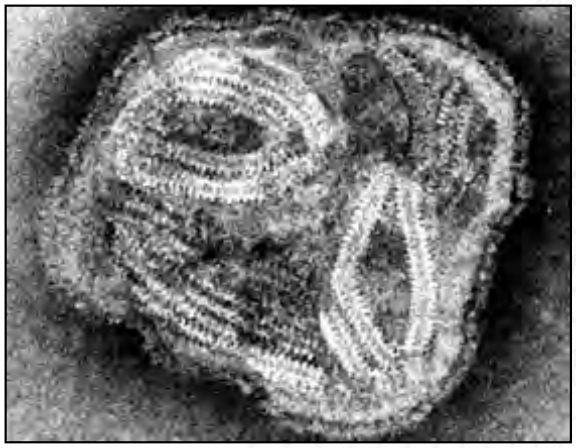
Consumer Reaction

- Loss of confidence in safety of food supply
- Most AI viruses not a human hazard
- Consumers may equate *any* AI virus with highly pathogenic zoonotic viruses
  - Lost income from decreased egg and meat purchases

Newcastle Disease

Newcastle Disease (ND)

- Most significant global poultry disease
- Exotic Newcastle disease
- Avian paramyxovirus-1 (APMV-1)
  - Virulent Newcastle disease virus (vNDV)
  - Family *Paramyxovirus*
- Range of clinical signs
  - Inapparent to virulent
- Transmission from poultry, psittacines, wild birds

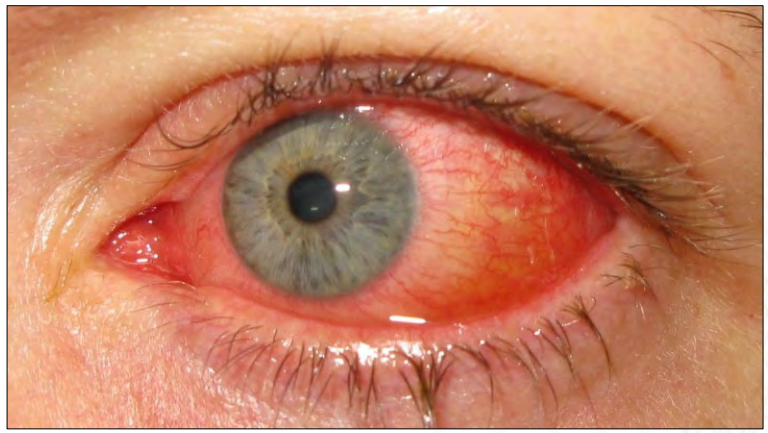


History of ND

- First reported outbreaks
  - England, Indonesia, 1926
- Outbreaks resembled AI
- Several panzootics
  - Spread slowly at first, then rapidly
- Pneumoencephalitis outbreak—California, 1930s
  - Milder form of APMV-1
  - Death rate below 15%

Newcastle Disease: Zoonotic Impact

- Conjunctivitis
  - Exposed to large quantity
  - Not life-threatening
- Immunocompromised may experience severe pneumonia
  - Fatal pneumonia in stem cell transplant patient



Diversity of APMV-1

	Lentogenic	Mesogenic	Velogenic	
			Neurotropic	Viscerotropic
US Occurrence	Common	Uncommon	Absent	
Virulence	Subclinical	Intermediate: Occasional neurologic	Serious disease among poultry	
Signs	Mild respiratory disease, decreased egg production & quality, weight loss		Death without clinical signs possible	
Mortality	Negligible	Low	High	
Trade	No effect		Shut down	

International Definition (OIE)

- Infection with APMV-1 virus meets one of these criteria
  - Intracerebral pathogenicity index in day-old chicks of 0.7 or greater **OR**
  - Contains characteristic pattern of amino acids along fusion protein
- United States considered free of vNDV

Newcastle Disease in the United States

- 1950, California
  - Chukars, pheasants from Hong Kong
  - 5 premises
- 1971, California
  - South American parrots
  - 1,300+ poultry premises
  - 12 million birds, $56 million
- 2002, CA, NV, AZ, TX, NM
  - Illegal cockfighting
  - 2,000+ premises
  - 4 million birds, $160 million
  - Trade restrictions cost $395 million

Newcastle Disease: Economic Impact

- Free of ND
  - Vaccination, biosecurity, surveillance
- Endemic ND
  - Limits commercial poultry industry, trade
  - Ongoing vaccination
- Continued losses impact those reliant on poultry
  - Meat, eggs important source of protein

Economic Impacts of AI and ND Eradication

Losses Incurred from Eradicating AI and ND	
Direct	**Indirect**
Examining and diagnosing cases	Lost international trade
Depopulating and disposing of carcasses	Increased food costs
Paying owners/producers for depopulated animals (paying indemnity)	Other losses
Cleaning and disinfecting affected premises	
Continued surveillance to prove freedom from disease	

Recognizing HPAI and ND

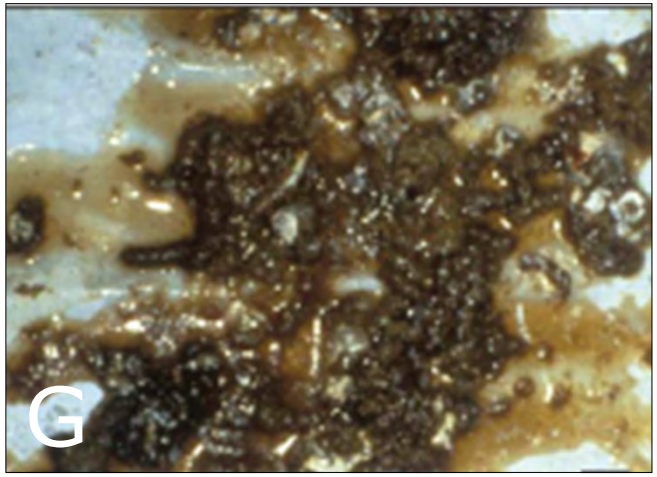
Recognizing HPAI and ND

- **Clinically indistinguishable**
- Nonspecific clinical signs
  - Vary with outbreak, flock
- No signs pathognomonic

HPAI and ND Clinical Signs

HPAI and ND Clinical Signs


F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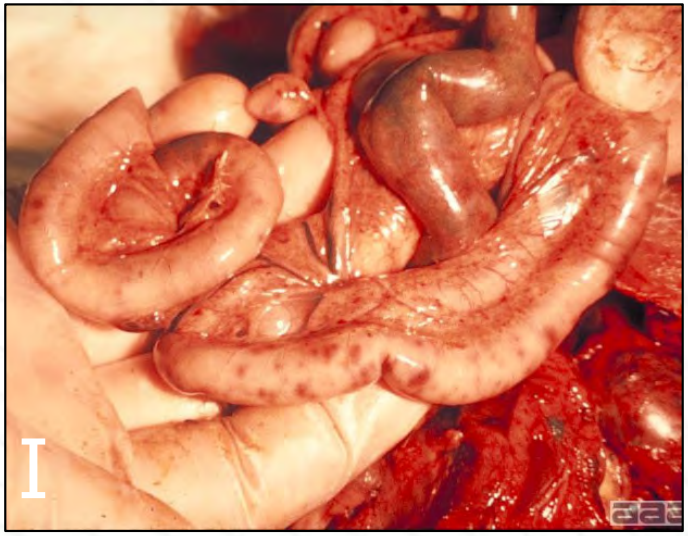
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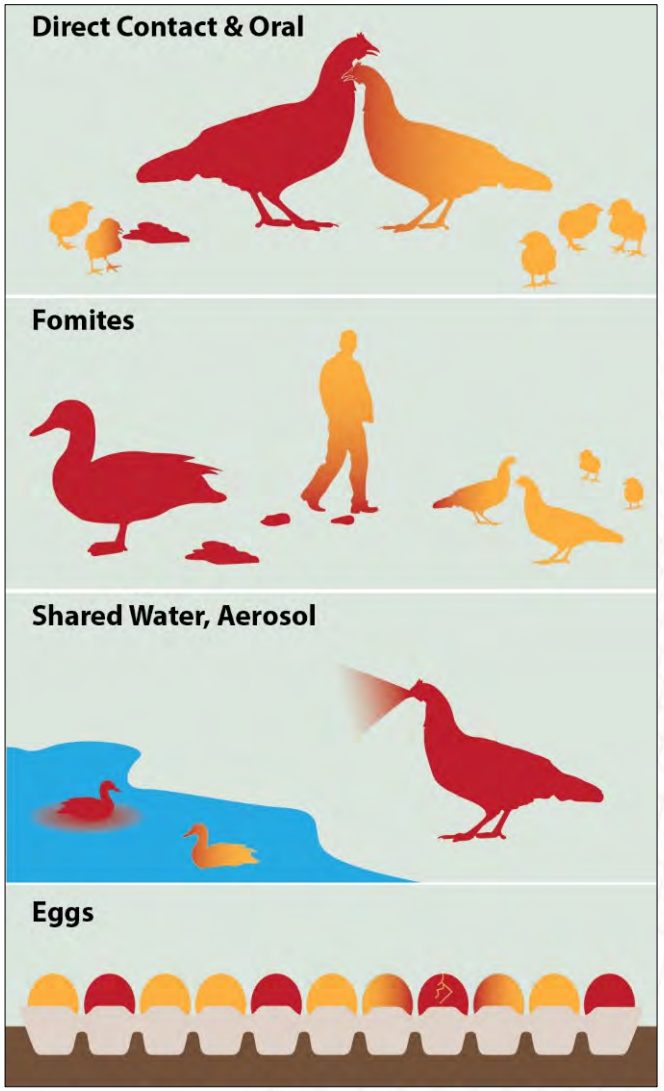
HPAI and ND Clinical Signs

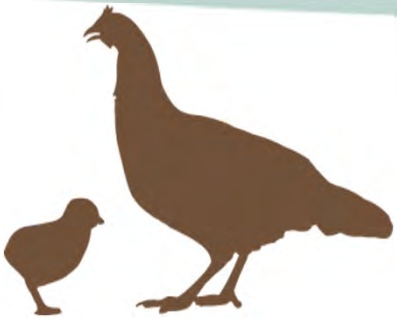
Videos were provided by Dr. Shafqat Fatima Rehmani of the Quality Operations Laboratory at the University of Veterinary and Animal Science in the University of Lahore (UVAS), Pakistan and were part of joint UVAS- Southeast Poultry Research Laboratory research project funded by the US Department of State and entitled Molecular characterization of Newcastle disease virus and development of approaches to vaccination

Direct and Indirect Transmission

- Oral, aerosol, fomites, vectors
- Eggs (infected embryos)
  - vNDV, embryo usually dies
    - Low virus dose might survive and hatch
  - HPAI probably die
    - Cracked eggs may transmit virus to other chicks in incubator



Differential Diagnoses



Poultry

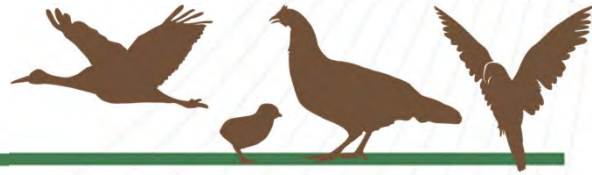
- Fowl cholera
- Fowl pox
- Infectious coryza
- Infectious laryngotracheitis (ILT)
- Mycoplasmosis
- Infectious bronchitis
- LPAI
- Disease of management

Psittacines

- Aspergillosis
- Pacheco's disease

All Avian Species

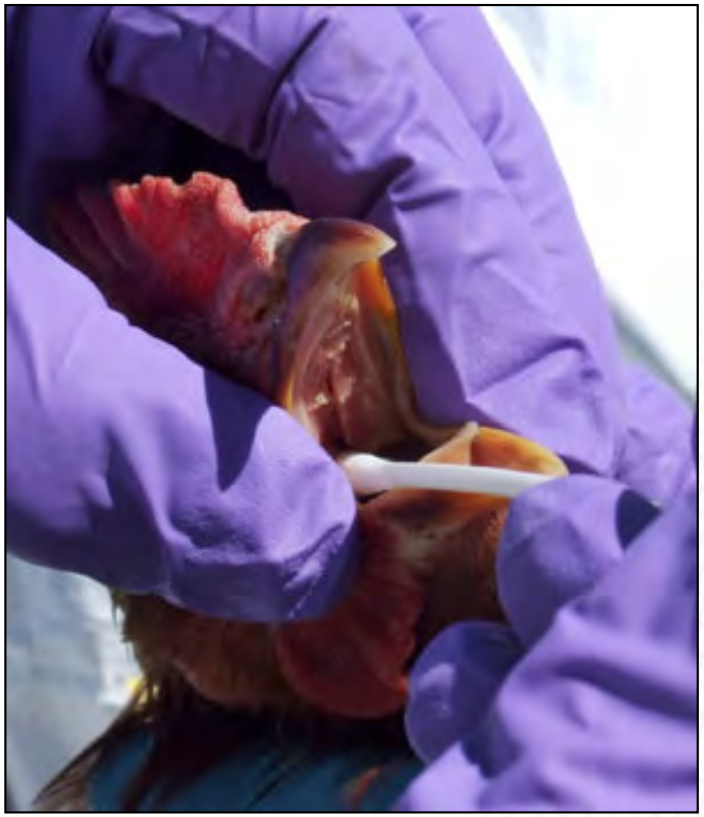
- Chlamydiosis
- Calcium deficiency
- Encephalomalacia
- Severe parasitism
- Salmonellosis
- Toxicosis



Surveillance and Reporting

Surveillance for AI

- Field detection
- Routine surveillance
- Import of birds
- Interstate shipment
- USDA LPAI surveillance plans
  - National Poultry Improvement Plan
  - Live Bird Marketing System



NPIP Avian Influenza Clean

- Requirements based on birds, type of production unit
- Avian Influenza Clean
  - No AI-seropositive birds found in a flock
- H5/H7 AI Clean
  - Turkey breeding flocks, hobbyist and exhibition waterfowl, exhibition poultry, game bird breeding flocks, and meat-type waterfowl breeding flocks

Live Bird Marketing System (LBMS) H5/H7 AI Program

State participation

- Licensing/registration requirements
- Bird testing and record keeping
- Sanitation and biosecurity
- Surveillance and inspections
- Education and outreach

Positive LBM

- Mandatory closure
- Depopulation
- Cleaning and disinfection
- Environmental testing
- Inspection to reopen
- Subject to more frequent surveillance and inspection

Passive Surveillance

- All laboratories that perform diagnostic procedures on poultry must examine all submitted cases of:
  - Respiratory disease
  - Unexplained egg production drops
  - Unexplained severe mortality
- Must use approved serological tests, antigen detection tests

Additional Surveillance

- Backyard poultry
  - Owners encouraged to report illness in flock
  - State may perform
    - Routine surveillance
    - Free post-mortem examinations
    - Free testing for AI
- Wild birds

Surveillance for ND

- Tested upon import
  - Poultry, pet birds, ratites, zoo birds
- Passive surveillance
- Sampling of sick/dead birds at exhibitions
- Commercial samples
  - Swabs, dead bird pickup at no or reduced cost

Reporting Clinically Evident AI or ND

- **IMMEDIATELY** contact SAHO and AD
- Do **NOT** attempt diagnosis
  - FADD will obtain and submit samples to authorized lab
- H5 and H7 LPAI reportable in all States

Indemnity Benefits for Participation

- USDA provides 100% indemnity to participating flocks in States that:
  - Participate in passive surveillance
  - Have APHIS approved Initial State Response and Containment Plan
- 25% indemnity provided for commercial facilities that do not participate in active surveillance
- Non-commercial facilities in participating States are 100% indemnified

Scenario

Scenario

- Poultry veterinarian
- Clients range from large, modern egg production facilities to small broiler farms
  - Varied biosecurity
  - Large migratory flyway

Inspecting a Drop in Production

- Large egg-production facility
- Caged birds
- Excellent biosecurity
- Participation in several aspects of the NPIP program
- Drop in egg production, decreased feed consumption
- Two houses, 38,000 birds in each house, aged 30 weeks

Inspecting a Drop in Production

- 100 dead birds in one house
- Feed consumption down 10%
- Egg production 94–95% of normal
- Respiratory signs, watery stools
- Birds in second house appear normal

Walk-Through

- Lethargic, moribund
- Cyanotic wattles, combs
- Subcutaneous hemorrhages
- 300+ dead birds today
- Affected birds at front
  - Birds at back of barn seem healthy
  - Producer concerned about poison

Report Suspicions to SAHO, AD

- Describe history and clinical signs
- Await contact by an FADD
  - Collect and submit samples to test for AI and ND at no cost
- May be instructed to:
  - Remain on the farm, or
  - Follow strict biosecurity exit protocols; go directly home; avoid animal contact
- Contact veterinary clinic

Report Suspicions to SAHO, AD

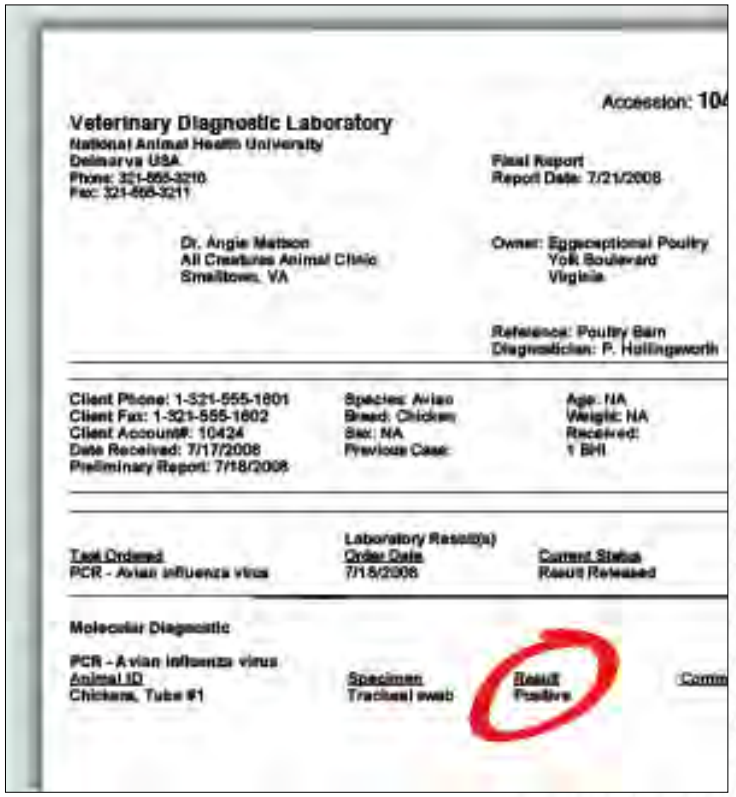
- Farm must act as if AI or ND is present and take action
  - Stop movement of animals, people, vehicles to/from property
  - Await biosecurity instructions
- Seek regulatory official advice

Biosecurity

- Single entrance/exit with sanitizing station
- Clean and contaminated areas of farm
- Minimize movement on/off farm
  - Essential farm workers, family only
  - Special arrangements for supply delivery

Outcome

- H5N2 HPAI positive
- Investigation under guidance of FADDs, eradication teams
  - Responsible until outbreak ends
  - Until surveillance determines no infected birds remain in United States



Accession: 10

Veterinary Diagnostic Laboratory
National Animal Health University
Delmarva USA
Phone: 321-555-3210
Fax: 321-555-3211

Final Report
Report Date: 7/21/2008

Dr. Angie Matson
All Creatures Animal Clinic
Smalltown, VA

Owner: Eggceptional Poultry
York Boulevard
Virginia

Reference: Poultry Barn
Diagnostician: P. Hollingsworth

Client Phone: 1-321-555-1801
Client Fax: 1-321-555-1602
Client Account#: 10424
Date Received: 7/17/2008
Preliminary Report: 7/18/2008

Species: Avian
Breed: Chicken
Sex: NA
Previous Case:

Age: NA
Weight: NA
Received:
1 BHI

Laboratory Result(s)

Test Ordered	Order Date	Current Status
PCR - Avian influenza virus	7/18/2008	Result Released

Molecular Diagnostic

PCR - Avian influenza virus			
Animal ID	Specimen	Result	Comm
Chickens, Tube #1	Tracheal swab	Positive	

Investigation Steps

- Quarantine
- Hold orders
- Depopulation of infected and exposed poultry
  - Disposal of carcasses, contaminated material
  - Owner compensated

Investigation Steps

- Cleaning and disinfection
  - Premises and remaining equipment
- Epidemiological investigation
  - Origin of infection
  - Trace-in and trace-out flocks
  - Other at-risk flocks
- Testing epidemiologically linked flocks
- Area flock surveillance

Investigation Steps

- Public health investigation
  - Zoonotic potential
  - Serum sampling
- Restocking
  - Time empty depends on extent of infection
- Enhanced and follow-up surveillance
  - Population and environmental sampling

Prevention Practices

- Participate in NPIP AI Clean and/or LBMS programs
- Participate in ND surveillance programs
- Practice biosecurity, review with clients
  - Biosecurity handouts available
  - Live bird markets, flea markets, poultry veterinarians

Summary

- Economic and public health impacts of disease outbreaks
- Clinical signs of avian influenza (AI) and Newcastle disease (ND)
- Concerns associated with H5/H7 LPAI viruses
- Roles of National Poultry Improvement Plan (NPIP) and Live Bird Marketing System (LBMS) programs
- Collection and submission of samples
- Reporting positive results
- Biosecurity measures

Supplemental Training

- This informational presentation has been approved expressly to serve as one unit of supplemental training for participants in USDA's NVAP
- Please ensure you complete, sign, and **retain a certificate** stating that you attended this presentation
- Contact your VS District Office for more details

Acknowledgments

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Previous versions of this module were reviewed by

- **USDA-APHIS-VS:** C. Stephen Roney, DVM, MAM, DACPV, Senior Staff Veterinarian, National Poultry Improvement Plan; Tom Gomez, DVM, MS, National Center for Animal Health Emergency Management, Liaison to the CDC; Angela M. Pelzel, DVM, Regional Poultry Epidemiologist; Patricia Klein, MS, VMD, DACPV, DACVPM, Senior Staff Veterinarian, Avian Disease Specialist
- Scott Gustin, DVM, MAM, DACPV

Questions?

The NVAP website can be found by typing "NVAP" into your search engine.

To Report a Suspected FAD

866-536-7593

EQUINE PROCEEDINGS

June 5–7, 2022

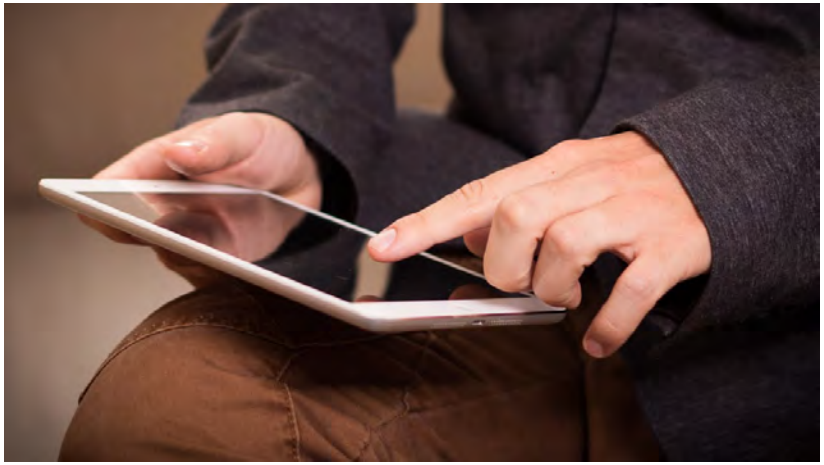
Module 34 – Veterinary Export Health Certificate System

USDA–APHIS Veterinary Medical Officers

USDA APHIS
Veterinary Services
National Veterinary
Accreditation Program

Module 34:
Veterinary Export Health Certification System

Sign in with an iPad or paper to get credit.



- Retain certificate of completion
- 2-step process to renew accreditation

If you don’t know your six digit NAN, pass the iPad, see us after session.

- Tap each field
- Select Submit
- Review information for accuracy
- Select Confirm
- Pass iPad

Your name must match what is in our records.

- If “No Match Found” displays, re-enter
- Pass tablet and see us after

All modules are available on our Website.

- Type “NVAP” into your search engine e.g. Bing, Google, Yahoo.

Intended to familiarize accredited veterinarians with animal health regulatory concepts and activities.

- Does not supersede regulations
- Most up-to-date regulations and standards
  - Code of Federal Regulations
  - Area Veterinarian in Charge (AVIC)

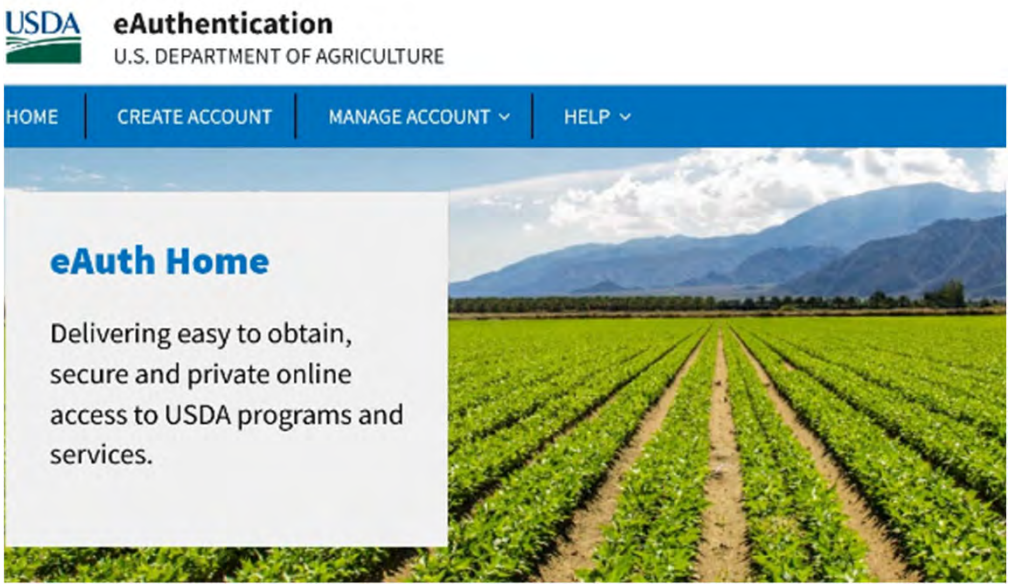
USDA **United States Department of Agriculture**
Animal and Plant Health Inspection Service

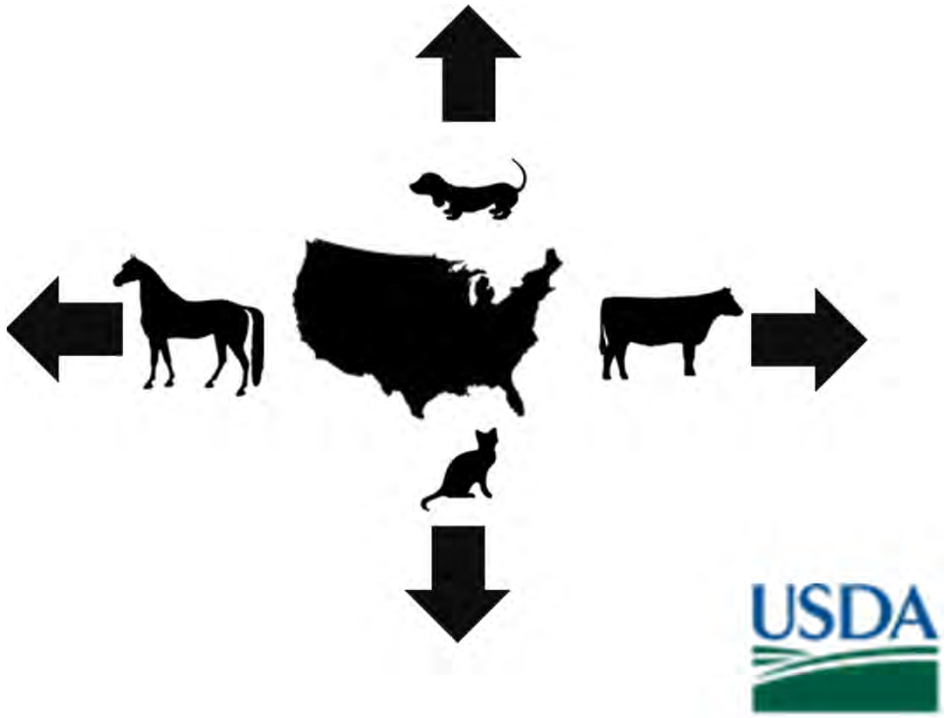
After completion, you will be able to:

- describe VEHCS and give an overview of its functionality;
- determine the extent to which VEHCS can be used for a health certificate's issuance and endorsement;
- know how to access VEHCS;
- explain how to submit health certificates to APHIS using VEHCS; and
- know the location of VEHCS user resources, including user Quick Reference Guides, and help desk contacts.

Describe VEHCS and give an overview of its functionality.

VEHCS is an online, secure electronic system for creation, issuance, submission, and endorsement of international certificates for export of live animals.



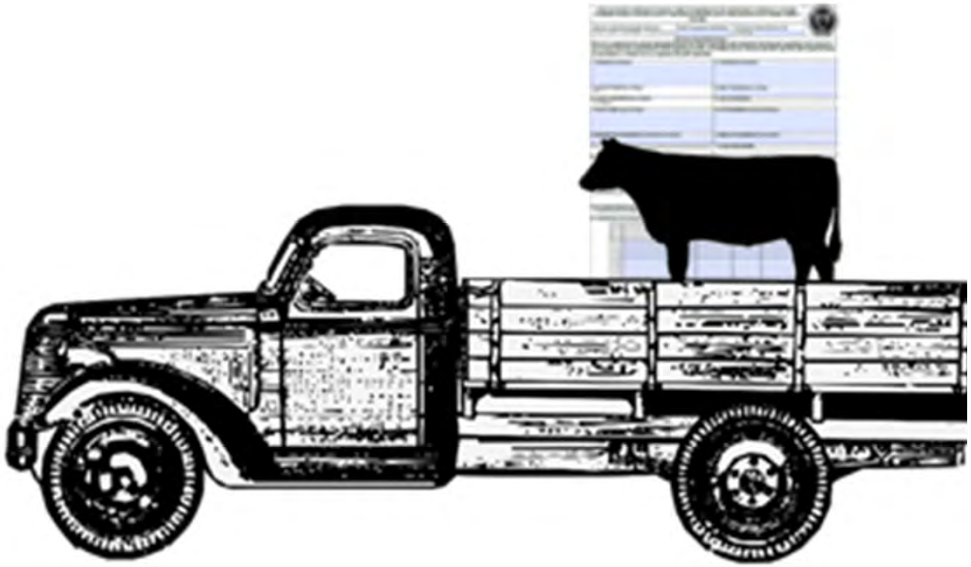


You can submit health certificates and supporting documentation electronically for review and endorsement.

Describe the extent to which VEHCS can be used for a health certificate's issuance and endorsement.

IRegs web page and Pet Travel Website will state if VEHCS can or must be used to for endorsement of a health certificate.

NEVER Acceptable



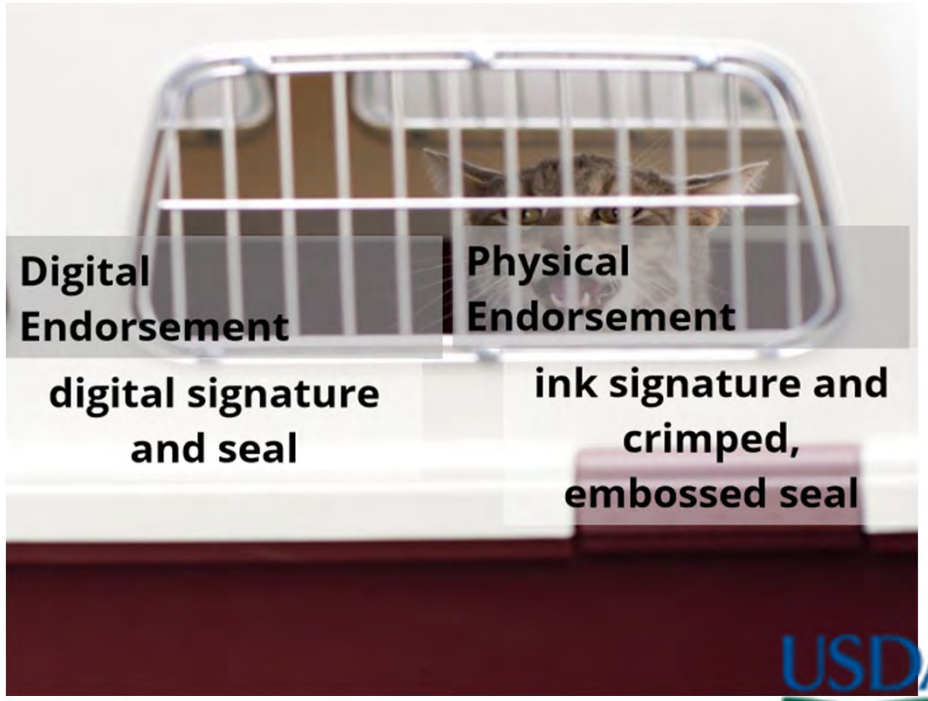
Acceptance of electronic signatures only applies to health certificates issued in VEHCS

AV cannot electronically sign a paper certificate or email or fax it to VS.

Use VEHCS instead of paper

USDA

Check PTWS and IRegs to determine what type of health certificate destination country accepts.



Colored banners explains extent to which country permits use of electronic signatures in VEHCS.

	Red	Orange	Green	Yellow	Purple
Accredited Veterinarian Signature	Original Signature	Electronic Signature	Electronic Signature	Electronic Signature for SOME commodities Only refer to IREGS or PTW)	Electronic Signature
APHIS VMO Signature	Original signature and physically embossed with raised seal	Original signature and physically embossed with raised seal	Digital signature and digital seal (not embossed)	Digital signature and digital seal (not embossed) for SOME commodities only (refer to IREGS or PTW)	Digital signature and digital seal (not embossed) for SOME commodities only (refer to IREGS or PTW)

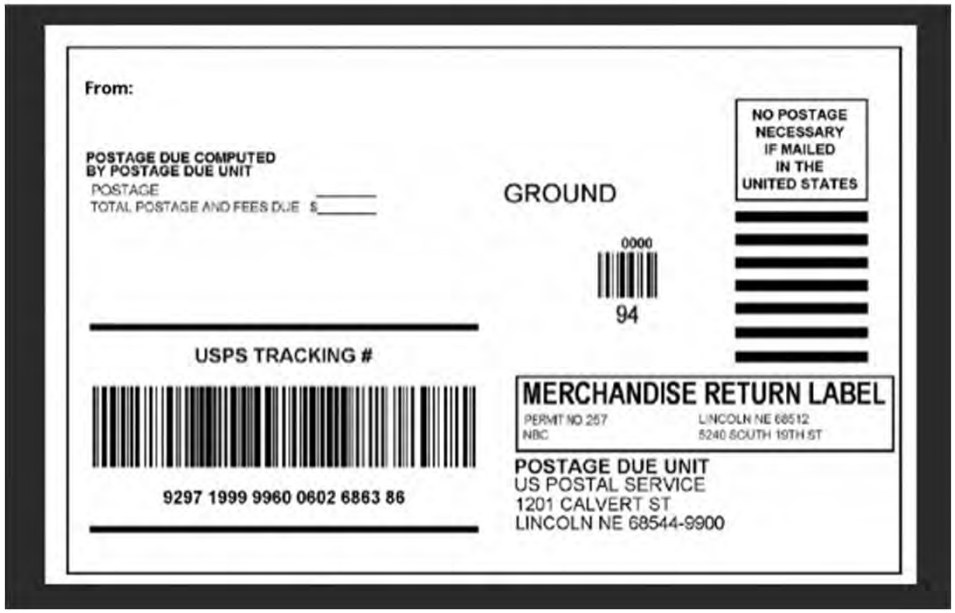
USDA

Once health certificate is endorsed, status changes to “Completed”

Print endorsed health certificate, give to client to accompany animal to destination country

Certain destination countries don’t accept VS’s digital endorsement, instead require wet ink signature and physical embossment.

A Return Shipping Label

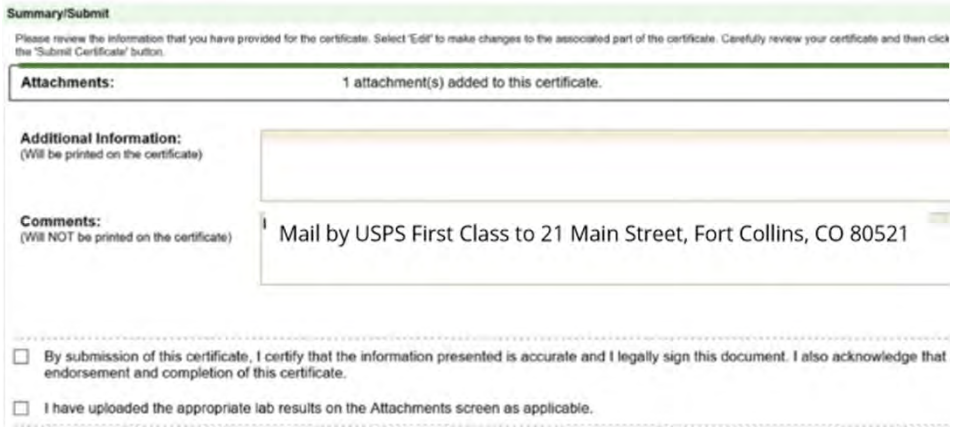


Upload as an attachment

Or you may inquire with your VS Endorsement Office in advance of submission, if possible, to pick up after endorsement.

USPS.COM®

First-Class Mail



Summary/Submit

Please review the information that you have provided for the certificate. Select 'Edit' to make changes to the associated part of the certificate. Carefully review your certificate and then click the 'Submit Certificate' button.

Attachments: 1 attachment(s) added to this certificate.

Additional Information:
(Will be printed on the certificate)

Comments:
(Will NOT be printed on the certificate)

Mail by USPS First Class to 21 Main Street, Fort Collins, CO 80521

☐ By submission of this certificate, I certify that the information presented is accurate and I legally sign this document. I also acknowledge that endorsement and completion of this certificate.

☐ I have uploaded the appropriate lab results on the Attachments screen as applicable.

USDA

Payment of user fee must be provided before your VS Endorsement Office can endorse health certificate.

The Food, Agriculture, Conservation, and Trade (FACT) Act or **1990 Farm Bill.**

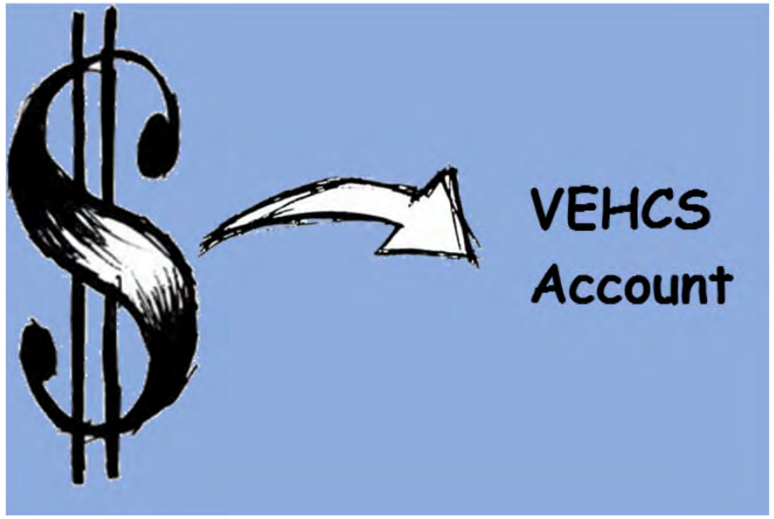
endorsement of international export health certificates

USDA

It is your responsibility to ensure endorsement fee is available when you submit certificate in VEHCS.

exceptions include

There are two options for providing payment in VEHCS.



Or

USDA APHIS User Fee Credit Account

Describe how to access the system.

- Step one: Create an eAuth. account
  - Do not share your password or you may be subject to accreditation violations.
- Step two: Visit VEHCS website and log in.

USDA

Option 1

My business (non-USDA) does not have a VEHCS account,
and I will be the first user and administrator for my business in VEHCS.

Create Business Organization

Option 2

I'm joining an existing business or USDA organization and
my organization administrator has provided me with a Unique PIN.

Unique PIN: Join

It is your responsibility to maintain expiration dates in VEHCS.

- VEHCS will not permit you to issue a health certificate if your license or accreditation is expired.

VEHCS has three types of health certificates

How will you know which certificate type to use? Once you enter destination country, commodity and intended use VEHCS will…

1. Walk you through steps and you select certification statements (VEHCS Defined).

2. VEHCS instructs you to enter required certification statements (VEHCS Universal).

3. VEHCS instructs you to upload filled PDF health certificate (AV PDF Upload).

How to submit health certificates to APHIS using VEHCS.

Complete every field labeled with red asterisk

- completely and accurately fill in every field labeled with a red asterisk
- If not done your certificate could be returned which may result in delay of endorsement

- **Saving a certificate as a work in progress**
- Work can be saved before it is submitted by selecting “save as work in progress” button at the top right or bottom of the screen.

- **Creating a template**
- Information from the General and Export and Shipping screens can be saved and re-used.

Tracking number

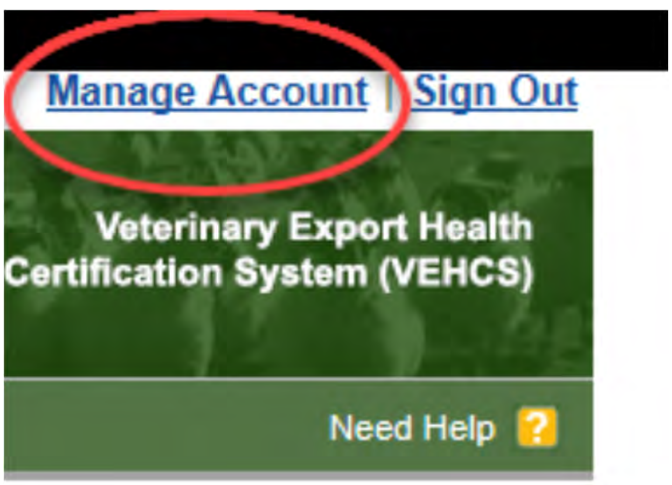
- Unique number assigned to certificate when it is created and saved
- Used for tracking from creation through the endorsement and completion

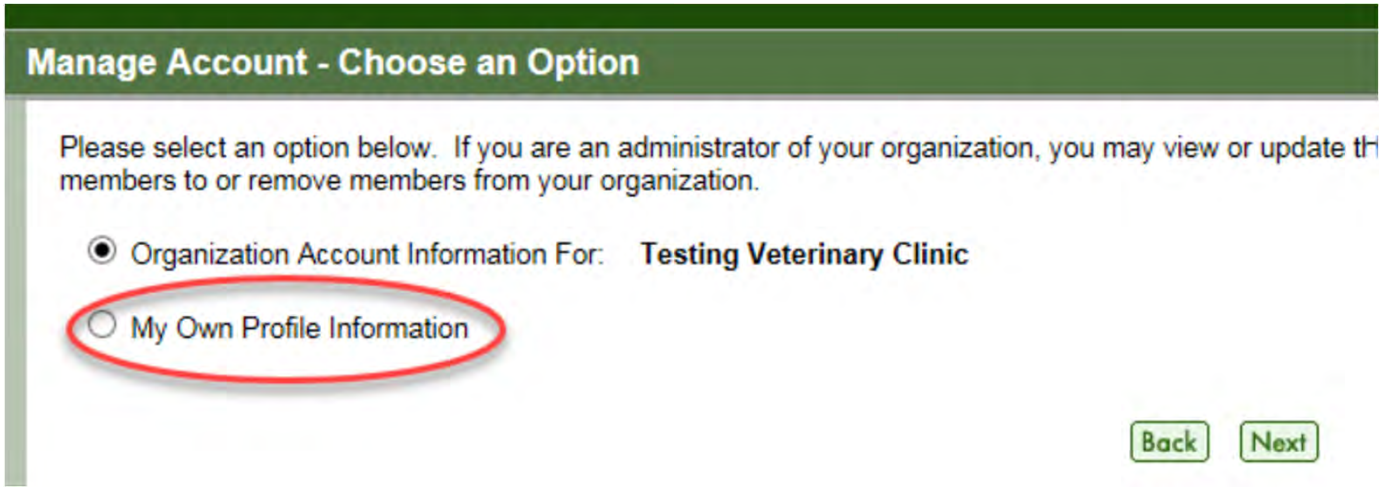
1. Log on to VEHCS
2. Select view certificate from left side of screen
3. Search for certificate to view its status

Certificates can be returned by Endorsement Office if something is done incorrectly, or not enough information was supplied.

- If status is “returned” select “comments” to view comments from your VS Endorsement Office
- Select “back”
- Select “view/edit” to make changes requested by VS Endorsement Office
- Re-submit certificate

- Maintain your license and accreditation information in your profile.
- Check here if VEHCS does not have your name available as accredited veterinarian for health certificate you are issuing.



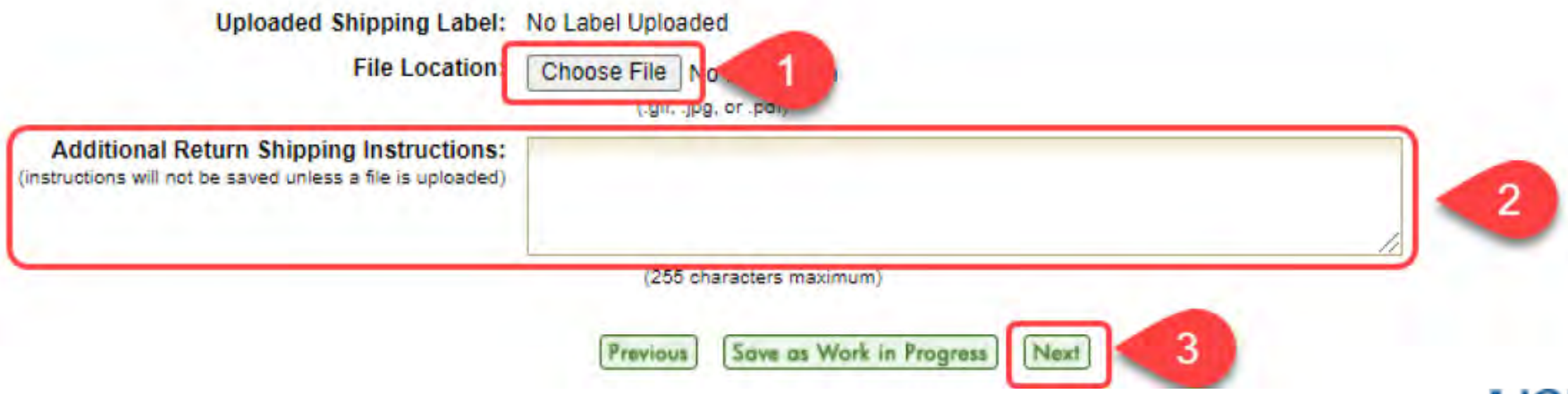


Payment must be provided before the health certificate can be endorsed.

- Deposit money into your VEHCS account under “Financial Management”
- Provide your USDA APHIS User Fee Credit Account information

Upload a pre-paid shipping label as an attachment if the certificate cannot be digitally endorsed.

This will allow the endorsed hardcopy of the health certificate to be sent to you or your client so it can accompany the animal(s) at the time of travel.



The location of VEHCS user resources

For more information on how to use VEHCS, including detailed explanations of the information presented within this Module, please visit the VEHCS Help Page

USDA

References for Accredited Veterinarians on How to Use VEHCS

- Why should USDA Accredited Veterinarians use VEHCS?
- Video: Overview of the advantages to using VEHCS, including a refresher of your role in issuing health certificates
- Video: How to correctly complete a health certificate in VEHCS
- Country Acceptance List for VEHCS
- Step-by-step guide to using VEHCS (updated regularly!)

Step-by-step tutorial of how to use VEHCS and VEHCS Quick Reference Guides

VEHCS Quick Reference Guides

These guides are provided in Adobe Reader PDF format.

VEHCS Quick Reference Guides
Simple guides, broken down by topic and user, to help you quickly understand the basics of using VEHCS.

For Accredited Veterinarians, AV Support Staff, and Exporters

- Frequently Asked Questions (FAQ)
  December 2019
- Accessing VEHCS for the First Time
  September 2019
- How to Create a Defined Health Certificate
  July 2020
- Manage Account for AV Users

For VS Personnel and Offices

- Internal VS Upload PDF Certificate
  January 2018
- Manage Account and Access VEHCS for VS Users
  January 2018
- Processing a Health Certificate
  August 2018
- VMO Processing an AV Uploaded Health Certificate

Technical support is also available for your eAuthentication account or VEHCS.

eAuthentication Support	VEHCS/PCIT Help Desk	APHIS-VS Endorsement Office
• User ID and password assistance • Technical support related to login. 1-866-794-2827 aehd@usda.gov	• System IT support (technical glitches with VEHCS itself) • Organization administration issues 1-866-HLP-PCIT (1-866-457-7248) pcithelpdesk@usda.gov	• Export certificate issuance • Interpretation of regulations and requirements • Saving and uploading PDF files • Certificate status information • Payment (user fees) • Creating & uploading shipping labels • Accreditation questions Contact Your APHIS VS Endorsement Office

After completion, you will be able to:

- describe VEHCS and give an overview of its functionality;
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If you suspect a FAD IMMEDIATELY

contact your

VS Area Veterinarian in Charge (AVIC)

at 1-866-536-7593

and

your State Animal Health Official (SAHO)

USDA

Notes