The Armed Forces Institute of Pathology
Department of Veterinary Pathology
WEDNESDAY SLIDE CONFERENCE
2001-2002

CONFERENCE 17
17 February 2002

Conference Moderator: LTC Denzil Frost
Chief, Research Branch
The Armed Forces Institute of Pathology
Washington, DC 20306-6000

CASE I – 2443398 (AFIP 2788618)

Signalment: 10-day-old, male, Standardbred, horse (*Equus caballus*), equine

History: This foal was born with contracted tendons and placed in corrective splints. Serum IgG levels indicated partial failure of passive transfer, so plasma was administered. The colt developed respiratory signs, including rapid labored breathing, purulent nasal discharge, coughs, fever, and died in spite of antibacterial therapy.

Gross Pathology: Lungs were diffusely consolidated with disseminated pale foci throughout the parenchyma on cut section.

Laboratory Results:
Lung aerobic culture: *Streptococcus zooepidemicus* >1,000 cfu
Klebsiella pneumoniae >1,000 cfu

Lung virus isolation: Negative for Equine Rhinopneumonitis and Equine Influenza viruses.

Contributor's Morphologic Diagnosis: Lung: acute, severe, diffuse fibrino-suppurative bronchopneumonia, with mixed bacterial colonization.

Etiology: *Streptococcus zooepidemicus* and *Klebsiella pneumoniae*

Contributor's Comment: This severe fulminant pneumonia is generally seen with *Streptococcus zooepidemicus* following a predisposing viral infection. In this case the predisposing incident appeared to be partial failure of passive transfer. Both *S. zooepidemicus* and *Klebsiella pneumoniae* are considered important respiratory pathogens in horses.

AFIP Diagnosis: Lung: Bronchopneumonia, necrosuppurative, subacute, diffuse, severe with septal and pleural edema, multifocal plant material, and myriad bacteria, Standardbred, equine.

Conference Comment: Bronchopneumonia is the term given to pulmonary inflammation that originates at the bronchoalveolar junction. Bacteria, via aerogenous routes, are the most common cause of significant bronchopneumonia. Infection occurs when overwhelming numbers of organisms reach the lower airways often when host defenses are suppressed or impaired (e.g. concurrent viral infection, acquired or congenital immunodeficiencies, or stress). Factors that lead to susceptibility of the bronchiolar-alveolar junction include it being the major site of deposition for particles .5-3 um in diameter, lack of a protective mucous blanket or an effective alveolar macrophage system, and narrow lumina of parent bronchioles that can become easily plugged. The characteristic cranioventral distribution is thought to be due to short abruptly branching airways, gravitational sedimentation of particles, and regional differences in collateral ventilation.

Streptococcus zooepidemicus is gram positive; frequently causes pneumonia, lymphadenitis, and sepsis; and contains numerous virulence factors. Virulence factors associated with streptococci include nonantigenic, antiphagocytic capsules; cell wall M proteins that interfere with complement opsonization; and numerous exotoxins (e.g. streptolysins, hyaluronidase, DNAse). In addition to *Klebsiella pneumoniae* (gram negative, heavily encapsulated bacillus), the differential diagnosis discussed in conference included *Streptococcus equi* ("bastard strangles") and *Rhodococcus equi*. *R. equi* is a gram positive, weakly acid fast bacteria that passes through a cycle from coccal to bacillary form and is associated with pyogranulomatous pneumonia.

Contributor: Michigan State University, Animal Health Diagnostic Laboratory, Lansing, MI 48909

References: 1. Biberstein E, Hirsh D: Corynebacteria. *In*: Veterinary Microbiology, eds. Hirsh D, Zee Y, pp. 132-134. Blackwell Science, Malden, MA, 1999
2. Biberstein E, Hirsh D: Streptococci. *In*: Veterinary Microbiology, eds. Hirsh D, Zee Y, pp. 120-126. Blackwell Science, Malden, MA, 1999
3. Dungworth D: The respiratory system. *In*: Pathology of Domestic Animals, eds. Jubb K, Kennedy P, Palmer N, 4th ed., vol. 2, pp. 591-597. Academic Press, San Diego, CA, 1993
4. Rooney J, Robertson J: Respiratory system *In*: Equine Pathology, 1st ed., pp. 24, 40-41, Iowa State University Press, Ames, Iowa, 1996

CASE II – MSU2 (AFIP 2801925)

Signalment: Three-year-old, mixed gender, pen-reared quail, *Colinus virginianus*

History: Found drooped over with feathers ruffled and wobbling; next day are dead.

Gross Pathology: The esophagus had areas of ulceration covered by large accumulations of necrotic debris. Associated with this lesion were numerous nematodes. Esophageal sections from all three birds contained similar lesions.

Laboratory Results: No significant pathogens were isolated on standard culture.

Contributor's Morphologic Diagnosis: Esophagus, capillariasis, diffuse, subacute, marked

Contributor's Comment: Sections of the quail esophagus or crop, depending upon the slide, exhibit a mild to moderate increase in the thickness of the mucosa due to embedded parasites. The parasite sections are those of a nematode, as they possess a pseudocoelom, a digestive tract and a reproductive tract. The outer cuticle lacks recognizable lateral cords. Underlying muscles are polymyarian coelomyarian, with some sections showing bacillary bands. The digestive tract has uninucleate cells. Depending upon the section, double operculated eggs can be found in the glandular uterus. The operculums are not directly opposite one another, but are at a slight angle. Some sections exhibit portions of the male reproductive tract. The mucosa covering the adult parasites has mild ballooning change. Focal areas have eggs free within the mucosa and there is a marked inflammatory response of heterophils, lymphocytes and macrophages associated with them. Similar inflammatory cells extend into the submucosa where they can be found surrounding submucosal vessels as well as being diffuse within the connective tissue.

AFIP Diagnosis: Esophagus: Esophagitis, proliferative, chronic-active, diffuse, moderate, with intraepithelial adult nematodes and eggs, etiology consistent with *Capillaria* sp., quail (*Colinus virginianus*), avian.

Conference Comment: *Capillaria* sp. belong to the group of nematodes known as aphasmids. The defining feature of aphasmids is the absence of a tiny pair of sensory papillae (phasmids) on their caudal end; a feature which is not readily identifiable on histological examination. Bacillary bands, double operculate eggs,

and the presence of a stichosome are also characteristic of this group. The stichosome, which is deeply basophilic, is composed of stichocytes or esophageal gland cells and surrounds the esophagus.

Additional intraepithelial nematodes discussed in conference include *Trichosomoides crassicauda* in the urothelium of rats, *Gongylonema* sp. in the esophagus of ruminants and nonhuman primates, *Anatrichosoma* sp. in the nasal mucosa of nonhuman primates, and *Eucoleus boehmi* in the nasal mucosa of dogs.

Contributor: Murray State University, Breathitt Veterinary Center, Hopkinsville KY 42241

References: 1. Chitwood M, Lichtenfels J: Identification of Parasitic Metazoa in Tissue Sections, Reprinted from Experimental Parasitology 32:407-519, 1972
2. Gardiner C, Poyton S: An Atlas of Metazoan Parasites in Animal Tissues, pp. 40-43. Armed Forces Institute of Pathology, Washington, DC, 1999
3. Montali R, Migaki G: Comparative Pathology of Zoo Animals. Proceedings of a Symposium at the National Zoological Park, October 24, 1978. Smithsonian Institute Press, Washington, DC, 1980

CASE III – 99-2230 (AFIP 2687173)

Signalment: 4-week-old, male, domestic sheep, breed unspecified, *Ovis aries*

History: This lamb was used acutely at the research facility.

Gross Pathology: The investigative group performed the necropsy, noticed abnormalities in the cranioventral lung fields, and submitted affected tissues to the diagnostic facility for histopathologic evaluation.

Laboratory Results: Microbiology: Heavy growth of *Pasteurella haemolytica*

Contributor's Morphologic Diagnoses: 1. Pneumonia, bronchointerstitial, proliferative and lymphohistiocytic, with proliferation of type II pneumocytes, subacute to chronic/active, multifocal to coalescing, severe, cranioventral lung fields.
2. Bronchiolitis and alveolitis, fibrinosuppurative, with intralesional bacterial rods, subacute to chronic/active, diffuse, severe, cranioventral lung fields.

Contributor's Comment: Consolidated sections of lung from cranioventral fields exhibit features of both proliferation and exudation. Proliferative changes include hyperplasia of bronchiolar epithelium with concomitant loss of cilia, and proliferation of type II pneumocytes. Moderate numbers of lymphocytes, histiocytes and a few plasma cells surround bronchioles and, along with neutrophils, thicken interalveolar septa. Alveoli packed with fibrin, dense streams of fusiform, degenerate leukocytes ("oat cells"), and extracellular colonies of bacterial rods comprise the exudative component of the lesion. Similar material is present in bronchioles. In some sections, there is also perivascular and pleural edema and/or inflammation. The cranioventral distribution, combined with evidence of both proliferation and exudation, is characteristic of chronic enzootic pneumonia of lambs.

Enzootic pneumonia is an epidemiologic term referring to pneumonia that is prevalent in groups of young animals, usually calves, lambs or pigs, kept in close contact. Acute enzootic pneumonia is typically initiated by infection with one or more viruses, and a superimposed bacterial bronchopneumonia may be fatal. In the chronic form, as seen here, mortality is low and the self-limiting, cranioventral consolidations are usually detected only in animals dying from other processes. There still exists some uncertainty regarding the cause(s) of chronic enzootic pneumonia in lambs. It is generally considered to result from the synergistic interactions of *Mycoplasma ovipneumoniae*, *Pasteurella haemolytica* and/or *Bordetella parapertussis* in persistent infection.

Heavy growth of *P. haemolytica* was obtained from aerobic culture of this lesion (*B. parapertussis* was not isolated). Small bacterial rods mingled with the intra-alveolar exudate, alongside the streaming "oat cells" that are classically seen in *Pasteurella* pneumonia. The oat cells represent leukocytes secondarily altered by *Pasteurella* leukotoxin.

Although a commensal in the upper aerodigestive tract, *P. haemolytica* possesses several well-documented virulence factors that aid its opportunistic colonization of the lower respiratory tract when pulmonary defenses are compromised. Cell-associated products include an antiphagocytic polysaccharide capsule, fimbriae that permit adherence, lipopolysaccharide that induces neutrophil influx, outer membrane proteins, proteins involved in iron acquisition, and a periplasmic superoxide dismutase. Extracellular products include O-sialoglycoprotein endopeptidase, neuraminidase, and a potent leukotoxin.

The 104 kDa leukotoxin belongs to a broadly distributed group of RTX (Repeat in Toxin) toxins. The cytotoxicity of RTX toxins depends on a repeat domain consisting of multiple copies of a 9 amino acid consensus sequence shared among a variety of bacterial genera. Like several other RTX, the RTX of *P.*

haemolytica is cytolytic at high concentrations, presumably via pore formation and membrane permeabilization. However, at low concentrations, *P. haemolytic* RTX induces BL3 cells to undergo changes consistent with apoptosis, including cytoplasmic blebbing (zeiosis) and nuclear condensation.

Lesions of chronic enzootic pneumonia are also partially attributed to mycoplasma infection. *M. ovipneumoniae* is the mycoplasma most often isolated from sheep lung, but its role in pathogenesis is unresolved. An immunoperoxidase technique has been used to detect the presence of both intracellular and extracellular *M. ovipneumoniae* (and *P. haemolytica*) in sections of pneumonic lung from lambs, in close association with the presence of lesions. However, there is a lack of information about potential virulence factors such as toxins and adhesion molecules in this organism, although a recent report demonstrated the presence of a polysaccharide capsule that may mediate attachment to cilia. Capsules may also impart antiphagocytic properties and undermine the development of an effective immune response. *M. ovipneumoniae* has also been shown to suppress the phagocytic and cytolytic properties of sheep macrophages *in vitro*, as well as alter the expression of receptors and MHC on their surfaces.

In this case, the marked lymphofollicular cuffing around airways usually described in longstanding mycoplasma infections had not developed. This may reflect the relatively short duration of disease in this lamb, which was only 4 weeks old at the time of euthanasia. Culture of mycoplasma was not attempted.

AFIP Diagnosis: Lung: Pneumonia, neutrophilic and histiocytic, subacute, diffuse, severe, with type II pneumocyte hyperplasia and oat cells, sheep (*Ovis aries*), ovine.

Conference Comment: Since the submission of this case for WSC (1999), the trehalose-negative strain of *Pasteurella haemolytica*, which is most often associated with clinical pneumonia in ruminants, has been reorganized under the new genus *Mannheimia*.

As part of the pathogenesis in bovine *M. haemolytica* infection, recent studies describe increases in acute phase proteins such as serum amyloid A, haptoglobin, and lipopolysaccharide (LPS) binding protein (LBP). LPS-LBP complexes bind CD14 molecules on endothelial cells and leukocytes (primarily macrophages) activating them. In addition to numerous cytokines, activated macrophages express IL-8. IL-8, which is chemotactic for neutrophils, is also expressed by bronchial and bronchiolar epithelial cells. Eventually, newly recruited neutrophils will become the dominant source of IL-8 expression perpetuating the cycle. As the contributor stated, these neutrophils, subsequently altered by leukotoxins, form the characteristic "oat cells".

Additional pulmonary conditions of sheep discussed in conference included ovine progressive pneumonia and ovine pulmonary carcinoma. Ovine progressive pneumonia (Maedi) is caused by ovine lentivirus, a nononcogenic retrovirus. Following a long incubation period within monocytes and macrophages, ovine lentivirus results in a severe interstitial pneumonia. The condition is characterized by numerous lymphocytes and macrophages, fibrosis and smooth muscle hyperplasia, and secondary bacterial infection. Type II pneumocyte hyperplasia is nominal. Ovine pulmonary carcinoma (pulmonary adenomatosis, jaagsiekte) is a slow virus disease caused by an oncogenic retrovirus, jaagsiekte sheep retrovirus (JSRV). Neoplastic cuboidal or columnar cells line airways forming papillary projections and can metastasis to regional lymph nodes. Interstitial inflammation is minimal; concurrent bronchopneumonias are common.

Contributor: University of Washington, Department of Comparative Pathology, Seattle, WA, 98195

References: 1. Angen O, Mutters R, Caugant D, Olsen J, Bisgaard M: Taxonomic relationships of the [Pasteurella] haemolytica complex as evaluated by DNA-DNA hybridizations and 16S rRNA sequencing with proposal of Mannheimia haemolytica gen. nov., comb. nov., Mannheimia granulomatis comb. nov., Mannheimia glucosida sp. nov., Mannheimia ruminalis sp. nov. and Mannheimia varigena sp. nov. Int J Syst Bacteriol **49**:67-86, 1999
2. Brogden K, Lehmkuhl H, Cutlip R: *Pasteurella haemolytica* complicated respiratory infections in sheep and goats. Vet Res **29**:233-254,1998
3. Dungworth D: The respiratory system. *In*: Pathology of Domestic Animals, eds. Jubb K, Kennedy P, Palmer N, 4th ed., vol. 2, pp. 629-691. Academic Press, San Diego, CA, 1993.
4. Haziroglu R, Diker K, Turkarslan J, Gulbahar M: Detection *of Mycoplasma ovipneumoniae* and *Pasteurella haemolytica* antigens by an immunoperoxidase technique in pneumonic ovine lungs. Vet Pathol **33**:74-76, 1996
5. Hodgson J, Brennand S, PorterJ: Effects of interactions between *Pasteurella haemolytica* and *Bordetella parapertussis* on *in vitro* phagocytosis by lung macrophages. Biologicals **24**:325-328, 1996
6. Lopez, A: Respiratory system, thoracic cavity, and pleura. *In*: Thomson's Special Veterinary Pathology, eds. McGavin M, Carlton W, Zachary J, 3rd ed., pp. 125-191. Mosby, St. Louis, MO, 2001
7. Malazdrewich C, Ames T, Abrahamsen M, Maheswaran S: Pulmonary expression of tumor necrosis factor alpha, interleukin-1 beta, and interleukin-8 in the acute phase of bovine pneumonic pasteurellosis. Vet Pathol **38**:297-310, 2001
8. Niang M, Rosenbusch R, Lopez-Virella J, Kaeberle M: Expression of functions by normal sheep alveolar macrophages and their alteration by interaction with *Mycoplasma ovipneumoniae*. Vet Microbiol **58**:31-43, 1997

9. Schroedl W, Fuerll B, Reinhold P, Krueger M, Schuett C: A novel acute phase marker in cattle: lipopolysaccharide binding protein (LBP). J Endotoxin Res **7**:49-52, 2001
10. Wang J, Kieba I, Korostoff J, Guo T, Yamaguchi N, Rozmiarek H, Billings P, Shenker B, Lally E: Molecular and biochemical mechanisms of *Pasteurella haemolytica* leukotoxin-induced cell death. Microbial Pathogenesis **24**:317-331, 1998

CASE IV – 532/01 (AFIP 2790958)

Signalment: 8-year-old, female, warm-blooded horse

History: The horse showed CNS symptoms including ataxia, apathy and dysphagia for 2 days. Additionally, the body temperature was elevated (40.3°C). The animal was not vaccinated against rabies. Despite intensive treatment consisting of antibiotics, glucocorticoids, diuretics, and Vit. B the condition did not improve and the horse was euthanized because of poor prognosis.

Gross Pathology: The brain showed a moderate, diffuse whitish clouding of the meninges. Liver, lung and spleen displayed acute congestion and additionally, the lung showed an acute moderate diffuse alveolar edema.

Laboratory Results: The official investigation for the presence of rabies virus antigen by direct immunofluorescence revealed a positive result, which was confirmed by immunohistology.

Contributor's Morphologic Diagnosis: Brain: acute - subacute, moderate, multifocal, nonpurulent encephalitis and numerous neuronal intracytoplasmic inclusion bodies and mild to moderate acute diffuse gliosis.

Contributor's Comment: Histologically, a moderate (2-4 cell layers), multifocal perivascular infiltration with lymphocytes, macrophages and plasma cells was observed mainly in the brain stem, hippocampus, thalamus, mesencephalon and the spinal cord (cervical part). In the Purkinje cells of the cerebellum numerous eosinophilic round - ovoid intracytoplasmic inclusion bodies of approximately 2-4 um in diameter were found; similar inclusion bodies could be observed occasionally in neurons of the hippocampus and medulla oblongata. Additionally, a moderate diffuse infiltration with glial cells was observed. In some locations, glial nodules (Babes' nodules) were found. In addition, sections of the spinal cord (lumbar part) displayed focal hemorrhages.

The genus *Lyssavirus* belongs to the family *rhabdoviridae* of the single-stranded negative-sense RNA-viruses (order: mononegavirales). Based on virion properties and serologic relationships, four genera containing animal viruses have been recognized in the family Rhabdoviridae, the genera *Lyssavirus*, *Vesiculovirus*, *Ephemerovirus*, and *Novirhabdovirus*. The genus *Lyssavirus* comprises rabies virus and closely related viruses, including Mokola virus, Lagos bat virus, and Duvenhagen virus from Africa, European bat viruses 1 and 2, and Australian bat lyssavirus. Each of these viruses is considered capable of causing rabies-like disease in animals and humans. The genome encodes five genes encoding for five proteins, the glycoprotein contains neutralizing epitopes, which are targets of vaccine-induced immunity; it and the nucleoprotein have epitopes involved in cell-mediated immunity.

Rabies virus can infect all warm-blooded animals (incl. humans), and in nearly all instances the infection ends in death. The disease occurs throughout the world, with certain exceptions: there is no rabies in Japan, the United Kingdom, New Zealand, Antarctica, and many smaller islands such as Hawaii and most of the islands of the Caribbean basin. In many countries, wildlife rabies (sylvatic form) has become of increasing importance as a threat to domestic animals and humans. In most countries of Asia, Latin America, and Africa endemic dog rabies (urban form) is a serious problem. In recent years the immunization of wild animal reservoir host species, especially foxes, by the aerial distribution of baits containing attenuated virus vaccine has become the method of choice. Since 1990, fox rabies, the only endemic rabies in much of Europe, has been eliminated from Switzerland and large areas of France and southern Germany. The incidence of rabies in horses is low when compared to wildlife or domestic small animals. However, it continues to climb in the US and continental Europe. In 1990 there were still 5583 cases of rabies (34 of them horses) in Germany, 1995 only 155 cases (4 horses) and 2000 192 cases (0 horse). Until now (31.05.2001) about 50 cases of rabies and only 1 case of horse rabies are reported.

The clinical features of rabies are similar in most species, but there is great variation between individuals. Following the bite of a rabid animal the incubation period is usually between 14 and 90 days, in experimentally infected horses the average incubation period was 12.3 days and average morbidity 5.5 days. There is a prodromal phase prior to overt clinical disease that often is overlooked or is recalled only in retrospect as a change in temperament. Two clinical forms of the disease are recognized: furious and dumb or paralytic. In the furious form, the animal becomes restless, nervous, and aggressive. The animal often cannot swallow water, giving rise to the old name for the disease, "hydrophobia". There is often excessive salivation, exaggerated responses to light and sound, and hyperesthesia. As the encephalitis progresses, fury gives way to paralysis, and the animal presents the same clinical picture as seen in the dumb form of the disease. Terminally, there are often convulsive seizures, coma, and respiratory arrest, with

death occurring 2 to 14 days after the onset of clinical signs. In horses, clinical signs are ranging from mild, e.g. muzzle tremors and lethargy, to severe, e.g. charging observers and persistent recumbency, also pharyngeal paresis or pharyngeal paralysis, ataxia or paresis, lameness, colic, hyperesthesia, loss of tail and anal sphincter tone and fever can be observed.

The proportion of animals that develop rabies after exposure depends on the location and severity of the bite and the species of animals involved (foxes can carry up to 10^6 infectious units of virus per milliliter of saliva). The virus must gain entry into peripheral nerves by binding specifically to the receptor for the neurotransmitter acetylcholine at neuromuscular junctions. Neuronal infection and centripetal passive movement of the viral genome within axons deliver virus to the central nervous system, usually via the spinal cord. Virus reaches the limbic system of the brain, where it replicates extensively, and causes the release of cortical control of behaviour; this leads to the fury seen clinically. Spread within the central nervous system continues, and when replication occurs in the neocortex, the clinical picture changes to the dumb or paralytic form of disease. Depression, coma, and death from respiratory arrest follow.

In animals species that serve as reservoir hosts of rabies virus, late in the infection viral genome moves centrifugally from the central nervous system through peripheral nerves to a variety of organs: the adrenal cortex, pancreas, and, most importantly, the salivary glands. Thus, at the time when viral replication within the central nervous system causes the infected animal to become furious and bite indiscriminately, the saliva is highly infectious. But already up to several days before the clinical onset of rabies the saliva contains infectious virus. On histopathologic examination, rabies causes typically a nonsuppurative encephalomyelitis with ganglioneuritis and parotid adenitis. Inflammatory changes are usually present but can be very mild or absent. Perivascular lymphocytic cuffing, focal gliosis and ring hemorrhages are common. The severity of lesions could reflect the duration of the clinical disease. Most severely affected brain regions extend from the pons to the hypothalamus or to the cervical spinal cord, with relative sparing of the medulla. This relative sparing of the medulla appears to apply to all the domestic species. The most severe lesions of the disease are generally found in dogs, whereas other species, especially ruminants, which are highly susceptible, may show occasionally a sparse perivascular cuffing with lymphocytes and few focal glial nodules (Babes' nodules), and this in spite of having numerous Negri bodies. Babes' nodules are composed of microglia, and they occur in both white and grey matter and could vary greatly in size.

Neuronal degeneration in carnivores may be very extensive, but may be very slight in pigs and herbivores. Neurons of any distribution may contain the typical inclusion bodies (Negri bodies), which are always intracytoplasmic round-oval, and range from 0.25 to 27 um in diameter with a thin clear halo, but they tend to be

scarce where the inflammatory reaction is severe. Negri bodies may be found only in neurons that are histologically normal; they are not present in degenerated neurons. Most commonly they occur in the hippocampus of carnivores and Purkinje cells of herbivores, rarely found in ganglion cells of the adrenal medulla, salivary glands, and retina.

In most countries, laboratory diagnosis of rabies is done only in approved laboratories by qualified, experienced personnel. If rabies is suspected, the suspect animal must be killed and brain tissue collected for testing. Postmortem diagnosis involves direct immunofluorescence to demonstrate rabies antigen in touch impressions of brain tissue (medulla, cerebellum, and hippocampus). Antemortem diagnosis is only done in suspected human rabies cases. For this, skin biopsy, corneal impression, or saliva specimens are used. Only positive results are of diagnostic value.

The observation that some rabid horses bite at objects and express the furious form of rabies would render horses a real potential source of human exposure and stresses the importance of equine rabies as a potential zoonosis.

AFIP Diagnosis: Brain stem and cerebellum: Meningoencephalitis, nonsuppurative, multifocal, mild, with neuronal, intracytoplasmic, eosinophilic, inclusion bodies (Negri bodies), etiology consistent with rabies virus, warmblood horse (breed not specified), equine.

Conference Comment: The contributor has provided a complete review of this entity.

Contributor: Institut fur Veterinar-Pathologie, Frankfurter Str. 96, 35398 Giessen

References: 1. Hudson L, Weinstock D, Jordan T, Bold-Fletcher N: Clinical presentation of experimentally induced rabies in horses. J Vet Med B **43:**277-285,1996
2. Jubb K, Huxtable C: The nervous system. *In*: Pathology of domestic animals, eds. Jubb K, Kennedy P, Palmer N, 4th ed., vol. 1, pp. 403-406, Academic press, San Diego, CA 1993
3. Murphy F, Gibbs E, Horzinek M, Studdert J: Veterinary Virology, 3rd ed., pp. 165-167, 432-439. Academic press, San Diego, CA,1999

Brad A. Blankenship, DVM
Captain, Veterinary Corps, U.S. Army

Wednesday Slide Conference Coordinator
Department of Veterinary Pathology
Armed Forces Institute of Pathology
Registry of Veterinary Pathology*

*Sponsored by the American Veterinary Medical Association, the American College of Veterinary Pathologists and the C. L. Davis Foundation.