

Chapter 5A

THE DIGESTIVE SYSTEM - UPPER ALIMENTARY TRACT

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5.1 AN OVERVIEW

The digestive system is one of the largest organ systems in the body; it includes the alimentary tract, salivary glands, tonsils, liver, pancreas, and peritoneum. This system is involved, either directly or indirectly, in most of the diseases that affect animals. In fact, any well transcribed clinical record will include a large component of information relative to its status; i.e., feed consumption, water intake, appearance of the oral mucous membranes, bowel movements, consistency of the feces, etc. Body temperature (**another vital component of clinical assessment**) is commonly taken per rectum. This parameter is a relatively accurate reflection of deep body temperature.

There are numerous factors that predispose the digestive system to diseases, including exposure to the external environment via the mouth and rectum. Other significant factors include methods of animal husbandry, age and genetic background. Likewise, there are many protective mechanisms (**digestive juices, mucus, movements and IgA activity**) which aid the animal in avoiding infections, trauma, indigestion or other types of G.I. disturbances. It is important to remember that diseases of the digestive system will usually result in disturbances throughout the body.

For discussion purposes, the Digestive System has been divided into three parts:

PART A: Upper Alimentary Tract

PART B: Lower Alimentary Tract

PART C: Liver and Biliary Tract

The upper alimentary tract includes the lips, oral cavity, buccal cavity, pharynx, salivary gland, esophagus, forestomachs, and true stomach (**including the abomasum**). The lower alimentary tract includes the small intestine, cecum, colon, rectum, and peritoneum.

5.2 OBJECTIVES

At the conclusion of this section, each student should be able to perform the following tasks.

- 1. Briefly, discuss the possible causes, clinical manifestations, gross findings, and significant microscopic alterations associated with stomatitis in animals.
- 2. Briefly, discuss the differentiating features and similarities between foot and mouth disease, vesicular stomatitis, and vesicular exanthema.
- 3. Characterize the diseases listed below on the basis of causative mechanisms, clinical manifestations, gross lesions, microscopic findings, pathogenesis and prognosis.
 - -- Malignant catarrhal fever
 - -- Actinobacillosis
 - -- Bovine papular stomatitis
 - -- Contagious ecthyma
 - -- Traumatic reticulitis
- 4. Briefly, discuss the differences and similarities between actinomycosis, nocardiosis, and actinobacillosis on the basis of the etiologic agents, clinical manifestations, and lesions.
- 5. Describe the manner in which injury to the esophagus may lead to esophageal ulcerations, stenosis, **"choke,"** ruminal bloat, gangrenous pneumonia, and esophageal dilatation.
- 6. Distinguish between simple indigestion, ruminal impaction, rumenitis, and acute carbohydrate engorgement on the basis of etiologic mechanisms, clinical signs, and necropsy findings.
- 7. Discuss the differentiating features and similarities between **"frothy bloat"** and "free gas bloat" on the basis of causative mechanisms, clinical signs, prognosis, predisposing factors, and necropsy findings.
- 8. In the case of a 12 year old Coonhound with gastric torsion, twist of more than 180 degrees, give the likely predisposing causes, exciting causes, prognosis, clinical manifestations, pathogenesis, and necropsy findings.
- 9. For left-sided displacement of the abomasum in an 8 year old holstein, give the likely causes, clinical manifestations, prognosis, and necropsy findings.
- 10. Briefly discuss the development of gastric ulcers in young pigs and calves.
- 11. Provide correct answers for self-study questions at the end of this unit.

KEY WORDS

"Lumpy jaw" -- Ruminal stasis -- Vesicular

Rumen overload -- Toxemia -- Gingivitis

Noma -- Septicemia -- Phlegmon

Primary bloat -- Palatoshisis -- Secondary bloat

Chiloschesis -- Free gas bloat -- Brachygnathia

Frothy bloat -- Prognathism -- Ruminal tympany

Mucormycosis -- Chilitis -- Papilloma

Dental malocclusion -- Eructation -- Parakeratosis

Varicosity(esophageal)-- Pulpitis -- Trichobezoar

Lymphoid leukosis -- Periodontitis -- Phytobezoar

Dental caries -- Gastritis -- Dental calculi

Abomasitis -- Odontodystrophy -- Torsion

Ameloblastoma -- Volvulus -- Epulis

Obligate parasite -- Adamantinoma -- Diverticulum

Odontoma -- Fore stomachs -- Peyer's patches

True stomachs -- Wooden tongue -- Sulfur granules

Stomatitis -- Glossitis -- Lampas

"Orf" -- Tonsillitis -- Sialadenitis

"Sore mouth of sheep" -- Sialangitis -- Mucocele

Sialolith -- Aptyalism -- Ptyalism

Sialosis -- Ranula -- "Choke"

Dysphagia -- Deglutition -- Megaesophagus

5.4 ORAL CAVITY AND ASSOCIATED STRUCTURES

5.4.1 INFLAMMATION OF THE ORAL CAVITY

The term stomatitis refers to diffuse inflammation of the oral mucosa and includes glossitis (**inflammation of the lingual mucosa**), lampas (**inflammation of the hard palate**) and gingivitis (**inflammation of the gums**). Chilitis (**inflammation of the lips**) will also be included.

Stomatitis may be caused by physical agents (**foreign bodies, maloccluded teeth, hot water, etc.**), chemical agents (**acid, alkali, etc.**), bacterial agents

(**Fusobacterium necrophorum, etc.**), viral agents (**viruses responsible for vesicular stomatitis, vesicular exanthema, bovine virus diarrhea, malignant catarrhal fever, etc.**), and fungi (**usually Monilia spp.**). Clinically, the condition is characterized by partial or complete loss of appetite, chewing movements and smacking of the lips, profuse salivation and slow and painful mastication. Stomatitis is classified according to the types of exudate present and the reaction may be limited to the mucosa or involve deeper structures. Catarrhal stomatitis is the type most commonly observed in animals; it involves the superficial mucosa and is always acute. Catarrhal stomatitis frequently develops during the course of many infectious and/or acute debilitating diseases. In uncomplicated cases, healing occurs rapidly. Vesicular stomatitis (**characterized by the formation of vesicles**) occurs in horses, cattle and swine. Such lesions are associated most commonly with foot and mouth disease, vesicular stomatitis or vesicular exanthema. Oral erosions, ulcers and abscesses may develop in association with these diseases. Noma is a rapidly spreading pseudomembranous or gangrenous stomatitis which is associated with pathogenic activity of normal oral flora. The organisms present in the lesions are chiefly spirochetes and fusiforms. The condition, which is observed occasionally in dogs, is in many respects similar to oral necrobacillosis.

5.4.2 FOREIGN BODIES IN THE ORAL CAVITY

A wide variety of foreign bodies may cause injury to the mouth and pharynx. The most common types of injury are produced by coarse feeds, thorns or sharp objects in the feed. Remember, the presence of food in the mouth of a carcass is abnormal. In most cases, it is attributable to intracranial diseases which result in paralysis of deglutition or semiconsciousness. The food is usually poorly masticated and readily differentiated from that regurgitated just prior to death. Swine have a diverticulum in the posterior wall of the pharynx (**immediately above the esophagus**) in which various foreign bodies may lodge. This occurs primarily in young pigs and death follows a phlegmonous inflammation.

5.4.3 CIRCULATORY CHANGES

Careful examination of the oral mucous membranes for circulatory changes is an essential detail in any clinical or necropsy examination. Pale mucous membranes may indicate anemia (**this can be misleading in a carcass**). In cyanosis, the mucosa is a dark reddish-blue color. Small mucosal hemorrhages are indicative of septicemia (**minute petechial hemorrhages on the ventral surface of the tongue and frenulum in horses are regarded as being diagnostic of equine infectious anemia**).

5.4.4 PIGMENTATIONS:

Melanotic Pigmentation is normal and common in most breeds of animals and it increases with age. Diffuse yellowish pigmentation of the mucosa may be seen with icterus. Blackish-yellow coloration has been noted with canine plumbism.

5.4.5 CONGENITAL ANOMALIES OF THE ORAL CAVITY

Congenital anomalies of the oral cavity are usually expressed as clefts which result from failure of integrated growth and fusion. Cleft palate (**palatoschisis**) is characterized by inadequate bilateral in-growth of the palatine shelves from the maxillary processes resulting in a central defect or communication between the oral and nasal cavities. Cleft lip (**chiloschisis**) is due to incomplete fusion of the frontonasal process with the maxillary processes (**rabbits normally have a split upper lip providing the basis for the term "hare lip"**). Oftentimes, cleft lip and cleft palate occur together.

Anomalies in growth of the jaws occur quite frequently in animals. Brachygnathia superior refers to a shortened maxilla. This is an inherited characteristic in some breeds of dogs and swine (**Boxer, Boston Terrier, Berkshire Pigs, etc.**). Brachygnathia inferior (**micrognathia**) refers to a shortened mandible. This is a breed characteristic in "long nose" dogs and occurs to a mild degree in sheep and cattle. Prognathism refers to an abnormal prolongation of the mandible. The condition is rather common, especially in sheep. Remember, it is oftentimes difficult to determine which of the jaws, the upper or lower, is actually affected.

5.4.6 NEOPLASMS OF THE ORAL CAVITY

A wide variety of neoplasms may arise from tissues of the mouth and related structures. In dogs, viral oral papillomas occur most frequently. They tend to disappear spontaneously in 1-3 months if not removed. Squamous cell carcinomas frequently occur in cats, dogs, cattle, and horses. Fibrosarcomas are the most common sarcoma of the oral cavity. Osteogenic sarcomas may arise in the alveolar processes of the mandible and maxilla.

5.5 TEETH AND DENTAL STRUCTURES

Dental diseases are common and important in animals. However, they are oftentimes overlooked on clinical examination and at necropsy. Some of the more common and important pathologic conditions are considered below. More detailed consideration will be given in your medicine courses.

5.5.1 INFLAMMATION OF THE TEETH:

The vascular tissue of teeth is the dental pulp or connective tissue core of the tooth. This is the only tissue of teeth capable of exhibiting inflammatory changes. Pulpitis is usually caused by bacterial agents or their products which enter via fractures, defects in the enamel, etc. Many cases terminate in suppuration, necrosis or gangrene. Periodontal disease is an inclusive term commonly used to describe inflammatory disease of the gingiva (**gingivitis**) or, in more severe cases, inflammation of the gingiva with extension of infection to underlying tissues of the periodontium (**periodontitis**). Periodontitis is common in dogs, older horses and ruminants. The cheek teeth are usually involved.

5.5.2 DENTAL CARIES:

Caries refer to a condition in which there is destructive decalcification of the dental enamel which is followed by destruction of underlying tissues. Caries usually develop

when the normal alkaline condition of the mouth changes to acid. After the **pH change**, anaerobic streptococci which are found in the mouth of all animals, attack **sugars** and **CHO**, resulting in lactic acid production. The lactic acid accumulates around teeth resulting in decalcification. A developing carie usually presents the appearance of a "black cavity" in the tooth. Eventually, the entire tooth turns black or a dark color. Caries that develop in the molars of the lower jaw may be complicated by abscess formation at the tip of the root. This infection may gravitate and eventually break through the jaw bone and through the skin forming an open fistulous wound along the lower edge of the mandible. If an abscess develops in a tooth in the upper jaw, it may eventually extend into a sinus, resulting in a purulent sinusitis.

5.5.3 DENTAL CALCULUS:

Dental calculus (**tartar**) refers to calcified masses of bacteria and desquamated cells. In horses, tartar is rather soft and chalky, whereas in the dog, it is hard and located along the neck of the teeth. In sheep and goats, tartar is usually black to red. Tartar development is associated with

- (1) stagnation of saliva,
- (2) particles of food clinging to teeth and
- (3) the consistency of the diet.

5.5.4 ODONTODYSTROPHY:

Odontodystrophy refers to hypoplasia of the enamel. This condition may be congenital or acquired. Odontodystrophy is usually caused by or associated with some systemic disturbance which interferes with the function of ameloblasts during the active stage of development. Factors that may cause hypoplasia of the enamel include chronic fluorine poisoning, canine distemper virus infection and malnutrition.

5.5.5 PIGMENTATION

Pigmentation of teeth may occur under several conditions. Tetracyclines may stain or affect the color of teeth after administration. The tetracyclines have an affinity for minerals, such as calcium. Thus, tetracyclines may bond to calcium molecules while the two are circulating in the bloodstream. Subsequently, when calcium is deposited anywhere in the body (**bones, teeth, etc.**) the tetracycline molecule is deposited in exactly the same position and is permanently fixed there, unless resorption takes place which again releases the calcium. In dogs, tetracyclines will cause yellowing of the enamel of deciduous teeth if given to pups between 2 to 3 weeks of age. The most critical age for permanent teeth to be affected falls between 8 and 24 weeks of. In addition, the tetracycline molecule contains a region which will fluoresce if exposed to ultraviolet light. If affected teeth are subsequently exposed to UV light, the fluorescence can be detected.

5.5.6 CONGENITAL PORPHYRIA:

Inborn errors in porphyrin metabolism may cause discoloration of the dentin of teeth (**pink tooth**); bones are also involved. This is a rare defect in the metabolism of hemoglobin in which hemoglobin related pigments circulate in the blood. In young

animals the teeth are reddish while in older animals they appear brownish. The pigment does not adversely affect teeth but sometimes it causes photosensitization in the animal's skin if it is exposed to UV light.

5.5.7 CANINE DISTEMPER VIRUS:

CDV will cause defects in the enamel of permanent teeth if infection occurs while they are forming. The virus attacks epithelial cells and infected cells are unable to function normally which results in defective enamel production. Affected dogs may have brownish spots in the enamel.

5.5.8 DENTAL MALOCCLUSION:

Malocclusion of the teeth refers to improper alignment of grinding surfaces of opposing teeth. The condition occurs for a variety of reasons (**imperfect aligned jaws, cribbing in horses, etc.**). In rodents and rabbits, the incisors grow continuously and normal length is maintained by constant grinding of opposing dental arcades.

5.5.9 ODONTOGENIC NEOPLASMS AND NEOPLASTIC-LIKE LESIONS:

The following neoplasms or neoplastic-like lesions are derived from the dental germ

(Reference: Tumors of Domestic Animals, J. E. Moulton, Second Ed., p. 240).

5.5.9.1 Ameloblastoma (acanthomatous epulis)

is a true neoplasm which arises from periodontal squamous epithelial cell residues of the tooth germ. This malignant neoplasm is invasive but does not metastasize. It is usually difficult to differentiate an ameloblastoma from a squamous cell carcinoma. However, there is a tendency for the ameloblastoma to mimic the structure of the enamel organ. This neoplasm is rare in all domestic animals.

5.5.9.2 Periodontal Fibromatous Epulis

is a benign neoplasm that has the same origin as the ameloblastoma. It is fairly common only in the dog. Grossly, fibromatous epulis is usually small, pink, fibrous, gritty, or bony.

5.5.9.3 Odontoma:

Odontomas are considered to be hamartomatous lesions rather than true neoplasms. They represent abnormal development of dental tissues and consist of enamel, dentin and cementum.

5.6 SPECIFIC DISEASES OF THE ORAL CAVITY AND ASSOCIATED STRUCTURES

In addition to oral lesions, some of the diseases discussed below are characterized by pathologic alterations in other organs and tissues. Also, oral lesions may be a feature of diseases discussed elsewhere in this section.

5.6.1 MALIGNANT CATARRHAL FEVER:

Malignant catarrhal fever is an acute, highly fatal, infectious disease of cattle caused by a herpesvirus. The disease is characterized by:

- (1) an erosive stomatitis and gastroenteritis
- (2) catarrhal to mucopurulent inflammation of the eyes (**keratoconjunctivitis**) and nostrils
- (3) erosions in the upper respiratory tract
- (4) lymph node enlargements and
- (5) encephalitis.

Cattle and buffalo are the only domestic animals in which the clinical disease occurs. However, sheep and goats (**as well as wildebeest**) can develop an unapparent infection

North American outbreaks of malignant catarrhal fever most commonly occur when cattle are housed with sheep, which is considered to be evidence of cattle to sheep transmission of the virus; but spread by direct contact between cattle does not seem to occur. Clinically, the disease may occur in one of several forms (**Peracute form, "mild" form, head and eye form and alimentary tract form**). However, these are artificial separations with cases being classified on the basis of the predominate clinical sign. Conjunctivitis and rhinitis may occur. The eyes are sensitive to strong light and the cornea becomes opaque in the later stages. Erosions develop on the skin of the muzzle and on the inside of the mouth (**stomatitis**).

The infectious agent responsible for malignant catarrhal fever results in a generalized infection of primitive mesenchymal cells. This herpes infection is not characterized by typical in vivo herpetic necrosis in most tissues, but is more strongly aligned with effects considered to be consistent with lymphotropic viruses. However, there is a vascular component which probably accounts for a significant proportion of the injury that is noted. Grossly, lesions in the mouth, nasal cavity, pharynx, and esophagus may range from minor hemorrhage and erythema, to discrete erosions, through severe inflammation. Catarrhal enteritis with swelling and ulceration of Peyer's patches is a constant feature. All lymph nodes are swollen, edematous and often hemorrhagic. Microscopically, a necrotizing vasculitis of the media of arteries and arterioles is the basic and characteristic lesion. Infiltration by mononuclear cells occurs throughout most organs and tissues, including the brain, liver and kidneys.

(Reference - Pathology of Domestic Animals, Jubb and Kennedy, Vol. 2, p.102).

5.6.2 ACTINOBACILLOSIS

Actinobacillosis is a specific infectious disease caused by *Actinobacillus lignieresii*. The disease is characterized by inflammation of the tongue and regional lymph

nodes. It occurs most frequently in cattle where the tongue is the favorite site for infection. The chronically indurated tongue is known clinically as "wooden tongue." In sheep, characteristic lesions may occur in subcutaneous tissues of the head and neck and consist of suppurative granulomas. Tongue lesions do not usually occur in this species. *Actinobacillus ligniersi* is believed to be an obligate parasite of the upper respiratory alimentary mucosa which gains entry to the tongue and other soft tissues through abrasions of mucous membranes.

Clinically, there is excessive salivation and gentle chewing of the tongue. Upon examination, the tongue is swollen and/or hard, especially near its base. Nodules and ulcers are present along its sides. In later stages, lingual fibrosis is a prominent feature. At this stage, the tongue becomes indurated, shrunken and immobile. The parotid, submaxillary and retropharyngeal lymph nodes are enlarged and palpable. Grossly, suppurative and/or granulomatous lesions are observed in the soft tissues of the oral cavity (**especially the tongue**), and in regional lymph nodes. Minute "sulfur granules" may be observed upon close examination. These granules represent bacterial colonies. Microscopically, lesions are characterized by discrete colonies of organisms surrounded by radiating clubs suspended in pus. These foci are encapsulated by a rather dense connective tissue. The bacterial colonies are gram-negative and rodshaped. Remember, actinobacillosis must be differentiated from actinomycosis and nocardiosis.

(References: - Pathology of Domestic Animals, Jubb and Kennedy, vol.2 p. 35. and Veterinary Medicine, Blood and Henderson, 5th edition, p. 524.

5.6.3 BOVINE PAPULAR STOMATITIS

This is a mild self-limiting viral disease characterized by raised papular lesions within and around the mouth which spontaneously regress after a few weeks. In addition to the mouth, lesions develop in the nostrils, esophagus and forestomachs. Clinical cases are most frequently encountered in young cattle from 2 weeks to 2 years of age. The causative virus has many of the characteristics of the pox group and is classified as a para-vaccina (pseudo cow-pox) virus. Bovine papular stomatitis is of little importance in its own right, but the lesions may serve as a portal of entry for secondary bacterial infection. Following exposure to infected cattle, erythematous papules may develop on the hands and arms of man. In man, the disease is considered to be an occupational hazard because he often becomes infected while examining the mouths of infected animals. Grossly, the lesions in animals begin as small papules which become dark red in color. Subsequently, they expand peripherally so that lesions are always round or nearly so. As the lesions expand, their periphery becomes reddened and their center appears depressed and grey-brown in color. Microscopically, there is ballooning degeneration and the presence of cytoplasmic inclusion bodies in affected epithelial cells.

5.6.4 CONTAGIOUS ECTHYMA

Contagious ecthyma is a highly infectious viral disease of sheep and goats characterized by the presence of vesicular, pustular and/or "**scabby**" lesions on the lips, oral mucosa, udder and feet. The causative virus is similar to pseudocowpox virus. The disease occurs wherever sheep are raised and the incidence within a flock may be as high as **90%**.

Grossly, the lesions initially develop as papules which progress to pustules. Subsequently, thick scabs cover raised ulcerative areas. The first lesions develop along the oral mucocutaneous junction; afterwards, they spread to the epithelium of the muzzle and nostrils. Microscopically, cytoplasmic inclusion bodies occur but are transitory. In addition to sheep and goats, cattle may also be naturally affected. Clinical disease may occur in humans working with infected sheep. Contagious ecthyma is also referred to as "**orf**", "**contagious pustular dermatitis**" and "**soremouth**."

5.6.5 VESICULAR STOMATITIDES

There are several important viral diseases of large animals which are characterized by vesicular eruptions in the mouth (**foot and mouth disease, vesicular stomatitis, and vesicular exanthema**). In these diseases, vesicles develop as accumulations of serous fluid in the deeper layers of the epithelium or between the epithelium and the basement membrane. The lesions may coalesce to form bullae. Erosions occur subsequent to rupture of the vesicles/bullae. The transition from vesicles to erosions can occur very rapidly. In naturally occurring infections, the vesicular stage may not have been manifested clinically.

Foot and Mouth Disease is an acute, extremely contagious naturally occurring viral disease of "cloven-footed" animals which is characterized clinically by fever and vesicular eruptions in the mouth and on the feet. The disease is a problem of worldwide concern. This disease is epizootic in large areas of Africa, Europe and South America. North America, Japan, Australia and New Zealand are currently free of the disease because of quarantine restrictions and geographical isolation. There are at least seven major strains of the causative enterovirus. Strains A, O and C are classics. However, there are substrains with different serological and immunologic characteristic and with different degrees of virulence. Clinically, there is an initial period of high fever (**104-106 degrees F.**) which is accompanied by anorexia and is followed by an acute painful stomatitis. Salivation is abundant and smacking of the lips is noted. Vesicles and bullae appear on the buccal mucosa, the dental pads and tongue. The vesicles and bullae rupture within 24 hours leaving a raw painful surface which heals in about one week. Concurrently with the oral lesions, vesicles appear on the feet (**in the cleft between the digits and on the coronet**). Also, vesicles may occur on the teats. Abortion and subsequent infertility are common sequels. A malignant (**in terms of clinical course**) form of foot and mouth disease may occur in young animals. In these cases, vesicles may not be recognized. Death occurs rapidly and is associated with acute myocardial failure. Histologically, there is a myocarditis in which hyaline changes and foci of necrosis are prominent features. In sheep, goats and pigs, the disease is usually mild and is important mainly because of the danger of transmission to cattle. In general, these animals only exhibit an occasional small lesion in the mouth, but all four feet may be badly affected and the animal may be severely lame.

Vesicular Stomatitis is an infectious rhabdoviral disease characterized by the development of vesicles on the mouth and feet. It is a disease primarily of horses but has come to assume major importance in cattle and swine (**sheep are resistant**). In general, vesicular stomatitis has no great intrinsic importance. However, because of its similarity to foot and mouth disease, prompt and accurate diagnosis of the disease is essential. Remember, foot and mouth disease does not occur in horses and vesicular exanthema occurs naturally only in swine. The lesions of vesicular stomatitis are indistinguishable from those of foot and mouth disease. The disease may occur in man.

Vesicular Exanthema of Swine is an acute, febrile infectious viral disease characterized by the development of vesicles in the mouth and on the feet. The disease occurs naturally only in swine. Experimental animals cannot be reliably infected under usual laboratory circumstances. The causative virus is classified as a calicivirus and 13 antigenic strains have been isolated. Except for isolated outbreaks in Hawaii and Iceland, the disease has occurred only in the United States. It was eradicated from the USA in 1959, twenty seven years after its initial appearance. The vesicular lesions are indistinguishable from those of foot and mouth disease and vesicular stomatitis. Vesicular stomatitis of swine cannot be differentiated from foot and mouth disease or vesicular stomatitis by clinical or necropsy examinations. A definitive diagnosis can only be made on the basis of transmission experiments or by the use of serological tests.

5.7 PATHOGENS IN THE ORAL CAVITY OF ANIMALS

It is relevant at this point to mention some of the infectious agents that may be associated with the mouth of animals. This is important because of the possibility of infection of the veterinarian and others and/or its importance in regard to treatment and control. Remember, some species may have infectious agents in the mouth that are not found in others. Therefore, knowledge of the particulars of the species becomes important in handling animals, protecting the health of workers, and in the treatment (**or recognition that no treatment is possible**).

5.7.1 SNAKES:

Most snake venom contains either a neurotoxin, a necrotoxin, a hemolytic toxin, or some combination of these toxins. These agents, even with no complications, present the veterinarian with a problem that may tax his skills to treat. However, numerous infectious agents almost always accompany snake bites. These in turn can cause severe complications. The three most common complications include the possibility of

- (1) tetanus infection,
- (2) pseudomonas infection and
- (3) staphylococcal infection.

Tetanus organisms can be destroyed before they become active if, initially, the possibility of their presence is taken into consideration. Pseudomonas organisms are always difficult to treat. Staphylococcal organisms may or may not be resistant to treatment.

5.7.2 MONKEYS:

A monkey bite may transmit numerous staphylococcal organisms which are pathogenic to man. In addition, monkeys are capable of transmitting an infectious disease known as monkey B (**herpes virus simiae**). Monkey B virus causes a fatal ascending myelitis and an encephalitis in man. The infection is obtained by direct contact with monkeys. The disease is transmitted via the bite of monkey in which either saliva or monkey tissue is deposited into the wound. Remember, the disease is usually unapparent in some species of monkeys. Occasionally, in these carriers, ulcers may develop on the tongue, but these heal within 7 to 14 days without leaving an apparent scar. One exception to the recovery of monkeys is the marmoset in which the disease is fatal. Monkey bites must always be considered as serious accidents and veterinarians must always take the above mentioned infections into consideration when handling pet monkeys. Records indicate that people may die as early as 18 hours after being bitten by what appeared to be a perfectly normal monkey. Actually, the virus has been isolated from the brains of normal monkeys and serological tests reveal that from **20% to 75%** of monkeys in some colonies have antibodies to this virus.

5.7.3 CATS:

So-called cat scratch fever is an infection in people associated with the handling of cats. The person may have been bitten or scratched. In either case, a rather tender lymphadenitis develops which may be followed by abscesses in the infected lymph glands. In addition, unidentified diseases have been associated with severe cat bite wounds. It must be remembered, that cats have extremely sharp teeth. A severe bite by a cat could drive the canine teeth into bone and result in an infectious osteomyelitis.

5.7.4 SKUNKS:

Probably the most serious infection associated with skunk bites is rabies. Skunks are not the only animals capable of transmitting rabies by bite, many other mammals can also do this. However, for some **reason(s)**, skunks are able to tolerate the rabies virus for a longer period of time than most animals. Therefore, skunks may be infected with the rabies virus for a rather prolonged period and present no evidence of clinical illness or infection. But sooner or later, the disease makes its appearance and then the disease can be transmitted via a bite. Remember, care should always be taken when handling live or dead skunks. There is a case on record with the U.S. Public Health Service in which a mother skunk was killed and left lying by the road. Several days later, two baby skunks were captured nearby. The baby skunks were "decented" and later sold as pets. Two months later, one of these pet skunks bit the owner, and he died of rabies. The obvious conclusion is that the baby skunk had become infected from the mother skunk while she was living and had carried the rabies virus for more than two months.

5.8 PHARYNX, TONSILS AND SALIVARY GLANDS

5.8.1 PHARYNX:

The pharynx is common to both the respiratory and digestive system and shares the misfortunes of both. Pharyngitis refers to inflammation of the pharynx. Clinically, the condition is characterized by coughing, painful swallowing and lack of appetite. The causes of pharyngitis are the same as those described for upper respiratory and upper alimentary tract diseases. Foreign bodies may lodge in the pharynx or in the pharyngeal diverticulum of pigs. Paralysis of the pharynx is characterized by the inability to swallow and regurgitate. The condition accompanies several specific diseases including rabies, other encephalitides, botulism, etc.

5.8.2 TONSILS:

The tonsils are composed of lymphoid tissue covered with a layer of squamous epithelial cells. In some species (**dogs and cats**), the tonsils are rather well defined structures. In other species (**cows and horses**), the tonsils are ill defined and represented by only a collection of lymph nodules. Inflammation of the tonsils is referred to as tonsillitis. Tonsillitis is usually the result of extension of infection from nearby structures. In dogs, tonsillitis often accompanies canine distemper. In cats, infectious feline enteritis virus is a cause of tonsillitis. Also, tonsillitis is commonly associated with anthrax in swine and strangles in horses. Sheep are the domestic animal with tonsils most similar to those in man.

5.8.3 SALIVARY GLANDS:

Functional disturbances (**excessive or reduced secretion of saliva**) are the most common afflictions of the salivary glands. In general, inflammation of the salivary glands is uncommon in animals.

5.8.3.1 Ptyalism

-- Ptyalism (**sialosis**) refers to excessive secretion of saliva resulting in an abnormal accumulation in the mouth. Ptyalism is associated with a variety of diseases and/or conditions including stomatitis, encephalitis, heavy metal poisoning, etc.

5.8.3.2 Ranula

-- Ranula refers to cystic dilatation and an accumulation of saliva in the salivary duct of the sublingual region. Gradually, the tongue becomes elevated and the animal is unable to close its' jaws.

5.8.3.3 Mucocele

-- Mucocele refers to an accumulation of salivary secretion adjacent to salivary ducts.

5.8.3.4 Sialolithus

-- Sialoliths refers to calculi or concretions that form in salivary glands. They occur most commonly in horses, and may result in obstruction and inflammation. There is a gradual build up of phosphates and calcium carbonates, as well as other salts and

organic material, around a small nucleus of desquamated epithelial cells, bacteria or other foreign materials.

5.8.3.5 Sialoadenitis

-- Sialoadenitis refers to inflammation of the salivary glands. This condition is usually caused by bacterial agents which enter by way of the salivary ducts or via the bloodstream. Sialoadenitis is associated with some viral diseases (**canine distemper, etc.**). In general, inflammation of the salivary glands is uncommon in animals. Remember, most of the cytomegalo viruses have a particular affinity for the salivary glands.

5.8.3.6 Sialangitis

-- Sialangitis refers to inflammation of the salivary ducts. The condition is usually caused by the entry of foreign bodies into the ducts from the mouth. These objects are usually contaminated with bacterial agents.

5.9 ESOPHAGUS

5.9.1 INFLAMMATION OF THE ESOPHAGUS:

Inflammation of the esophagus is referred to as esophagitis. The condition is usually associated with trauma (**foreign bodies, etc.**), ingestion of caustic chemicals (**lye, etc.**) and infectious diseases (**papular stomatitis, malignant catarrhal fever, etc.**). Dilatation of the esophagus (**esophagectasia**) and esophagitis oftentimes accompany stomatitis. Dysphagia (**difficulty of swallowing**) is the classical clinical sign of esophageal disease. Remember, dysphagia is also a sign commonly associated with diseases of the oral cavity and pharynx.

Any severe injury to the esophageal wall (**ulceration, etc.**) is feared because of subsequent healing by granulation. As the granulation tissue matures, the resulting scar tissue may constrict and restrict the esophageal lumen, which is termed "**stricture**" (**a narrowing of the lumen**). Esophageal injury that occurs subsequent to the lodgement of foreign bodies or the ingestion of caustic chemicals is commonly associated with stenosis. Surgical procedures on the esophagus are oftentimes not successful because of the post surgical sequel of contracting granulation tissue. Esophageal stenosis interferes with normal swallowing in all animal species. In cattle, ruminal gases cannot be eructated in a normal manner and acute secondary bloat may develop. Remember, if the esophageal injury is extreme, complete perforation of the wall may occur, but this is uncommon.

5.9.2 CHOKE:

The term choke is used to refer to partial or complete obstruction of the esophageal lumen by foreign materials. The condition is characterized by dysphagia, regurgitation of food and water and bloat in ruminants (**especially cattle**). The common causes of choke in animals are discussed in Jones and Hunt.

5.9.3 DILATION OF THE ESOPHAGUS:

Esophageal dilatation refers to an increased caliber of the lumen. The condition may be either localized, generalized, congenital or acquired. Remember, dilatations should be differentiated from diverticula of the esophagus. Dilatations involve the entire circumference of the affected portion; whereas, diverticula involve only a portion of the circumference. Acquired dilatations may be seen in any animal species. The dilated segments are located proximal to the point of obstruction (**stenosis, foreign bodies, etc.**). Megaesophagus refers to a diffuse (**non-localized**) dilatation of the esophagus. This condition frequently occurs in puppies. The cause of megaesophagus in the dog has not been established. However, most authors agree that it is a neuromuscular disease in which the vagus nerve is primarily affected. Remember, there is no anatomic esophageal cardiac sphincter in the dog. Therefore, megaesophagus is not due to inability of the cardiac sphincter to relax during the act of swallowing, which is the case in achalasia.

5.9.4 RUPTURE OF THE ESOPHAGUS:

Esophageal rupture may be a consequence of impaction, foreign bodies, external penetrating wounds, neoplasms or improper instrumentation. Ruptures at any location within the esophagus usually prove fatal because material contaminated with bacteria can gain entry into the fascial planes of the neck resulting in phlegmon or gangrene. Since the fascial planes are occupied by loose alveolar connective tissue and communicate with the anterior mediastinum, infections are very likely to spread rapidly and may enter the pleural cavity. The rapid spread of infection along the facial planes is aided by esophageal peristalsis, carotid pulsations and the negative pressure of inspiration.

5.9.5 NEOPLASMS OF THE ESOPHAGUS:

Neoplasms of the esophagus are uncommon. However, neoplasms of epithelial and connective tissue origins have been reported. Occasionally, papillomas are observed in the esophagus of dogs and cattle. The papillomas tend to be small, multiple, and recovery is spontaneous. Fibrosarcomas and osteogenic sarcomas have been reported with some frequency in dogs with spirocercosis.

5.9.6 PARASITES OF THE ESOPHAGUS:

Spirocerca lupi are nematodes which are generally found within nodules in the esophageal wall. For some unknown reason, these parasites may initiate the formation of neoplasms in the esophagus. *Hypoderma bovis* and *lineatum* larvae are commonly observed in the esophageal submucosa. *Gongylonema pulchrum* is commonly observed in the mucosa and submucosa of the esophagus of cattle, sheep, goats, swine and horses. The parasites are usually embedded in a **"zig-zag"** fashion in the mucosa and submucosa. *Sarcocystis* is common in the striated musculature of the esophagus of sheep. The cysts are readily visible projecting from the external surface as ovoid white nodules approximately **1.0 cm** in length.

5.10 THE FORESTOMACHS OF RUMINANTS

The forestomachs of ruminants are closely associated anatomically and functionally and disease of one usually affects the others. The condition of the rumen is generally used as an indicator of the state of the other stomach compartments because it is so easily examined clinically. The two major functions of the forestomach are:

- (1) bacterial digestion and fermentation and
- (2) physical maceration by contraction of the stomach walls.

These two functions are interdependent and abnormality of one leads to abnormality of the other. Motility is most readily examined clinically. Therefore, rumen motility is used as an index of digestive function in these species.

When food enters the forestomachs it normally divides into layers (**an upper layer of free gas and a lower layer of fluid containing suspended food particles and gas bubbles**). Mixing of the contents of the rumen and reticulum takes place during ruminal movements which occur at the rate of 1 to 3 per minute. Functional mixing motility of the forestomachs occurs in cycles, commencing with a double reticular contraction. The first is accompanied by a strong contraction of the anterior sac of the rumen. These contractions pour the fluid reticular content over the bulky food mass in the rumen. In the second reticular contraction, a contraction of the ventral ruminal sac follows and fluid is returned to the reticulum. During each reticular contraction, fluid and food particles pass into the reticulo-omasal orifice and into the omasum and abomasum. Eructation contractions begin in the dorsal sacs, pass forward to the cardia of the esophagus, the cardia relaxes and gas is expelled. Eructation contractions are independent of mixing contractions. Their rate depends upon the pressure of the gas in the rumen. Rumination also depends upon additional ruminal contractions which keep the area of the esophageal cardia flooded with reticular fluid.

Examination of ruminal fluid for abnormalities is a valuable aid in the diagnosis of diseases of the ruminant forestomachs. Changes in the pH of ruminal fluid and protozoan numbers and activity occur with carbohydrate engorgement and other forms of indigestion.

In contrast with most other parts of the ruminant alimentary tract, specific lesions of the mucosa of the forestomachs are uncommon. The most common disturbances are functional disorders.

Among domestic animals, the cow, sheep and goat have four stomach compartments. Camels and llamas have three compartments.

5.10.1 SIMPLE INDIGESTION:

Simple indigestion is characterized by anorexia, lack of ruminal movement and constipation. It develops subsequent to atony of the forestomachs. It is common in dairy cattle and stall-fed beef cattle because of the variability in quality and the large amounts of food consumed. Atony of the forestomachs is commonly related to abnormalities in the dietary intake, such as indigestible roughage, mold and overheated or frosted feeds. Simple indigestion is not usually a fatal disease. In this condition, there is no systemic reaction and the pulse, temperature and respiration rates are unaffected. Remember, the difference between simple indigestion and

acute impaction of the rumen is largely one of degrees. There is a marked clinical difference between the two syndromes.

5.10.2 RUMENITIS:

Rumenitis refers to inflammation of the rumen. The condition commonly occurs after overeating on rapidly fermentable carbohydrate (*see below*). Rumenitis may also develop subsequent to the ingestion of caustic chemicals. Inflammatory lesions in the forestomachs are common in some of the specific vesicular and erosive diseases that were discussed earlier in this section (*see stomatitis*). Severe inflammation of the rumen will provide a portal of entry for fungi and *F. necrophorum* to invade the portal circulation which could result in liver necrosis and abscessation.

5.10.3 RUMENITIS AND ACIDOSIS CAUSED BY CARBOHYDRATE ENGORGEMENT:

A condition known as "**carbohydrate engorgement**" in ruminants develops following the ingestion of large amounts of highly fermentable carbohydrate rich feed. This acute disease occurs subsequent to the excessive production of lactic acid in the rumen. Clinically, the disease is characterized by severe toxemia, ruminal stasis, dehydration, weakness, recumbency and a high mortality rate. Acute carbohydrate engorgement is also referred to as grain overload, rumen overload and acidosis.

Oftentimes, cattle with sudden access to highly fermentable feed (**grains, etc.**) will consume far greater amounts at a more rapid rate than the rumen can accommodate. Spoilage commences and excessive lactic acid is produced. Due to the acidic conditions within the rumen, the pH drops from a normal of about 4.5 - 6.5 to an abnormal pH of about 2.0. The normal ruminal flora are also altered. Atony develops and saliva secretion stops. In addition, the increased ruminal lactic acid causes an increase in ruminal osmotic pressure. The net result is that fluids are drawn from the bloodstream and tissues into the rumen.

Subsequently, hemoconcentration and dehydration develop. Gradually, the animal becomes completely anorexic and toxic substances are absorbed into the bloodstream (**toxemia**). At necropsy, the rumen is filled with foul-smelling, fermenting feed and acute passive congestion of the ruminal wall is prominent. A few petechial and ecchymotic hemorrhages may be found over the heart and serosal surfaces. If the postmortem examination is done within one hour after death, determination of the ruminal PH may be of value in confirming the diagnosis. However, after one hour, the pH of the rumen contents begins to increase and its measurements may not be reliable.

5.10.4 RUMINAL TYMPANY (BLOAT):

Ruminal tympany (bloat) refers to overdistention of the rumen and reticulum with the gases generated by fermentation. These gases may be present in the form of a persistent foam mixed with the rumen ingesta (**frothy bloat**) or in the form of free gas separated from the ingesta (free gas bloat). Frothy bloat or primary ruminal tympany is dietary in origin and occurs in cattle on legume pasture and in feedlot cattle which are fed diets containing high levels of grain. Free gas bloat or secondary tympany is

usually due to failure of eructation of free gas because of a physical interference with eructation. In general, primary ruminal tympany is caused by some direct action within the rumen, whereas secondary ruminal tympany is caused by some condition elsewhere in the body with an indirect action on the rumen (**stenosis of the esophagus, etc.**). Only these two types of bloat will be considered.

5.10.4.1 Primary Ruminal Tympany (**Frothy Bloat**):

Primary ruminal tympany or frothy bloat is due to the production of a stable foam which traps the normal gases of fermentation in the rumen. The small gas bubbles which have mixed with the rumen contents do not coalesce, eructation cannot occur, and pressure within the rumen increases. Leguminous bloat is due to the foaming qualities of the soluble leaf proteins in legumes and other forages frequently associated with bloat. The cause of frothy bloat in cattle receiving high level grain diets has not been completely elucidated. However, circumstantial evidence suggests that:

- (1) feeding finely ground grain promotes the formation of foam,
- (2) feeding large amounts of grain results in a marked change in the ruminal flora; thus, allowing some species of encapsulated bacteria to substantially increase in number and produce a slime which may result in the formation of a stable foam and
- (3) insufficient saliva to exert a buffering effect on the pH of rumen may promote the tendency for foam to develop.

In summary, evidence suggests that primary ruminal tympany or frothy bloat develops when:

- (1) the ingesta contains foaming substances (especially soluble leaf proteins),
- (2) the pH of the rumen is suitable for the growth of encapsulated bacteria which produce extracellular polysaccharides (slime), and
- (3) salivation is insufficient either because of the failure of the diet to stimulate it or because of the individual salivary paucity of the animal.

Remember, ruminal distension and death may occur within a few hours.

5.10.4.2 Secondary Ruminal Tympany (**Free Gas Bloat**):

Secondary ruminal tympany or free gas bloat is usually due to some physical obstruction which prevents the gases of normal fermentation from escaping via the esophagus (**interference with eructation**). Physical obstruction to eructation occurs in esophageal obstruction caused by foreign bodies, stenosis, pressure from enlargements outside the esophagus, etc. In addition, interference with the nerve pathways responsible for maintenance of the eructation reflex may cause free gas bloat. The receptor organs in this reflex are found in the dorsal pouch of the reticulum. The free gas is usually present on top of the solid and fluid ruminal contents. Treatment in these cases consists of passage of a stomach tube or trocarization which results in the

expulsion of large quantities of gas. Frothy bloat cannot be effectively relieved by these procedures. Free gas bloat may be acute or chronic.

At necropsy, bloated animals are usually presented with a markedly distended abdomen and with blood oozing from the body orifices. The animal's blood is dark and does not clot (**indicative of death from anoxia**). Lymph nodes in the head and neck regions are congested and hemorrhagic. The lungs are compressed, the cervical portion of the esophagus is congested and hemorrhagic, but the thoracic portion is pale. In general, congestion is marked in the front quarters and less prominent or absent in the hind quarters. The rumen is distended with bulky ingesta (**frothiness of the ruminal contents noted in frothy bloat**). There is usually pressure ischemia of the abdominal visceral organs. Remember, it is difficult to diagnose bloat at necropsy, especially in animals found dead on pasture in warm weather.

It should be emphasized that all legumes do not cause frothy bloat. Those legumes that contain protein precipitants (**usually condensed tannins**) generally do not result in this type of bloat. Interaction of the protein precipitants and soluble leaf protein within the rumen reduces the amount of protein remaining in solution to a level insufficient to give rise to foam

5.10.5 IMPACTION OF THE RUMEN:

Ruminal impaction refers to a rumen which is firm and almost completely filled with very dry ingesta. Atony is present and there is usually no fermentation or digestion occurring.

5.10.6 PARAKERATOSIS OF THE RUMEN:

Parakeratosis of the rumen may be associated with rumenitis. The condition has been reported in animals fed pelleted feed and in animals in which the ruminal contents have been dry for a prolonged period of time. Remember, horny papillae occur normally in the rumen of cattle where most are located around the esophageal opening. They are long, hard, curved, and project from the rumen wall like a small keratinized horn.

5.10.7 FOREIGN BODIES IN THE RUMEN:

It has been estimated that approximately **50%** of adult cattle have foreign bodies in the rumen and reticulum. It is possible that many of the lighter and smaller objects are regurgitated. Foreign bodies consisting chiefly of hair or wool (**trichobezoars**), or plant fiber (**phytobezoars**) may form in the rumen. The most important foreign bodies are those which dissolve and cause intoxication (**lead, etc.**) and those which are sharp and penetrate the wall. Sheep and goat seldom have foreign bodies in their forestomachs. Sheep have rather selective eating habits whereas goats tend to chew all kinds of foreign materials but apparently do not swallow them.

5.10.8 POSTMORTEM MACERATION OF RUMINAL EPITHELIUM:

Within a few hours after death, large patches of ruminal epithelium detach when the ingesta is disturbed. This postmortem change is referred to as maceration.

5.10.9 TRAUMATIC RETICULITIS AND ITS COMPLICATIONS:

Traumatic reticulitis affects cattle more than any other ruminants. When a cow eats roughage, only two physical changes take place in this mass of dry, coarse material which has been taken into the mouth for the first time:

- (1) the mass is rolled around in the mouth until a loose ball has been formed and
- (2) the mass is moistened with enough saliva to be swallowed.

In view of this minimum amount of manipulation of roughage, it is apparent that it would be possible for a nail or piece of wire or some other solid object to be incorporated in a bite of hay without the animal's knowledge. When such a bolus of roughage is swallowed, it drops on the surface of the rumen fluid. Very soon thereafter, the heavy object (**nails, etc.**) will filter through and gradually find their way to the bottom of the rumen and then into the reticulum. The heavy solid objects lodge in and eventually penetrate the reticulum. As soon as the wall of the reticulum has been penetrated, the tissues are inoculated with bacteria. If the object happens to be pointed in the direction of the diaphragm, it will penetrate it and continue on its way to the pericardium, epicardium and myocardium. Occasionally, the object will move laterally to the right and penetrate the liver. In either case, a severe infection is established and fistulous tracts develop with the formation and accumulation of purulent exudate. Traumatic reticulitis is usually seen after 2 years of age. Pregnancy and parturition may precipitate the problem.

5.11 THE TRUE STOMACH

5.11.1 INFLAMMATION OF THE STOMACH:

Inflammation of the monogastric stomach is referred to as gastritis, whereas inflammation of the abomasum is commonly referred to as abomasitis. Unless otherwise indicated, the term "**gastritis**" is used in this discussion to refer to inflammation of the monogastric stomach as well as the abomasum.

Gastritis in animals causes altered motility and is manifested clinically by vomiting. The condition seldom occurs without involvement of the intestine (**gastroenteritis**). The causes of gastritis include physical agents, bacteria, viruses, chemicals, etc. In primary gastritis, the etiologic agent acts directly on the stomach mucosa, whereas in secondary gastritis, there is some primary disease elsewhere in the body and the gastric mucosa is secondarily involved (**uremia, etc.**). Acute catarrhal inflammation is the most common response of the stomach to injury. Grossly, the mucosa is reddened and swollen and there is increased secretion of mucus. Microscopically, there is desquamation of the epithelium together with leukocytic infiltration. Remember, active hyperemia is a normal physiological response when the stomach is active (**digestive processes, etc.**). This type of hyperemia must be distinguished from acute inflammatory reactions. Chronic gastritis is characterized by thickening of

the mucosa which is usually covered by a tenacious glassy mucus. The mucosal folds are also exaggerated.

5.11.2 FOREIGN BODIES IN THE STOMACH:

Foreign bodies found in the stomach may include such items as rubber balls, bottle caps, the plastic covering of meat packages, nylon stockings, etc. An animal with a depraved appetite tries to satisfy a craving for some element that is absent from its diet. A dog with rabies, for example, will bite on objects that he passes and foreign bodies may be swallowed in this manner. In cattle, very few foreign bodies reach the abomasum. Occasionally, a part of a placenta will be found. Foreign bodies are seldom found in the stomach of horses. Piloconcretions or hair balls occur most commonly in long haired cats and in calves. Phytoconcretions are formed in the stomach around a nucleus of indigestible plant fiber.

5.11.3 TORSION OF THE STOMACH:

Gastric torsion is characterized by rotation of the stomach on its long axis at the levels of the cardia and pylorus. Torsion of the "simple stomach" only occurs commonly in dogs (**which are apparently predisposed to the condition by the relative free motility of the organ**). In the dog, the stomach is held in position largely by adjacent viscera and the abdominal muscles. The major gastric ligaments (**gastrophrenic and gastrohepatic**) attach to the stomach at the fixed points, cardia and pylorus, about which it rotates. Thus, these ligaments have little effect upon preventing rotation. Large, older, deep chested dogs are more prone to gastric torsion than the smaller breed. The specific cause(s) of gastric torsion has not been clearly elucidated. However, it has been proposed that the condition is a consequence of acute gastric dilatation due to overeating. In fact, torsion occurs most common in dogs shortly after they have consumed a full meal.

The rotation of the stomach is usually "**clockwise**", as viewed from behind (**the distal portion of the esophagus is twisted and the direction of its spiral folds indicates the direction of the torsion**). The degree of torsion determines the severity of the condition. If the twist is less than 180 degrees, circulatory changes are minor and some food can still pass out of the stomach. However, the digestive processes are severely impaired. If the twist is greater than 180 degrees, severe circulatory changes (**passive hyperemia and hemorrhage**) will develop. The stomach lumen fills quickly with gas and fluid. The abdomen becomes distended and tense. The animal's pulse rate is increased and body temperature gradually drops. Feces is scanty and pain is severe. If the condition is not relieved, death due to shock (**markedly reduced venous return of blood to the heart**) occurs within a few hours. At necropsy, the wall of the stomach is thickened (**hyperemic, hemorrhagic and edematous**) and it is tightly draped by the omentum. Its lumen contains considerable amounts of blood-stained brownish fluid. The spleen rotates with the stomach and traction on its ligaments bends it into the shape of a "**V**" at its middle. Also, there may be displacement of the spleen to a position near the posterior aspect of the abdominal cavity.

5.11.4 DISPLACEMENT OF THE ABOMASUM;

Left sided displacement of the abomasum refers to displacement of the organ from its normal position on the abdominal floor to the left side of the abdomen between the rumen and the left abdominal wall. The condition is more a clinical and surgical problem than one related directly to pathology, since fatal cases seldom occur and pathologic alterations are minimal. The specific cause(s) of abomasal displacement has not been elucidated. However, the condition occurs most commonly in large high producing adult dairy cows during late pregnancy or immediately after parturition. Abomasal atony is considered to be the primary dysfunction in this condition.

Abomasal displacement is not usually fatal, but carcasses of affected animals are sometimes observed at abattoirs. The trapped abomasum (**between rumen and left abdominal wall**) contains varying amounts of fluid and gas. However, there is little or no interference with its blood supply. Therefore, passive congestion and its associated problems (**necrosis, etc.**) are not observed, but there is some interference with digestion and movement of ingesta out of the abomasum. Clinically, the animal is uncomfortable, the appetite drops, the amount of feces is reduced, and atony of the abomasum is noted. The majority of the cases are not urgent and some recover spontaneously.

5.11.5 DILATATION AND RUPTURE OF THE STOMACH:

Dilatation of the stomach can be caused by obstruction of the pylorus by foreign bodies or achalasia, by gross overeating, by drinking excessive amounts of water, etc. The condition occurs most commonly in horses and larger breeds of dogs. In the horse, ingestion of large amounts of fermentable grain is the usual cause of gastric dilatation. Subsequent to acute dilatation, rupture of the stomach may occur in the equine, but this is not common in other species. The rupture occurs most often along the greater curvature of the fundic region. Death due to shock often occurs before there is time for peritonitis to develop. Remember, the rupture may be complete or incomplete. If it is complete, it take about 6 hours after the rupture for peritonitis to be apparent to the naked eye. Clinically, gastric dilatation is manifested by severe pain and occasionally by projectile vomiting, in species capable of vomition. At necropsy, there is an antemortem tear in the stomach wall and its lumen may contain doughy foul smelling ingesta and cloudy fluid. A peritonitis may be observed if the rupture is complete.

5.11.6 ULCERATION OF THE STOMACH:

Gastric ulcerations occur in all animal species, but they are most frequent in farm animals (**cattle, swine and horses**) where they are usually traumatic or associated with erosive or ulcerative diseases (**bovine virus diarrhea, etc.**).

In calves, abomasal ulcers occur relatively commonly between two and three month of age. The cause(s) of abomasal ulcerations is not clear in either calves or adult cattle. Among the possible causes in both age groups are trauma, stress and diet (**high levels of carbohydrates**). The ulcerations are multiple, discrete and usually located in the pyloric region. In most cases, these lesions are clinically silent and heal uneventfully. Occasionally, the ulcers are complicated by secondary infections (**mycotic or bacterial**) or they may perforate and be responsible for ulcers.

Esophagogastric (**squamous portion of the stomach**) ulceration is a disease of major importance in swine. The cause(s) of this disease has/have not been clearly established. Evidence suggests dietary and management factors (**predominantly stress**) are capable of causing the disease, either alone or in combination. The condition is seen most commonly in confinement reared pigs which are receiving heavy grain rations. Clinically, the majority of pigs with this disease show no evidence of ill effects i.e., growth rates and feed conversion rates are normal. The clinical signs which may suggest the existence of this disease are related to hemorrhage from the ulcerations, provided the affected animal survives for 12 to 48 hours or longer. The usual clinical presentation is sudden death. If the animal has survived long enough, marked pallor, weakness, anorexia and black pasty feces may be seen. At necropsy, ulcerations are generally confined to the esophageal region of the stomach. Clotted and or nonclotted blood may be found in the lumen of the stomach and intestine. The earliest lesion consists of hyperkeratinization and denudation of the mucosa. Gastric ulcerations should be suspected in any pig presenting with extreme pallor and a history of sudden death. This condition must also be differentiated from hemorrhagic bowel syndrome and acute swine dysentery.

5.11.7 NEOPLASMS OF THE STOMACH:

This type of growth disturbance (**primary neoplasia**) is relatively uncommon in domestic animals. However, neoplasms with multicentric origin; e.g., lymphosarcoma, have been noted to occur with more frequency. Primary gastric neoplasms have been reported more frequently in dogs than any other domestic species.

5.11.8 MALIGNANT LYMPHOMA:

Involvement of the abomasum in lymphosarcoma of adult cattle is quite frequent and, with the exception of the lymph nodes, the abomasum is the most frequent organ involved. However, in calves this neoplasm is confined primarily to lymphoid organs and the abomasum is seldom involved. Grossly, the neoplastic growth may be diffuse or nodular. Malignant lymphomas may also involve the stomach of pigs, cats, and dogs.

5.11.9 SQUAMOUS CELL CARCINOMA:

Squamous cell carcinomas of the pars esophagea of the equine stomach are among the most frequent of the gastric carcinomas in animals, however, this neoplasm is still not considered to be common. This is a slow growing neoplasm which is usually well differentiated and largely confined to that portion of the stomach lined by squamous epithelium, but metastasis are known to occur.

5.11.10 ADENOCARCINOMA:

Adenocarcinoma of the stomach occurs primarily in dogs. They arise in the fundus and they may become sclerotic.

5.11.11 PARASITES OF THE STOMACH:

Gastrophilus infection is common in the stomach of horses. The adult flies are not parasitic. The important species of "**bots**" in this country are:

- (1) *Gastrophilus intestinalis* which is often referred to as the "**common bot**;" adults deposit their eggs upon hair of any part of the body, but especially on the forelegs and shoulders,
- (2) *G. hemorrhoidalis* which is often referred to as the "**nose**" or "**lip bot**;" its eggs are deposited on the lips and
- (3) *G. nasalis* which is commonly referred to as the "**throat bot**;" its eggs are deposited in the hair of the submaxillary region.

The larvae of all three species reach the mouth and remain for about a month. They subsequently pass into the stomach and attach to the mucosa. After a period of approximately 8 to 10 months, the larvae pass out in the feces and pupate in the soil. Clinical signs are mild and consist of nonspecific digestive disturbances. Each bot causes a small umbilicated ulcer at the site of its attachment to the stomach mucosa. However, the larvae are usually tolerated without recognizable effect. Gastric habronemiasis occurs in horses. The large stomach worms in the horse are:

- (1) *Habronema musca*,
- (2) *H. microstoma* and
- (3) *Draschia megastoma*.

D. megastoma occurs in tumor-like swellings in the stomach wall whereas the other species live free in the lumen.

House and stable flies act as intermediate hosts for these parasites. Of the equine gastric parasites, *Draschia* produces the most severe lesions. The tumor-like nodules it produces may rupture leading to fatal peritonitis. In addition, the large stomach worm larvae (**especially *Draschia***) may be associated with granulomatous skin lesions referred to as "**summer sores**."

The common stomach worms of cattle are *Hemonchus placei*, *Ostertagia ostertagia*, and *Trichostrongylus axei*. In sheep, *Hemonchus contortus*, *Ostertagia circumcincta*, and *T. axei* are the principal stomach worms. *Hemonchus* lives free in the lumen of the abomasum and feeds by piercing the mucosa to suck blood. Anemia is the most outstanding symptom. These parasites may be quite numerous and many small "bite" marks may be present over the abomasal mucosa. The infective larvae of *Trichostrongylus* and *ostertagia* burrow into the mucosa of the abomasum and develop to maturity in small nodules within the wall.

5.12 POST INSTRUCTIONAL SELF EXAMINATION

PART A: UPPER ALIMENTARY TRACT

Based on the knowledge and skills gained from study of the upper alimentary tract, each student should be able to provide appropriate answers for the following **questions**. Please review the instructional material as needed.

Questions

1. What are the anatomic components of the digestive system?
2. What factors protect the alimentary tract from infections, indigestion, etc.
3. What factors predisposes the alimentary tract to infections?
4. What term is used to describe each of the following:

_____ Inflammation of the oral cavity

_____ Abnormal prolongation of the mandible

_____ Inflammation of the tongue

_____ A defect or communication between the oral and nasal cavities

_____ A congenital shortening of the maxilla

_____ Inflammation of the hard palate

_____ A congenital shortening of the mandible

_____ Inflammation of the gums

_____ A harelip or split upper lip

_____ Inflammation of the pharynx

_____ Hypoplasia of tooth enamel

_____ Inflammation of the salivary glands

_____ Improper alignment of the grinding surfaces of opposing teeth

_____ Inflammation of the salivary ducts

_____ An accumulation of salivary secretions in the sublingual area

_____ Excessive secretion of saliva

_____ Calcified masses of bacteria and desquamated cells on the teeth

	Contagious ecthyma in man
	Reduced secretion of saliva
	Cystic dilatation and the accumulation of saliva in the salivary duct beneath the tongue
	Calculi in the salivary glands
	Loss of appetite
	Diffuse dilatation of the esophagus
	Failure of the cardiac sphincter to relax during the act of swallowing
	Bloat due to failure of the eructation of free gases
	Bloat characterized by the presence of foams mixed with the ingesta

5. Briefly discuss the causes and lesions associated with stomatitis in animals.
6. What is a so-called noma?
7. What is phlegmonous inflammation?
8. What is the significance of finding food in the mouth of a carcass?
9. In what animal species would you expect to find inflammatory changes, etc., associated with a diverticulum located in the posterior wall of the pharynx?
10. What congenital anomalies of the oral cavity result from failure of integrated growth and fusion?
11. Based on the developmental process, distinguish palatoschisis from chiloschisis.
12. Under what circumstances is brachygnathia superior considered to be normal? Explain.
13. In what animal species would you expect to observe a high incidence of brachygnathia inferior?
14. In what animal species would you expect to observe a high incidence of

prognathism?

15. Why would it be difficult on clinical examination to distinguish between prognathism and brachygnathia superior?

16. Describe the mechanisms by which teeth and bones are discolored by tetracyclines.

17. What portion of a tooth (enamel, etc.) is discolored by tetracycline?

18. Under what circumstances would you expect deciduous and/or permanent teeth to be discolored subsequent to dosages of tetracyclines?

19. Why would you expect teeth discolored by tetracyclines to fluoresce when exposed to ultraviolet rays?

20. Why would you expect a high incidence of dental malocclusion in rodents maintained as pets?

21. What are dental caries? Under what circumstances would you expect caries to develop?

22. What is odontodystrophy? Discuss the circumstances under which you would expect this condition to occur.

23. What is the fundamental cause of congenital porphyria. Would you expect the resulting pigments to accumulate in dentin or in the enamel of affected teeth?

24. How would you make a diagnosis of congenital porphyria in a two day old Holstein? What is "pink-tooth"?

25. Under what circumstances would you expect skin lesions to be associated with congenital porphyria? Discuss the effects of congenital porphyria on the health of an affected animal.

26. Discuss the manner by which the canine distemper virus is capable of causing defective tooth enamel production.

27. What is the dental formula for a 2 year old chicken?

28. How would you differentiate between a squamous cell carcinoma of the oral cavity and a papilloma of the oral cavity upon gross and microscopic examinations?

29. Discuss canine oral papillomas on the basis of etiologic agent, clinical signs, immunity, gross findings, microscopic lesions, prognosis and treatment.

30. Name the important neoplasms or neoplastic-like lesions derived from dental germ cells.

31. Name an invasive neoplasm derived from periodontal squamous epithelial cells.

32. What is an epulis? In what species does this growth disturbance commonly occur?

33. What is a hamartoma?

34. What is a choristoma?

35. How would you characterize malignant catarrhal fever? What virus is responsible for this disease?

36. In what domestic animal species would you expect to observe cases of malignant catarrhal fever? What is the interrelationship between cattle and sheep in the transmission of this disease?

37. Briefly, describe the gross and microscopic lesions of malignant catarrhal fever.

38. What significant lesion(s) are expected in the small intestine of a cow with malignant catarrhal fever? What is considered to be the basic and characteristic lesion of this disease?

39. Briefly characterize the disease known as actinobacillosis?

40. For actinobacillosis, please answer the following:

- -- Name the etiologic agent.
- -- What is the favorite body site for infection?
- -- What is so-called "wooden tongue?"
- -- What are the characteristic lesions in affected sheep?
- -- Distinguish this disease from actinomycosis.
- -- Give a likely pathogenesis for the commonly encountered tongue lesions.
- -- Give the pertinent gross and microscopic lesions. What are so-called "sulfur granules."

41. Papular stomatitis and contagious ecthyma are examples of specific diseases that affect the oral cavity. Briefly discuss the differentiating features and similarities of these two diseases.

42. Discuss the significance of papular stomatitis in animals. Give the likely etiologic agent.

43. Briefly describe the gross and microscopic lesions of papular stomatitis.

44. Briefly characterize the disease known as contagious ecthyma. What animal species are usually affected?

45. Discuss the occurrence of inclusion bodies in papular stomatitis and in contagious ecthyma.

46. What is a vesicular disease?

47. Briefly discuss the differences and similarities between foot and mouth disease, vesicular stomatitis and vesicular exanthema on the basis of:

- -- Etiologic agents involved.
- -- Animal species affected.
- -- Importance as a public health problem.
- -- Threat to the livestock industry of this country.
- -- Clinical signs.
- -- Gross lesions.
- -- Methods employed to diagnose them.

48. Which of the vesicular stomatitides is characterized by each of the following:

- -- Caused by an enterovirus.
- -- Caused by a rhabdovirus.
- -- Caused by a calicivirus.
- -- Characterized by acute myocarditis in calves.
- -- Occurs in "cloven-footed" animals.
- -- Occurs primarily in horses.
- -- Occurs primarily in swine.
- -- Horses are resistant.

49. Why is it important for veterinarians to be aware of the pathogens and or specialized defensive/offensive adaptations associated with the oral cavity of various animal species?

50. In examining the mouth of a snake, what factors should a veterinarian be aware of?

51. Discuss Monkey "B" on the basis of clinical signs, lesions, and morbidity/mortality which can be seen in monkeys and in man.

52. In what way would you expect the marmoset to respond differently from other monkeys exposed to herpes virus simiae?

- 53. Why is it important for veterinarians and others to handle apparently normal monkeys with care?**
- 54. Name a cytomegalic virus which has an affinity for the salivary glands.**
- 55. Name two diseases and/or conditions in which well-defined erosions and/or ulcers may be found in the esophagus.**
- 56. What does the term "choke" refer to?**
- 57. What is the interrelationship between esophageal injury (ulcers, etc.) and stenosis of the esophageal lumen?**
- 58. A 12 year old Quarter horse swallowed a corncob which lodged in the distal one-third of the esophagus and severely damaged the esophageal mucosa. After a period of time, the corncob passed into the stomach, but subsequently the horse had difficulty in deglutition. Give the pathogenesis of the condition described above.**
- 59. Why would you expect serious complications to occur in cattle subsequent to stenosis of the esophagus (explain)?**
- 60. Why would you distinguish between megaesophagus and achalasia?**
- 61. Why would you expect foreign body pneumonia to be a complication associated with "choke"?**
- 62. What is the interrelationship between achalasia and megaesophagus?**
- 63. What effects would megaesophagus have on the health of a 6 year old Coonhound?**
- 64. What is spirocercosis? What type of esophageal lesions would you expect to be associated with *Spirocerca lupi*. In What portion of the esophagus would you expect to find lesions? What lesions would you expect *Spirocerca lupi* to cause in the aorta of affected dogs?**
- 65. What parasite tends to embed in a "zig-zag fashion" within the esophageal mucosa and submucosa of cattle?**
- 66. Cattle, sheep and goats have four stomach compartments. Would you expect to find lobulated kidneys in each of these animals?**
- 67. What is bloat? Distinguish between primary and secondary bloat.**
- 68. What factors are usually responsible for primary bloat?**

- 69. What factors are usually responsible for secondary bloat?**
- 70. Under what circumstances is primary bloat rather easily relieved via a stomach tube?**
- 71. Under what circumstances is primary bloat very difficult to relieve or treat?**
- 72. What clinical signs would you expect to observe in an animal with bloat?**
- 73. Briefly explain how toxemia and respiratory distress develop subsequent to bloating.**
- 74. How would you describe the processes referred to as "belching" and rumination?**
- 75. What is the interrelationship between overeating, intoxication, and atony of the rumen?**
- 76. Discuss the events that occur when a cow dies subsequent to overeating green corn.**
- 77. What is the interrelationship between rumenitis and liver abscesses?**
- 78. List the major causes of rumenitis.**
- 79. What precautions should be taken when cattle are first placed on a fattening ration in order to minimize the incidence of rumenitis?**
- 80. What secondary bacterium is oftentimes associated with rumenitis?**
- 81. What would you expect to happen if a liver abscess ruptured into the portal vein?**
- 82. A large nail was swallowed by a six year old Holstein and traumatic pericarditis developed subsequently. Give the pathogenesis of this condition.**
- 83. Under what circumstances would you expect the stomach to rupture?**
- 84. Overdistention of the true stomach with gas occurs most frequently in what animal species?**
- 85. Why would an accumulation of gas in the stomach of a horse pose a serious problem for the affected animal? What portion of the stomach is usually torn?**

86. What complications may occur if a stomach rupture is complete?

87. Following complete rupture of the stomach, how long will it take before the resulting peritonitis becomes apparent to the naked eye?

88. A six year old Quarter horse died approximately ten hours after complete rupture of the stomach. At necropsy, how would you distinguish this antemortem rupture from a postmortem rupture?

89. A four year-old Quarter horse died approximately one hour following complete rupture of the stomach. How would-you distinguish this antemortem rupture from a postmortem rupture at the time of necropsy.

90. What are piloconcretions and phytoconcretions?

91. What is gastric torsion? What animals are most commonly affected?

92. What clinical signs occur in dogs with torsion of the stomach?

93. What factors predispose dogs to gastric torsion?

94. What significant gross lesions would you expect to find in a dog with torsion of the stomach?

95. What effects would stomach rotations of less than 180 degrees have on the affected dog?

96. What effects would stomach rotations of more than 180 degrees have on the affected dog?

97. Why is the statement oftentimes made that "left displacement of the abomasum is more a clinical and surgical problem than one related directly to pathology?"

98. What is displacement of the abomasum? Describe the normal position of the abomasum and the location of an abomasum which has been displaced.

99. Under what circumstances would you expect displacement of the abomasum to occur.

100. What clinical signs and gross alterations would you expect to be associated with a displaced abomasum?

101. What are gastric ulcerations and in what animal species would you expect this condition to occur most frequently?

102. Name the regions of the pig stomach. How does the esophageal

portion structurally differ from the glandular region?

103. Discuss the possible causes of ulcers involving the esophageal portion of the pig stomach.

104. What complications may occur in pigs subsequent to a progressive gastric ulcer?

105. Based on the appearance of the feces, how would you distinguish between hemorrhage that occurred subsequent to a gastric ulcer and hemorrhage associated with ulcers in the colon?

106. Distinguish between an ulcer and an erosion.

107. Discuss ulcers in calves based on incidence, causes, location, secondary invaders, prognosis, and gross lesions.

108. Discuss the clinical signs and lesions associated with gastrophilus infection in horses.

109. Name the important species of "bots."

110. What lesions would you expect to be associated with adult *Draschia megastoma* infection in the horse. What lesions may be associated with *Habronema* larvae?

111. Name the important "stomach worms" in the horse.

112. Name the important "stomach worms" in sheep.

113. Name the important "stomach worms" in cattle.

114. What lesions would you expect to find in the stomach of sheep with severe *Hemonchus contortus* infection?

115. Describe the events that occur in the development of generalized edema in a sheep with severe *Hemonchus contortus* infection.

116. What type anemia would you expect to occur in sheep with severe chronic *Hemonchosis*?

117. What abomasal lesions would you expect to be associated with *trichostrongylus* and *ostertagia* infections?

118. What is a diaphragmatic hernia? What are the identifying features of a hernia?

119. Why would you expect a rather large volume of abdominal viscera to

herniate through a relative small tear in the diaphragm?

120. Discuss the clinical signs of "choke" as seen in both cows and horses.

121. At necropsy, what important circulatory changes would you expect to be associated with a displaced abomasum?

122. Why is so-called "cat scratch fever" important to the veterinarians?

123. Name all parts of the alimentary tract of the horse (including all flexures).

124. Briefly. describe the causes, clinical signs and lesions associated with rumen overload in cattle.

5.13 SLIDE SESSION 5

PART A

UPPER ALIMENTARY TRACT

SLIDE 1: BOVINE MOUTH: NECROTIC STOMATITIS - The owner of this calf stored a caustic chemical in an old barn in which several young calves were housed. This chemical (an alkali) was allowed to leak into the water supply used by these animals. All of the calves died or had to be euthanized because of their debilitated conditions. Oral lesions were quite obvious on clinical examination and at necropsy. However, problems were encountered in attempts to determine the specific etiologic agent(s) and their source. At necropsy, significant lesions consisted of a diffuse necrotic stomatitis and esophagitis with necrotic lesions in portions of the rumen. In this slide, observe the necrotic changes involving the lips, gums, soft palate, and hard palate. Would you use the term "gangrene" to describe the changes? Can you define the following: lampas, tracheitis? What happened to the upper incisor teeth in this calf? What clinical signs would you expect this calf to exhibit? Name at least 8 specific diseases in which stomatitis is a prominent feature.

SLIDE 2: BOVINE TONGUE: NECROTIC GLOSSITIS - This tongue is from the calf denoted in slide #1; observe the discolored, cooked appearance of the tongue, pharyngeal, and laryngeal regions. What effects would such lesions have on the health of this calf?

SLIDE 3: BOVINE ESOPHAGUS: NECROTIC ESOPHAGITIS - Observe the extensive destruction of the esophageal mucosa with the accumulation of debris. This slide is from the same calf denoted in slides #1 and #2. In addition to the lesions observed in these slides, this calf had a severe necrotic, foul-smelling pneumonia which was confined primarily to the left lung. Give a likely pathogenesis for the pneumonic condition.

SLIDE 4: BOVINE RUMEN: NECROTIC RUMENITIS - Observe the diffuse necrosis in the reticulum and patchy areas of necrosis in the rumen. Can you give a reason(s) for this distribution of the lesions?

SLIDE 5: BOVINE TONGUE: GLOSSITIS (ULCERATIVE) - Observe the ulcerative lesion along the middle one-third of the tongue. Please give a likely pathogenesis for this condition. Would you consider "trauma" or an "infectious agent" as the most likely cause? How would you define an ulcer?

SLIDE 6: BOVINE TONGUE: GLOSSITIS (ULCERATIVE) - The tongue of this steer was traumatized during his attempts to eat berries that had grown along a barbed wire fence. Observe the well defined ulcerative lesions along the tip of the tongue.

SLIDE 7: EQUINE TONGUE: GLOSSITIS (HEALED ULCERS) - Observe the defects along the anterior one-third of the tongue. These are healed ulcerative lesions. Actually this horse died subsequent to enteric salmonellosis which was not related to the tongue lesions. What are some possible causes of the tongue lesions? What is a chronic inflammatory reaction? Is this an example of wound healing by "first intention"? Explain. What is wound healing by "second intention"? Is this an example of keloid formation?

SLIDE 8: BOVINE TONGUE: ULCERATIVE GLOSSITIS - This tongue is from a steer that had a specific infectious disease known as "bovine virus diarrhea." Observe the well defined ulcerative lesions over the dorsal surface of the tongue. Distinguish an "ulcer" from an "erosion." Is the larger ulcer noted on the tongue "acute" or "chronic?" Explain. Would you expect these ulcerative lesions to heal by first or second intention?

SLIDE 9: BOVINE TONGUE: GLOSSITIS - This cow had a disease commonly referred to as "malignant catarrhal fever." Note the destruction of epithelium and the rather intense red color. This cow had difficulty eating and drinking. Profuse salivation was noted prior to death. What precautions should veterinary medical students and clinicians take when examining the mouth of sick animals?

SLIDE 10: CANINE ORAL MUCOUS MEMBRANES: ICTERUS - This 15 year old dog had several well defined calculi within the lumen of the common bile duct. Subsequent to the presence of these calculi the mucous membranes and some other tissues became discolored as observed in this slide. What color are the mucous membranes? What type icterus would you expect in the dog? What substance is responsible for the discolored gums? What substance(s) is/are responsible for discolored mucous membranes when there is either excessive intravascular hemolysis and/or severe liver damage? Under what circumstances would you expect to observe pale or cyanotic oral mucous membranes in an animal? What disease is oftentimes characterized by petechial hemorrhages on the ventral aspect of the tongue?

SLIDE 11: OVINE HEAD/FACE: MULTIPLE CONGENITAL DEFECTS - This stillborn ovine fetus was submitted to the necropsy laboratory with multiple congenital defects. In this slide, note the protruded lower jaw, thickened or hyperplastic gums, absence of maxilla, distorted upper lip, and the single eye. In addition, a condition referred to "prosencephaly" was diagnosed at necropsy (refer to the CNS section of this syllabus). What is a cyclops? What factors may be responsible for congenital defects such as those present in this fetus? Distinguish "abortion" from "stillborn." Distinguish "hydranencephaly" from "hydrocephalus."

SLIDE 12: BOVINE MOUTH: PALATOSCHISIS (CLEFT PALATE) - This bovine fetus was aborted. Observe the defective maxillary process which resulted in a communication between the oral and nasal cavities. Laboratory findings were positive for bovine virus diarrhea. In addition to the defect observed in this slide, there was hypoplasia of the cerebellum. Distinguish between hypoplasia and atrophy. How would the cleft palate affect the health of this calf? In your opinion, is there a relationship between the cleft palate and hypoplastic cerebellum in this calf? Name a viral agent that may be responsible for cerebellar hypoplasia in the cat.

SLIDE 13: EQUINE FACE: BRACHYGNATHIA SUPERIOR - Observe the shortened maxilla. Both the mandible and maxilla are deviated to one side. How would you differentiate this condition from prognathism?

SLIDE 14: EQUINE FACE: BRACHYGNATHIA SUPERIOR - This is the same foal denoted in slide #13; however, the shortened maxilla is more pronounced. How would you differentiate between brachygnathia inferior and the condition observed in this slide? Please define the term used to refer to a "split upper lip."

SLIDE 15: CHICKEN MOUTH: AVIAN LEUKOSIS - Avian leukosis is a malignancy of lymphoid cells caused by a type C RNA virus which belongs to the family retroviridae in the subfamily oncovirinae. Initially, neoplastic transformation of lymphocytes occurs within lymphoid follicles of the Bursa of Fabricius. Subsequently, neoplastic lymphocytes metastasize to other organs and tissues. Avian leukosis must be distinguished from "Marek disease" which is caused by a herpesvirus. In this slide, observe the well defined neoplastic growth located in the mouth. At necropsy, the carcass was thin, emaciated, and dehydrated.

SLIDE 16: CANINE MOUTH: ORAL PAPILLOMATOSIS - Oral papillo-matosis is a "self-limiting," contagious, benign neoplastic disease caused by a virus. Immunity is conferred following recovery. In this slide, observe the cauliflower-like growths located within and around the mouth.

SLIDE 17: CANINE MOUTH: ORAL PAPILLOMATOSIS - Observe the well defined tumor masses located on the lips of this dog. How would you differentiate a benign neoplasm from a malignant neoplasm?

SLIDE 18: RABBIT TEETH: DENTAL MALOCCLUSION - Observe the elongated teeth as well as the improper alignment of opposing teeth. This animal had difficulty eating and became quite emaciated prior to euthanasia. What clinical signs would you expect this rabbit to exhibit? Under which circumstances would you expect rodents and lagomorphs to exhibit elongated teeth? How is the normal length of teeth maintained in rodents?

SLIDE 19: CANINE TEETH/GUMS: DENTAL CALCULI - Observe the brownish

material (calculi) on the premolar teeth as well as on the neck of the canine tooth. What factors are responsible for the development of tartar on teeth? Would you consider the oral mucous membranes to be normal or abnormal?

SLIDE 20: BOVINE TEETH/BONES: CONGENITAL PORPHYRIA - This 4 day old calf was submitted to the Necropsy Laboratory with a history of severe diarrhea. After the necropsy had been performed and appropriate tissues collected for microbiologic examination, an astute 2nd year student noticed a marked brownish discoloration of the ribs. Further examination revealed a distinct brownish discoloration of all bones as well as the teeth. A tentative diagnosis of "congenital porphyria" was made. The astute 2nd year student was "pleased" and "happy" until he failed to recognize the disease on a quiz given a few days later.

SLIDE 21: BOVINE TEETH/BONES: CONGENITAL PORPHYRIA - Observe the pink to red discoloration of teeth and bones. These specimens were exposed to ultraviolet light when the photo was taken. Give a likely etiologic mechanism as well as a pathogenesis for this condition. Give a probable reasons for the development of skin lesions if this calf had been exposed to intense sunlight for a prolonged period of time.

SLIDE 22: BOVINE TEETH: CHRONIC FLUOROSIS - In this slide, observe the discoloration of the lateral incisor teeth. The middle incisors had already erupted when the animal was exposed to fluorine. Chronic fluorosis (excessive fluorine) is characterized by involvement of the teeth, bones, and kidneys. Teeth are characterized by pigmentation of the enamel which is abnormally soft. Dental lesions develop only if the fluorosis is present during the time the tooth is being formed.

SLIDE 23: EQUINE MOUTH: ODONTOMA - Observe the large tumor-like mass that's in the mouth of this horse. Explain the origin of the tumor.

SLIDE 24: CHICKEN MOUTH: VITAMIN A DEFICIENCY - Have you heard the old expression "scarce as hen teeth? Can you give a reason for the popularity of this expression? How would you explain the small "slit" in the roof of this bird's mouth?

SLIDE 25: BOVINE HEAD: MALIGNANT CATARRHAL FEVER - This is one of several cows housed with a group of sheep and goats. Observe the exudate oozing from the eyes as well as the opaque cornea. Also, an erosive stomatitis developed. What is the etiologic agent responsible for this condition? What significant lesions would you expect to develop in other organs and tissues of this animal. Discuss the occurrence of this disease in sheep and goats. What are the major clinical forms of this disease?

SLIDE 26: BOVINE ORAL MUCOUS MEMBRANES: MALIGNANT CATARRHAL FEVER - This is the same cow denoted in slide #25. Note the oral erosions with loss of surface epithelium as well as reddening of the mucosa. Briefly, discuss the gross lesions associated with this disease.

SLIDE 27. BOVINE TONGUE: MALIGNANT CATARRHAL FEVER - Observe the erosions and/or inflammatory changes over the dorsum of the tongue. Would the animal have had difficulty eating. What other diseases have you encountered that are characterized by oral erosions? What significant gross lesion(s) would you expect to find in the intestinal tract of this cow? What are Peyer's patches?

SLIDE 28: BOVINE KIDNEY (MICRO): MALIGNANT CATARRHAL FEVER - In this microscopic kidney section, observe the vascular change which is characteristic of this disease. Note the mononuclear cells located within the vessels on this slide. What is considered to be the identifying microscopic lesion in cases of malignant catarrhal fever? Define the following terms: vasculitis, arteritis, perivascular cuffing.

SLIDE 29: BOVINE BRAIN (MICRO): MALIGNANT CATARRHAL FEVER - In malignant catarrhal fever, central nervous system lesions tend to develop during the terminal stages. Observe the well-defined arteritis characterized by mononuclear cells distributed throughout the vessel wall. Remember, the inflammatory cells may accumulate in perivascular spaces.

SLIDE 30: BOVINE TONGUE: ACTINOBACILLOSIS - This is a section through the tongue of a cow with actinobacillosis, note the upper mucosal surface and the nodular necrotic tissue. Fibrosis (pale tissue) is a prominent feature. Why is this condition referred to as "wooden tongue?" Name the etiologic agent. What lesions would you expect in a sheep with this condition? What clinical signs would be expected in sheep? Briefly describe the microscopic changes which

would be expected in either cows or sheep.

SLIDE 31: BOVINE LARYNGEAL REGION: ACTINOBACILLOSIS - Observe the swollen, hemorrhagic and distorted tissue in the laryngeal region of this cow. Remember, all soft tissues in the oral cavity and adjacent structures are susceptible to infection.

SLIDE 32: BOVINE MOUTH: PAPULAR STOMATITIS - Observe the erythematous and slightly raised lesions in the mouth of this calf. Prior to death, this calf was thin and dehydrated. A diagnosis of intestinal parasitism was also made. Give the exact location of the oral lesions observed in this slide. What is the usual prognosis for a calf with this disease? Name the likely etiologic agent.

SLIDE 33: BOVINE TONGUE: PAPULAR STOMATITIS - Observe the well defined lesions on the tongue which are in the process of healing. How would you define the following terms: macule, papule, vesicle, pustule, ulcer and erosion.

SLIDE 34: BOVINE LIPS: PAPULAR STOMATITIS - Observe the papular lesions on the lips (see arrow). What term is used to refer to inflammation of the lips? What characteristic microscopic lesions are associated with this disease?

SLIDE 35: OVINE LIPS: CONTAGIOUS ECTHYMA - Observe the lip lesions which are covered with scabs. A veterinary medical student had an opportunity to examine the mouth of this sheep before the specific diagnosis was established. What is the potential public health significance of this clinical examination? Give the likely etiologic agent. Briefly discuss the development of gross lesions in this disease. What site within cells would you expect to find inclusion bodies? What is "pseudocow-pox?" What animal species may be affected with this disease?

SLIDE 36: HUMAN FINGER (MILKER'S NODULES): CONTAGIOUS ECTHYMA - The lesions observed on the fingers of this young man developed following contact with the sheep denoted in slide #35. This photo was taken 15 days after infection. Observe the well defined pustules and areas of hyperemia. How would you differentiate a pustule from an abscess? How would you classify the type of necrosis observed in this slide?

SLIDE 37: BOVINE FACE/MOUTH: FOOT AND MOUTH DISEASE - This is an adult cow with foot and mouth disease. Observe the salivation associated with oral lesions. Remember, foot and mouth disease, vesicular stomatitis, and vesicular exanthema of swine cannot be distinguished on the basis of clinical signs, gross or histologic lesions. What virus and/or strains are responsible for this disease? What characteristic lesions are expected in the early and late stages? Briefly, discuss the clinical signs expected.

SLIDE 38: BOVINE MOUTH: FOOT AND MOUTH DISEASE - Observe the well defined healing ulcerative lesions involving the oral mucous membranes. Approximately how long does it take for oral lesions to heal in case of foot and mouth disease? Discuss the occurrence of foot and mouth disease in young calves, sheep, goats and swine.

SLIDE 39: BOVINE TONGUE: FOOT AND MOUTH DISEASE - Observe the healing ulcerative lesions on the tongue of this cow. How would you distinguish foot and mouth disease from malignant catarrhal fever on the basis of lesions that one may observe? What syndrome would you expect in horses exposed to the foot and mouth disease virus?

SLIDE 40: PORCINE SNOOT: VESICULAR STOMATITIS - In this pig, observe the well defined vesicular lesions on the snout. This is an earlier stage of vesicular stomatitis. What agent is responsible for vesicular stomatitis? What animal species is most commonly affected with this disease? Briefly, discuss the occurrence of vesicular stomatitis and foot and mouth disease in the USA.

SLIDE 41: PORCINE FOOT: VESICULAR STOMATITIS - Observe the necrotic and ulcerative lesions involving the foot of this pig. The animal encountered considerable difficulty in attempting to walk.

SLIDE 42: PORCINE FOOT: VESICULAR EXANTHEMA - Note the large vesicle on the foot of this pig. It should be remembered that the gross lesions of foot and mouth disease, vesicular stomatitis, and vesicular exanthema are very similar.

SLIDE 43: BOVINE ESOPHAGUS: ESOPHAGITIS (ULCERATIVE) - Esophagitis in

this steer was associated with the virus of bovine virus diarrhea. Observe the focal reddened areas over the mucosal surface. What lesions would you expect to find in the oral cavity of this steer?

SLIDE 44: CANINE ESOPHAGUS: FOREIGN BODY IN THE ESOPHAGUS - Observe the staple located in the lumen of the esophagus. Actually, this staple perforated the esophageal wall at the level of the thoracic inlet. A tentative diagnosis was based upon clinical examination (palpation etc.) and confirmed via x-ray and necropsy. What complications can arise subsequent to the condition observed in this slide? Would you refer to this condition as choke?

SLIDE 45: OVINE ESOPHAGUS: IMPACTION OF THE ESOPHAGUS - Observe the rather dry material in the lumen of the esophagus. A foreign body (corn cob) was found at the level of the esophageal opening into the stomach. Give a likely pathogenesis for this condition.

SLIDE 46: EQUINE ESOPHAGUS: ULCERATION AND STENOSIS - Observe the well-defined chronic ulcers in the lumen of the esophagus. Apparently, a foreign body damaged the mucosa. However, no foreign body was found at the time of necropsy. Prior to death, the horse developed a severe gangrenous pneumonia. How do you know that the lesions in this slide are chronic? Can you locate the anterior and posterior ends of the esophagus? What portion of the esophagus in this horse would you expect to be dilated? Discuss the development of the foreign body pneumonia in this horse.

SLIDE 47: EQUINE ESOPHAGUS: PERFORATION OF THE ESOPHAGUS - Observe the two well-defined puncture wounds. Apparently, the lesions were caused by a sharp object(s). Also, note the necrosis involving the esophageal mucosa. At necropsy, considerable exudate and ingesta were found in the subcutaneous tissues along the neck.

SLIDE 48: FELINE THORACIC CAVITY: FIBRINOUS PLEURITIS - The fibrinous pleuritis in this cat was associated with perforation of the esophagus within the thoracic cavity. Note the accumulated exudate; the lungs were atelectatic but pneumonia was not a significant feature.

SLIDE 49: PARROT CROP: CONCREMENT IN THE CROP LUMEN - Observe the

large solid mass in the crop lumen. This bird died subsequent to extreme emaciation. What is a "crop?" How would the mass observed in this slide affect the health of this parrot?

SLIDE 50: CANINE ESOPHAGUS: MEGAESOPHAGUS - This is an unopened esophagus from a dog with megaesophagus. Compare the size of the dilated esophagus with that of the stomach. This young dog was always hungry, but eating was followed by vomiting. Progressive weight loss occurred and aspiration pneumonia developed.

SLIDE 51: CANINE ESOPHAGUS: MEGAESOPHAGUS - This is the same esophagus denoted in slide #50. Observe the dilated and flaccid esophageal wall. Hemorrhages noted in the mucosa were associated with stagnated ingesta. What is achalasia? Can you differentiate achalasia from megaesophagus?

SLIDE 52: BOVINE ESOPHAGUS: PAPILLOMAS - Observe the pale white papillomas attached to the esophageal mucosa. These viral-induced benign tumors are usually incidental findings. In addition, there are rather well-defined ulcers in the esophagus.

SLIDE 53: CANINE ESOPHAGUS: SPIROCERCOSIS - Locate the esophagus and the stomach and observe the reddish nematodes as well as the well-defined nodules in the distal portion of the esophagus.

SLIDE 54: CANINE ESOPHAGUS: SPIROCERCOSIS - This microscopic section of the esophagus is characterized by a severe inflammatory reaction associated with the presence of *Spirocerca lupi*. Observe cross sections of the parasite.

SLIDE 55: CANINE ESOPHAGUS: SPIROCERCOSIS - Observe the rather characteristic ova of *Spirocerca*.

SLIDE 56: CHICKEN CROP: MYCOTIC ESOPHAGITIS (THRUSH) - Observe the normal and abnormal areas in the crop which should give you an appreciation

for the marked thickening of the mucosa associated with this disease. Rather well defined pseudomembranes are associated with the thickened wall.

SLIDE 57: CHICKEN CROP: MYCOTIC ESOPHAGITIS (THRUSH) - There are numerous fungal organisms (*Candida albicans*) in this microscopic section from a crop lesion. Thrush is an important mycotic disease of the digestive tract of poultry caused by *Candida albicans*. Lesions are frequently found in the esophagus, crop, and mouth which consist of a thickened mucosa covered by raised whitish pseudomembranes. The organisms may also be found in the mouth of piglets suffering from debilitating diseases.

SLIDE 58: BOVINE BODY: RUMINAL TYMPANY - Observe the recumbent steer with the outstretched legs. The ration in this feed lot consisted of large amounts of grain and little roughage. How would you distinguish postmortem bloat from antemortem bloat at the time of necropsy? What are the types of bloat? How would you distinguish primary ruminal bloat from secondary ruminal bloat? Briefly, discuss the cause of primary and secondary bloat.

SLIDE 59: BOVINE ESOPHAGUS: RUMINAL TYMPANY - This is the esophagus from a steer that was bloated. Note the pale as well as the congested portions. How would you explain the esophageal alterations observed in this slide? Can primary bloat be distinguished from secondary bloat on the basis of the esophageal lesions? What vascular changes would you expect to observe in the lungs of this steer? Briefly, discuss the gross alterations expected in cases of bloat.

SLIDE 60: BOVINE RETICULUM: TRAUMATIC RETICULITIS - This 12 year old Holstein ingested a piece of wire that penetrated the reticular wall. Observe the wire as well as the collection of fibrin over the serosal surface. What organs could this foreign body penetrate as it protrudes through the wall of the reticulum? Briefly, give a pathogenesis for the condition observed in this slide. What is traumatic pericarditis? What factors may predispose an animal to develop traumatic reticulitis?

SLIDE 61: BOVINE HEART: TRAUMATIC RETICULITIS/PERICARDITIS - This is the heart from the cow denoted in slide #60. The wire that penetrated the wall of the reticulum made its way to the heart. Observe the thickened pericardial sac

as well as the heavy accumulation of exudate (fibrin) over the epicardium. What effects would the lesions observed in this slide have on normal heart function? What changes would be expected in the liver of this cow?

SLIDE 62: BOVINE HEART: TRAUMATIC RETICULITIS/PERICARDITIS - Observe the massive amounts of purulent exudate present in the pericardial sac. Apparently the piece of wire that penetrated the reticulum carried a few pyogenic bacteria along with it.

SLIDE 63: SEAL STOMACH: CATARRHAL GASTRITIS - Observe the markedly reddened mucosa and the physical agents responsible for this response. In addition, there was increased mucous production which was washed away in order to obtain a better photograph. A total of 58 cents was found in the stomach of this seal. No evidence of obstruction was noted. What is a catarrhal gastritis? What is a secondary and/or primary gastritis? On the basis of gross and microscopic lesions, how would you distinguish between a physiologic active hyperemic of the stomach and the hyperemia which accompanies an acute catarrhal gastritis?

SLIDE 64: CANINE STOMACH: ULCERATIVE GASTRITIS - Observe the reddened mucosa as well as the discrete ulcerative lesions. This 10 year old Coonhound was submitted to the clinic with severe uremia which developed subsequent to a chronic kidney disease. What is uremia? What is the interrelationship between the kidney lesions and the gastric ulcers? Explain the mechanism(s) by which calcium salts may be deposited in various organs and tissues of this dog. Would you consider the gastric ulcerations observed in this dog to be primary or secondary?

SLIDE 65: BOVINE ABOMASUM: HEMORRHAGIC GASTRITIS - Observe the many tiny hemorrhages over the mucosa of the abomasum. This calf had scours and was treated for several days with very large doses of oral antibiotics. The antibiotics apparently altered the gastrointestinal flora.

SLIDE 66: CANINE STOMACH: GASTRIC FOREIGN BODY - This young dog had numerous roundworms in its small intestine and its owner also noted that its appetite was perverted. Observe the large amount of grass concentrated in the pyloric portion. Is there evidence of inflammation in this slide? How would this foreign material interfere with the normal digestive processes?

SLIDE 67: CANINE STOMACH: GASTRIC FOREIGN BODY - Observe the small rubber ball that was lodged in the pyloric portion of the stomach; this is referred to as a high obstruction. Animals with this type of obstruction may die rather acutely. Please refer to slide #68.

SLIDE 68: CANINE STOMACH: GASTRIC FOREIGN BODY - This is a close up view of slide #67. Observe the rubber ball as well as the site of obstruction (demarcated by a rather well defined ulcer). Depression and vomition were prominent clinical signs.

SLIDE 69: BOVINE ABOMASUM: GASTRIC FOREIGN BODY - Observe the piece of wire protruding from the abomasum. Note the exudate over the serosal surface. Is the abomasum the site you would normally expect to find a piece of swallowed wire? Why would you or why wouldn't you expect this? What type of exudate do you observe over the serosal surface of the abomasum? What complications would you expect to be associated with the condition observed in this slide?

SLIDE 70: CANINE STOMACH: TORSION OF THE STOMACH - At necropsy, the stomach of this dog was twisted more than 180 degrees. In this slide, you should suspect gastric torsion based on the markedly dilated stomach and the size and position of the spleen. Pretend that this is your German Shepherd. What history would you most likely give the attending veterinarian? How would you explain the size and the position of the spleen in this slide? Give a likely reason(s) for death of this dog. How would you explain the over distended stomach? What is gastric torsion? Why would you expect to find blood-tinged fluid in the stomach lumen? What clinical signs would you expect this dog to exhibit?

SLIDE 71: CANINE STOMACH: TORSION OF THE STOMACH - Observe the greatly distended and twisted stomach. This 10 year old German Shepherd ate a very large meal, drank lots of water, and went outside to play. Several hours later, the animal exhibited evidence of severe pain and its abdomen became very swollen and tense. Death occurred a few hours after the dog was admitted to a veterinary clinic. Do you observe evidence of inflammation in this slide? What factors predisposes an animal to torsion?

SLIDE 72: EQUINE STOMACH: RUPTURE OF THE STOMACH:- The stomach of this horse was over distended by grain, considerable quantities of gas

accumulated and the stomach subsequently ruptured. Note that the rupture is incomplete and located along the greater curvature. The horse went into shock and died before severe inflammatory changes were established. In which animal species would you expect this condition to develop most commonly? What is colic? What signs of colic would be expected in this horse? What necropsy changes are associated with rupture of the stomach?

SLIDE 73: CANINE: STOMACH: RUPTURE OF THE STOMACH: - This thin and hungry 12 year old Great Dane gained access to a storage room in which feed was kept. Being somewhat greedy, the dog over ate, but did not drink adequate water. Subsequently, its stomach markedly dilated and ruptured. Note that gastric dilation can still be seen in this slide.

SLIDE 74: PORCINE STOMACH: ULCERATION OF THE STOMACH - Observe the large amount of clotted blood in the stomach lumen of this pig. At necropsy, a large ulcer was found in the esophageal portion of the stomach. Is it possible for this pig to have died from anemia? Explain. Pretend that this pig lived long enough for blood to pass in the feces. How would you expect this to appear grossly?

SLIDE 75: EQUINE STOMACH: ULCERATIONS AND EROSIONS OF THE STOMACH - Observe the small ulcers and erosions in the nonglandular portion of the stomach. These lesions were associated with *Gastrophilus* spp. (bots). What is the margo plica? Is it possible for a squamous cell carcinoma to have its origin in the stomach of a horse? Why?

SLIDE 76: BOVINE ABOMASUM: ULCERATION OF THE STOMACH - Observe the focal ulcerative lesions in the abomasum. The cause of this condition was not determined. In addition to the ulcers, you should be able to observe numerous tiny, whitish, raised foci over the mucosa. These are parasitic nodules induced by stomach worms.

SLIDE 77: BOVINE ABOMASUM: ULCERATION OF THE STOMACH - This abomasum is from the group of calves.

SLIDE 78: BOVINE ABOMASUM: ULCERATION OF THE STOMACH - This 10 year old Holstein was submitted to the necropsy laboratory with multiple ulcerations

of the abomasal mucosa as well as a grayish-white neoplasm that had infiltrated the wall. Observe the well-defined ulcerative lesions. Name the neoplasm you would be most likely to find in the abomasal wall of this cow.

SLIDE 79: EQUINE STOMACH: ULCERATION OF THE STOMACH - This 3 month-old foal had several perforating and nonperforating ulcers in the stomach. The cause of this condition was not determined. There was also a severe peritonitis at the time of necropsy. How would you explain the peritonitis?

SLIDE 80: EQUINE STOMACH: GASTROPHILUS LARVAE INFECTION - There are numerous larvae attached to the stomach mucosa. At the site of attachment of each larva, a small umbilicated ulcer may occur. Actually, the bots act as very mild irritants and small proliferative foci may be observed. Can you identify the margo plica in this slide? Name the important bots in this country. Briefly, outline the life cycle of this parasite.

SLIDE 81: EQUINE STOMACH: GASTROPHILUS LARVAE INFECTION - Observe the bots attached to the mucosa. Give the exact location of the three species of bots within the gastrointestinal tract. What complications may occur due to the presence of large numbers of bots in the location depicted in this slide?

SLIDE 82: EQUINE HAIR: GASTROPHILUS INFECTION - Observe the bot eggs closely attached to hair removed from the shoulder of a horse. Give the most likely species.

SLIDE 83: EQUINE STOMACH: HABRONEMIASIS - Observe the well defined nodule in the wall of the stomach. The small nematodes observed near the ruler were expressed from the nodule. This was an incidental finding at necropsy. What nematode would you expect to find in this nodule? What complications can develop subsequent to the lesion observed in this slide?

SLIDE 84: EQUINE STOMACH: HABRONEMIASIS - Observe the morphology, size, etc., of nematodes removed from a stomach nodule in a horse. On the basis of morphology, size, etc., you should be able to differentiate this parasite from ascarids, setaria, strongyles, etc.

SLIDE 85:BOVINE ABOMASUM: TRICHOSTRONGYLOSIS - This 6 month old steer apparently had a heavy stomach worm infection. Observe the numerous tiny nodules over the abomasal mucosa. What parasite(s) would you expect to find in the abomasum of this steer?

SLIDE 86:OVINE ABOMASUM: TRICHOSTRONGYLOSIS - Observe the large mass of *Hemonchus* spp. taken from the abomasum.

SLIDE 87:OVINE ABOMASUM: TRICHOSTRONGYLOSIS - Observe the submandibular edema associated with a heavy infection with *Hemonchus* spp. Give a likely pathogenesis for the submandibular edema. Give reasons the edema localized in this site.