Chapter 6
Diseases of the Upper Respiratory Tract
John Pringle

**RHINITIS AND NASAL OBSTRUCTION**

A. Rhinitis in horses
   1. Patient profile and history
      a. Acute rhinitis in the horse is most commonly found in young horses and is associated with infectious (viral, bacterial) respiratory diseases. In such cases, the infection is accompanied by signs of equine rhinotracheitis (see II A 3 f).
      b. Fungal infections (cryptococcosis, rhinosporidiosis) also can cause rhinitis in the form of granulomatous, pedunculated masses. Cases are most common in the southern United States and are rare and sporadic. Tumors causing signs of rhinitis are uncommon in horses.

   2. Clinical findings. Mucoid, mucopurulent, or blood-tinged nasal discharge is evident, as is inspiratory stridor if the nasal passages are markedly inflamed. In some cases, there may be decreased or unequal airflow from the nostrils, accompanied by malodorous air if necrotic processes are present.

   3. Diagnostic and therapeutic plans (see II A 3)

B. Rhinitis and nasal obstruction in cattle
   1. Etiology and pathogenesis
      a. Causes include viruses, bacteria, fungi, allergens, and masses (tumors). Rhinosporidium-like organisms produce polyps in the anterior nares.
         (1) Allergic rhinitis occurs mainly in Channel Island breeds, but it has been reported as a familial tendency in other breeds, occurring in spring and fall pollen seasons.
         (2) Ethmoid carcinomas are sporadic causes of nasal obstruction. Although there may be a familial tendency for occurrence, there is also a viral origin implicated in the genesis of these tumors. The tumors are suggested to be of moderate malignancy and can metastasize to the lymph nodes and lungs.
      b. Pathogenesis. Nasal obstruction may cause severe dyspnea, cyanosis, and stertorous breathing.

   2. Clinical findings
      a. Allergic rhinitis. Signs include acute dyspnea and sneezing, accompanied by yellow-orange nasal discharge. Chronic cases present with multiple nodules in the anterior nares. Affected animals may rub their nasal cavities by pushing sticks or twigs up the nostrils, resulting in lacerations or foreign bodies in the nasal cavity.
      b. Ethmoid carcinomas. Signs include bulging facial bones, epistaxis, and dyspnea. The tumors are usually unilateral but can be bilateral, blocking both nasal passages. In these cases, open-mouthed breathing is often necessary. These tumors occur most often in older cattle, age 6–9 years.

C. Rhinitis and nasal obstruction in sheep and goats
   1. Patient profile and etiology. Rhinitis and nasal obstruction in sheep and goats have primarily parasitic causes (e.g., Oestrus ovis) but can be sporadically caused by nasal tumors (e.g., adenopapillomas, adenocarcinomas) and viral infections or allergies, as in the other species. Oestrus ovis is the most common cause of nasal obstruction in sheep, but occasionally goats may also be affected.

   2. Clinical findings include catarrhal to mucopurulent nasal discharge, sneezing, and difficult, snoring respiration.
3. Specific conditions
   a. Oestrus ovis
      (1) **Etiology and pathogenesis**
         a. The fertilized females deposit larvae at the external nares during the summer
         and fall months. After hatching, young larvae crawl up the nasal cavity to the
         dorsal turbinates and frontal sinuses where they remain for several weeks to
         months before migrating to the nostrils and being sneezed out to pulate on the
         ground.
         b. Irritation and secondary bacterial infection result in a purulent or mucoid
         nasal discharge, sneezing, low head carriage, and inspiratory dyspnea.
         c. The clinical signs are most prominent in young pigs with a History and
         immunity. Polymorphonuclear neutrophils are elevated in the nasal arcade.
         d. Prevention has been attempted with early administration of antibiotics
         (e.g., oxytetracycline, trimethoprim-sulfadixone) in the early creep feed. Control
         has been aimed either at total eradication by depopulation or reduction of
         infection through mass medication or vaccination of pregnant gilts with B. bronchiseptica
         followed by P. multocida bacteria.
   b. Allergic rhinitis
      (1) **Etiology and pathogenesis**
         a. Patient profile and history. This condition is most commonly seen in growing
         pigs that are raised in poor environments with heavy organic debris contaminating
         the air and environment. The condition is characterized by sneezing, discharge,
         and nasal irritation.
         b. Clinical findings. The disease is characterized by sneezing, nasal discharge,
         and epistaxis in growing pigs. Severe cases may exhibit impaired growth rates.
         c. Etiology and pathogenesis. There is substantial evidence to implicate Bordetella
         bronchiseptica as the inciting agent of the acute inflammation, followed by inva-
         sion of toxigenic strains of Pasteurella multocida. Up to 50% of finished pigs may
         have evidence of atrophic rhinitis. The true economic significance of atrophic
         rhinitis remains undetermined, as field studies have failed to show strong evidence
         of adverse effect on daily weight gains in growing pigs.
         d. Prevention has been attempted with early administration of antibiotics (e.g., tylo-
         sin, oxytetracycline, trimethoprim-sulfadixone) in the early creep feed. Control
         has been aimed either at total eradication by depopulation or reduction of
         infection through mass medication or vaccination of pregnant gilts with B. bronchiseptica
         followed by P. multocida bacteria.

4. Necrotic rhinitis. Otherwise known as bullnose, this disease is characterized by fa-
cial deformities and is often confused with atrophic rhinitis.
   a. Patient profile and history. This condition is most commonly seen in growing
   pigs that are raised in poor environments with heavy organic debris contaminating
   the air and environment. The condition is characterized by sneezing, discharge,
   and nasal irritation.
   b. Clinical findings. Initially, there is a cellulitis of the soft tissues of the nose and face
   with localized swelling that may interfere with respiration and mastication.
   c. Etiology and pathogenesis. Fusobacterium necrophorum is commonly isolated
   from the affected sites. If untreated, the inflammation spreads to the nasal bones
   and can cause facial deformity, toxemia, reduced appetite, and death.
   d. Differential diagnosis. The main differentiating feature of bullnose in comparison
   to atrophic rhinitis is the presence of soft tissue cellulitis, which is usually com-
elately lacking in atrophic rhinitis.
   e. Therapeutic plan and prevention. Antibacterials, such as sulphonamides, are effec-
tive when treating young infected pigs and early stages of the disease. However,
   the aim should be the reduction of incidence, which is best managed by im-
proved sanitation and disinfection of pens and elimination of any material that
may cause mouth or head injuries (sharp edges on feeding troughs or waterers).

5. Pseudorabies. Some outbreaks of pseudorabies (Aujeszky's disease) may show signs
   of rhinitis.
3. Specific conditions.

3.1 Patient profile and history
(a) Affected horses are primarily the young stock (age 2–3 years), but all ages are susceptible. Older horses usually have a milder infection or may show few signs other than a transient fever.
(b) The disorder usually occurs as an explosive outbreak of respiratory disease in stables, but where immunity is strong, either from past exposure or vaccination, signs may be limited to several horses in a stable showing only fever or mild hind limb edema. Less commonly there can be associated complications of myocarditis and myocardiitis.

3.2 Epidemiology
(a) A hallmark of this infection is its extremely rapid spread though a population of susceptible horses. In contrast to the shorter surviving human and swine types of virus, the equine type virus survives for up to 36 hours on environments to allow the horse to recover from the infection.

3.3 Etiology and pathogenesis
(a) The causative virus is a myxovirus, which is an RNA virus with two serologically distinct antigenic types (influenza A/equi 1 and A/equi 2). There seems to be no cross-species infection, and, as in most influenza viruses, the viruses are subject to antigenic drift. Thus, the commercially available vaccines, although affording protection, are seldom 100% effective.
(b) Infection is initiated by inhalation or contact with nasal secretions from infected animals. The virus can persist in an infected horse's secretions for up to 8 days, and the most common source of infection is coughed secretions.
(c) After an incubation period of 1–5 days, the infection results in clinical signs, which reflect the epithelial inflammation of the respiratory tract. When the virus invades the respiratory epithelium, changes include hyperemia, edema, and cellular desquamation. Superficial erosions to the upper and lower respiratory tracts can occur with a loss of normal mucociliary clearance mechanisms, which provide at least transient potential for secondary bacterial invasion.

3.4 Postinfection complications
(i) When the virus has cleared, the respiratory epithelium can take up to 3 weeks to fully recover to its normal state. This may be one reason that some horses continue to exhibit coughing for several weeks after apparent resolution of the infection.
(ii) Associated but less common complications include myocarditis with arrhythmias (atrial fibrillation), secondary bacterial pneumonia, pleuritis, persistent cough, and exacerbation of underlying chronic obstructive pulmonary disease.

3.5 Clinical findings
(a) The disease causes a fever (38.5°C–41°C) and a dry hacking cough, which later turns moist and persists longer than the fever. Nasal discharge is watery when present but is seldom prominent.
(b) Adenopathy. Although the submaxillary lymph nodes are not appreciably swollen, they are often painful to palpation in the early stages, which indicates the pharyngeal inflammation that occurs and may cause signs of dysphagia.
(c) Other signs include dyspnea with or without exercise, systemic signs associated with infection (e.g., fever, inappetence, lassitude), or muscular stiffness and limb edema. In uncomplicated cases, the signs usually resolve completely within 3 weeks.

3.6 Diagnostic plan and laboratory tests
(a) On the routine complete blood cell count (CBC), there can be a leukopenia with distinct lymphopenia, but this is transient. For a definitive diagnosis, virus isolation is necessary.
(b) Nasopharyngeal swabs must be collected in the first 48–72 hours of illness, beyond which viral culture is unlikely to be successful.
(c) Serologic confirmation of infection relies on a rise in antibody titer in paired sera collected 3 weeks apart, with a positive finding based on a fourfold rise in hemagglutination inhibitor or serum neutralization titer.
(d) For rapid diagnosis, a test based on direct immunofluorescence applied to nasopharyngeal swabs also has been successful in investigating outbreaks.

3.7 Differential diagnoses
(a) Viral infections. This includes mainly the other upper respiratory viral infections (herpessirus, rhinovirus, or adenovirus). Although equine viral arthritis (EVA) also can cause respiratory signs, these signs are often of secondary importance. Other signs, such as conjunctivitis, petechiation of mucous membranes, and limb and palpebral edema, are more prominent.
(b) Bacterial infection by Streptococcus equi also can cause similar initial clinical signs, but generally the submaxillary lymph nodes are obviously enlarged and painful.

3.8 Therapeutic plan
(a) As with most viral infections, there are few specific treatments available to hasten recovery. The main goal is to provide a clean, stress-free environment to allow the horse to recover from the infection.
(b) For increasing the comfort of the horse, nonsteroidal anti-inflammatory drugs (NSAIDs), such as phenylbutazone, can be used to decrease fever and maintain the horse's appetite during the acute phase of the infection. However, disadvantages of this approach include:
(i) The antipyretic action of NSAIDs might mask any fever due to secondary bacterial infection.
(ii) Owners might return the horse to competition or work before the effects of the disease have fully abated.
(c) Antibiotic treatment is appropriate if secondary bacterial infection is suspected or in high-risk animals, such as young foals. Ideally, the choice of drug should be based on culture results from transtracheal wash. In the absence of this, broad-spectrum antibacterials such as trimethoprim-sulfas can be administered, with a course when initiated of 5–7 days.

3.9 Prevention
(a) Vaccines. There are several manufacturers of killed-strain influenza vaccines, containing both A/equi 1 and A/equi 2. Manufacturers recommend two intramuscular injections several weeks apart initially, then revaccination annually.
(b) Reaction to vaccination. Vaccinated horses should be rested for several days after vaccination because they are often reported to be "off" the day following injection. Horses develop a transient reaction to vaccination that may include mild fever, malaise, and pain at the injection site.
(c) Frequency of vaccination. Vaccination results in at least partial immunity annually.
to disease, but not to infection. The duration of immunity from any of the vaccines is probably less than 1 year. Vaccination in the face of an outbreak may be beneficial if it can be done in advance of the spread of disease.

- For high-risk animals, such as young horses in the show season, it is recommended to repeat vaccination every 3-4 months during the high-risk period.
- For backyard horses with no new additions, annual vaccination may not even be necessary for those horses older than 3 years.
- For foals, the usual recommendation is to begin vaccination between 2 months and 6 months. Some workers suggest that beginning vaccination at 30 days of age may decrease the incidence of fetal pneumonitis.

b. Equine viral rhinopneumonitis (Equine herpesvirus 4 and 1) infection

1. Patient profile and history. Rhinopneumonitis usually presents in weanlings and yearlings but can also occur in nursing foals. The disease can be seen in horses of all ages, with the presenting complaints being fever, conjunctivitis, and coughing.

2. Epidemiology
   - Carrier animals are often present in herds and serve to maintain the infection from year to year. The disease can be reactivated in periods of stress or by corticosteroid administration.
   - Rapid spread through a herd is associated with high morbidity and mortality rates; the respiratory disease is generally mild. Outbreaks most commonly occur in fall and winter months, and 85% of respiratory outbreaks involve EVH in serologic surveys are attributed to EVH-4.

3. Pathogenesis
   - The disease is caused by several differing strains of EVH, with EVH-4 causing most of the outbreaks of respiratory disease in horses in any population at any time of the year.
   - EVH-1, serologically related to EVH-4 by only 20% homology, has two subtypes: Type 1 is associated with abortion. Type 2, while also abortogenic, can cause respiratory disease.
   - Neurologic disease can also accompany EVH-1 infection, but the pathogenesis is poorly understood (see Chapter 11).

4. Pathogenesis
   - This highly infectious disease is transmitted by inhalation or contact with infected secretions (e.g., placenta, nasal secretions, aborted fetuses) containing infectious EVH particles. However, the virus can also be transmitted from 15 to 45 days outside the animal in the environment, infections can occur in the apparent absence of an initiating case.
   - The virus rapidly proliferates in the mucosa of the nasal, tracheal, and pharyngeal regions, resulting the rhinitis, pyrexia, and associated respiratory signs. Following this, there is a short-lived viremia in which the virus is disseminated to other tissues, such as placenta, lung, and nervous tissues and induces respiratory damage.
   - Immunity. The virus can be present in nasopharyngeal swabs for up to 10 days and can be shed spontaneously at times of stress in carrier animals. Immunity to these forms of herpesvirus is weak, and an animal can become clinically affected several times. Passive immunity in foals in the form of antibodies declines to zero by 180 days after birth. However, even the presence of virus-neutralizing antibodies is not necessary an indication of resistance to infection, or an

5. Clinical findings
   - The respiratory signs vary with the amount of exposure, animal age, and immunity. Signs are similar to those of influenza but are milder and more transient. As with influenza, younger animals most obviously are affected in an outbreak, whereas older animals may show few or no signs of respiratory disease.

b. The most common findings are pyrexia (39.5°C-40.5°C), conjunctivitis, and rhinitis. Nasal discharge is a possible mild cough. The appetite of infected horses generally remains unaffected, and there may be slight enlargement of lymph nodes in the throat region.

(3) The clinical course is usually 4-7 days, but some horses may cough for up to 2 weeks. Whereas some horses may undergo anaplastic changes, very young foals can develop a primary or secondary pneumonia. Reinfections may occur within 4-5 months, but usually immunity from clinical infections lasts 6-12 months.

d. Endoscopic examination of the upper respiratory tract shows mild mucosal inflammations, consisting of rhinitis, pharyngitis, and laryngeal hyperplasia.

(4) For respiratory disease caused by EVH-4, there may also be abortions up to 4-6 months and possibly neurologic disease 6-11 days after respiratory infection on the same farm.

5. Diagnostic plan. Respiratory disease accompanied by abortions, neurologic signs, or both is a presage of presumptive evidence of EVH-4 infection. However, where respiratory signs are the only abnormality, confirmation of infection can be either by acute and convalescent serology, or by virus isolation from nasal secretions, which is possible for up to 10 days.

6. Laboratory tests. On the routine E6, there is 3, as in most viral diseases, a nonspecific leukopenia and lymphopenia. Confirmation of the infection is best accomplished by collection of acute and convalescent sera, but virus isolation can be successful from nasal washing obtained in first few days of infection.

7. Differential diagnosis. For problems that are solely showing signs of upper respiratory disease, the same considerations as for influenza are appropriate. These include equine rhinovirus, adenovirus, and viral arteritis. Bacterial infection by Streptococcus equi also can show similar initial clinical signs, but generally the mandibular lymph nodes are obviously enlarged and painful.

(8) Therapeutic plan. As for influenza and other upper respiratory infections, there is little specific treatment other than providing a clean, draft-free, low-stress environment and discontinuing work while the horse is allowed to recover.

(9) Prevention is with vaccination. Because the respiratory disease is sufficiently mild, a major goal of vaccination is the prevention of abortion. It is generally accepted that vaccination should be incorporated into routine health maintenance only when there is a known endemic problem.

(10) There are cell-culture-adapted live viruses that can be used safely for routine work in most breeding farms; however, the weakness is the brevity of immunity from this vaccine. Brood mares need to be vaccinated twice during the latter half of pregnancy, and use in foals and yearlings requires, at the minimum, trimonthly boosters.

(12) A killed vaccine is in use for protection against abortion due to EVH but should not be allowed to protect against respiratory signs.

(13) To protect against the respiratory form of the disease, a modified live EVH-4 combined with EVH-1 and influenza antigens is available. This vaccine is administered initially when a foal is 2-4 months of age, repeated in 4-6 weeks and then yearly until 2 years of age. Many clinicians recommend more frequent administration of this vaccine in those areas where the incidence of clinical disease is high.

c. Equine viral arteritis (EVA)

1. Patient profile and history. EVA is a disease seen in outbreaks on breeding farms, causes pregnant mares to abort. Serologic evidence suggests a high
d. Equine rhinovirus

(ERV-I), EVA is caused by an arterivirus similar to the agent that is implicated in porcine reproductive and respiratory syndrome. Virulence between strains varies, but there is little antigenic variation.

(2) Etiology. EVA js caused by an arterivirus similar to the agent that is implicated in porcine reproductive and respiratory syndrome. Virulence between strains varies, but there is little antigenic variation.

(3) Clinical findings.
- **(a)** This disease primarily causes abortion, with acute systemic illness including fever, significant signs and respiratory signs. Abortion occurs usually within a few days of clinical onset of disease. This is in contrast to abortions caused by equine viral rhinopneumonitis, which occur much later after clinical disease.

(b) The respiratory signs are usually of secondary importance to the disease and include nasal and ocular discharge that is initially serous but can become purulent. There is a cough, mucosal congestion, and in some horses, petechiation. In horses with pulmonary edema, dyspnea also may occur.

(4) Pathogenesis. Although outbreaks of the classic disease can occur, infections are commonly subclinical with sporadic abortions. Stallions can act as carriers, and there is an effective vaccine available for prevention where indicated.

(5) Therapeutic plan and prevention.

(a) Specific therapy for this viral infection is not available, so treatment is only directed against secondary bacterial infection if respiratory signs are present.

(b) Quarantine. Because the virus is contagious, the quarantine of any infected horse returning from a racetrack, sale, or show may be necessary for up to 4 weeks.

(c) A currently available modified-live virus vaccine may be useful to control infection with several types of rhinovirus in horses.

(6) Noninfectious equine rhinotracheitis can be the result of pharyngeal abscess, foreign body, irritation from orally administered medication, or nasogastric intubation. These causes are far less common than infectious causes.

(7) Chronic rhinitis. The most commonly managed chronic problem of the pharynx is lymphoid hyperplasia (follicular pharyngitis), as observed by endoscopy in performance horses. This abnormality does not appear to affect health but is suggested by some to affect athletic ability. This and other pharyngeal abnormalities, including structural or functional abnormalities of the soft palate, are seen in the realm of equine surgery.

B. Acute pharyngitis, laryngitis, and tracheitis in cattle

1. Clinical findings. The common upper respiratory tract infections in cattle frequently have nonspecific signs, and an etiologic diagnosis based solely on clinical signs is not possible. The exception to this is infectious bovine rhinotracheitis (IBR), in which there can be characteristic nasal reddening, plaques, and conjunctivitis.

2. Pathogenesis. Most of the incriminated bovine respiratory viruses are ubiquitous, and exposure and immunity are widespread. An important factor in these infections is the potential to compromise normal pulmonary defense mechanisms and allow colonization by bacteria that would normally be cleared. Therefore, although viral respiratory infections may not extend to the lower respiratory tract, secondary bacterial infection is the primary consideration in the management of both treatment and prevention.

3. Diagnostic plan and laboratory tests. Specific etiologic diagnosis requires virus demonstration or isolation and identification and is seldom successful in late stages of the disease. To maximize the potential of an etiologic diagnosis, a routine set of samples should be obtained in outbreaks.

a. From live, acutely infected feline untreated cattle, nasal swabs or conjunctival scrapings are suitable for most viral isolation. Acute and convalescent serum samples also can be collected and screened for the suspected viruses, with a fourfold
titer increase over 2–3 weeks proving active recent infection. For some viruses (e.g., bovine respiratory syncytial virus), immunofluorescence on lung lavage cells also has proven highly valuable.

b. Necropsy of animals can be useful but is seldom rewarding in chronically ill, treated, or cull animals or those dead long enough to have undergone significant organ autolysis. In a field necropsy, tissues selected for virus isolation include conjunctiva, tonsil, pharynx, lung, lymph node, spleen, liver, kidney, small intestine, cecum, spiral colon, and rectum. These tissues require placement in appropriate viral transport medium, freezing, and transport by overnight courier to the laboratory.

4. Therapeutic plan

a. Supportive care including ready access to feed and water and minimizing competition for feed and space is often all that is required.

b. Antibiotic therapy for possible secondary bacterial infection can be instituted. Although glucocorticoids impair defense mechanisms and are contraindicated in BR infections, NSAIDs (e.g., aspirin) may be beneficial in reducing fever and improving appetite. Therapy of individual animals is generally not as important as control of herd outbreaks.

5. Prevention. Vaccines are available for the more clinically important viruses and are often combined in a single product.

a. Modified live virus of bovine cell origin for IBR vaccines should not be used in pregnant cows because these vaccines may cause vaccine virus shedding, infertility, and abortions. Potential cub st bulls should be vaccinated with products containing the IBR antigen only at the owner’s request.

b. Products that are designed for intranasal administration of live vaccine stimulate strong local (immunoglobulin A) immunity and a rapid response, including local interferon.

c. When considering the young stock in the herd, any early vaccination, such as is sometimes performed by administration of intranasal vaccines soon after birth, should be repeated after 6 months of age because the colostral immunity has waned by that time for most of these viruses.

d. In dairy herds, annual or semiannual vaccination is recommended, as well as vaccinating any replacement animals 3–4 weeks before introduction.

e. In feedlot management, because of high stress of transport and shipping, killed or at least inactivated vaccines may be appropriate, unless modified live viruses can be administered at least several weeks in advance of anticipated stress periods.

6. Specific conditions

a. Infectious bovine rhinotracheitis (IBR), also known as bovine herpes virus-1 (BHV-1), is a highly infectious condition of worldwide distribution in cattle and some wild ruminants. Recent studies have revealed at least five major biotypes, which might explain the distinct clinical manifestations of this viral infection. The respiratory form is a common manifestation of infection in cattle and is usually restricted to upper respiratory signs, as uncomplicated IBR does not usually spread into the lungs.

(i) Patient profile and history. BHV-1 can affect all ages of cattle, but those older than 6 months are most commonly affected, with animals in beef feedlots experiencing higher morbidity than dairy herds.

(ii) Epidemiology. The morbidity rate varies from 8% to 20%–30% in feedlots, but in herds of low immunity, morbidity can approach 100%. However, the case fatality rate is usually low at 1% or less, where fertility rises to 10%, deaths are usually related to secondary bacterial bronchopneumonia (see Chapter 7).

(iii) Etiology and pathogenesis

(a) Transmission. The α-herpes virus BHV-1 is transmitted via respiratory aerosol, semen, fetal fluids and tissues, and somites.

(b) After infection in the field, it appears there is an incubation period of 10–20 days, although experimentally this only lasts 3–7 days. The BHV-1 virus multiplies in nasal mucosa, causing rhinitis and tracheitis. Viral excretion via the lacrimal duct causes conjunctivitis and, in some cases, corneal edema.

(c) Route of infection. From nasal mucosal infection, the virus travels up the trigeminal nerve, where it can establish latency or infect the central nervous system. The virus also can be transported by peripheral leukocytes to the placenta and transferred to the fetus, resulting in abortion.

(d) Carriers

(i) The virus can persist and be discharged from the animal as a result of natural infection or live virus vaccination for at least 2 years.

(ii) Carrier states are thought to occur in some cattle, and there also may be wildlife reservoirs of infection in wild ruminants.

(iii) Latent infections occur with the virus presumably sequestered in the trigeminal ganglion, which can recrudesce during stress or corticosteroid administration.

(iv) Immunity to BHV-1 is complex and requires both cell-mediated and humoral parts of the animal’s defenses. Therefore, it follows that systemic antibody levels, as determined by the many serologic tests available, correlate poorly with protection against disease.

(4) Clinical signs

(i) The various clinical manifestations of IBR include:

(a) Upper respiratory inflammation with or without prominent conjunctivitis.

(b) Venereal infections, such as infectious pustular vulvovaginitis (IPV) in cows and balanoposthitis in bulls.

(c) Encephalitis.

(d) Intermittent (eye) irritability.

(e) Abortion.

(f) With the respiratory form of IBR, there is a sudden onset of severe signs, including high fever (up to 42°C), anorexia, and severe hyperemia of the nasal mucosa.

(g) Necrotic plaques appear as greyish foci of necrosis on the nasal mucosa just inside the nares, accompanied by a serous to mucopurulent nasal discharge.

(h) Conjunctivitis with a serous ocular discharge is also a common sign. Although this may sometimes be mistaken for infectious keratoconjunctivitis caused by Moraxella bovis, the lesions are confined to conjunctiva, which are reddened and swollen with no invasion and ulceration of the cornea.

(i) A short, explosive cough can accompany these signs but is not always present in outbreaks. If lactating dairy cattle are affected, there is also an accompanying dramatic fall in milk production.

(j) Duration of infection. When restricted to respiratory signs, infection generally resolves in 10–14 days. Abortions can occur some weeks following clinical illness. The genital tract infections (IPV or balanoposthitis) can result in reproductive failure.

(2) Diagnostic plan and differential diagnoses. The classic signs of fever, nasal lesions, and bilateral conjunctivitis should suggest the respiratory form of IBR. Other possibilities include:

(a) Bovine respiratory disease complex (BRDC; shipping fever). With BRDC, there is toxemia, abnormal lung sounds, and affected animals respond well to antibiotics.

(b) Bovine virus diarrhea/mucosa disease (BVD/M). With BVD/M, there should be oral erosions and usually diarrhea in addition to nasal ulceration.

(c) Bovine malignant catarrh (BMC). BMC exhibits similar signs to BVD/M.

(d) Calf diphtheria. This condition may resemble IBR with inspiratory dyspnea but usually has severe toxemia and necrotic oral and laryngeal lesions.

(e) Allergic rhinitis. Although it resembles IBR, allergic rhinitis does not...
result in fever and usually is accompanied by sneezing and a characteristic thick greenish orange nasal discharge.

(6) Therapeutic plan

(a) Antibiotics. Although of no direct effect against the viral infection, antibiotics such as oxytetracycline or sulfa drugs can be given to control against secondary bacterial tracheitis and bronchopneumonia. As most cattle recover uneventfully without antibiotics, this must be weighed against the cost of treatment and possible need for appropriate withdrawal periods of milk or meat.

(b) Management strategies. As in other viral infections, it is important to aid recovery by reducing stress (e.g., crowding) and providing high-quality feed and access to water. In feedlot situations, this is best managed in a separate "sick pen" where competition for feed and space is reduced, and particular attention can be given to monitor for signs of onset of secondary bronchopneumonia.

(c) Glucocorticoids are specifically contraindicated in this disease.

(7) Prevention

(a) There are several effective vaccines commercially available. Because the disease can occur unpredictably at any time and even in what seem to be closed herds, vaccination by either an intranasal aerosol of modified live vaccine or intramuscular modified live or inactivated vaccines is indicated.

(i) The intranasal vaccines stimulate local as well as humoral immunity, and in addition to being safe for use in pregnant cows, can be used in the face of an outbreak because of stimulation of local interferon within 72 hours of administration. However, these vaccines are more labor intensive to administer and generally more expensive.

(ii) Intramuscular vaccines, if not inactivated, can cause abortion and infertility. Thus, if used, they should be given to heifers and cows at least 2 weeks before breeding.

(iii) Vaccination with modified live products, although stimulating a stronger immune response than that from inactivated products, may result in shedding of the virus.

(b) For those herds in which export of cattle or production of bulls for artificial insemination units is an economically important consideration, vaccination against BHV-1 is not advised because this may result in the rejection of these exports by the sale of animals. Adenovirus may play a role in enzootic calf pneumonia (see Chapter 7).

(8) Driving and management strategies. As in other viral infections, it is important to aid recovery by reducing stress (e.g., crowding) and providing high-quality feed and access to water. In feedlot situations, this is best managed in a separate "sick pen" where competition for feed and space is reduced, and particular attention can be given to monitor for signs of onset of secondary bronchopneumonia.

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(i) The intranasal vaccines stimulate local as well as humoral immunity, and in addition to being safe for use in pregnant cows, can be used in the face of an outbreak because of stimulation of local interferon within 72 hours of administration. However, these vaccines are more labor intensive to administer and generally more expensive.

(ii) Intramuscular vaccines, if not inactivated, can cause abortion and infertility. Thus, if used, they should be given to heifers and cows at least 2 weeks before breeding.

(iii) Vaccination with modified live products, although stimulating a stronger immune response than that from inactivated products, may result in shedding of the virus.

(b) For those herds in which export of cattle or production of bulls for artificial insemination units is an economically important consideration, vaccination against BHV-1 is not advised because this may result in the rejection of these exports and the sale of animals. Adenovirus may play a role in enzootic calf pneumonia (see Chapter 7).

(8) Driving and management strategies. As in other viral infections, it is important to aid recovery by reducing stress (e.g., crowding) and providing high-quality feed and access to water. In feedlot situations, this is best managed in a separate "sick pen" where competition for feed and space is reduced, and particular attention can be given to monitor for signs of onset of secondary bronchopneumonia.

(c) Glucocorticoids are specifically contraindicated in this disease.

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Pharyngitis, laryngitis, and tracheitis in sheep, goats, and swine. Although these conditions undoubtedly occur in sheep, goats, and swine, far less attention is given to these conditions in North American veterinary literature.

1. Viral causes of pharyngitis and tracheitis are similar to those in cattle (e.g., respiratory syncytial virus has been reported). A more common cause in the small goat herd or sheep flock is traumatic pharyngitis, caused by drenching or balling gun injuries as a result of owner-administered medications.

2. Pharyngitis in swine is reported in some outbreaks of pseudorabies (Aujeszky's disease) and is part of the manifestation of anthrax in this species.

III. LARYNGEAL OBSTRUCTION

A. Equine strangles, Strangles, also called horse distemper, occurs worldwide and historically caused major epidemics in cavalry horses. This remains an important disease of horses in developed countries because of the resulting disruption in management in broad mare or racing farms, time and expense of treatment, and unpleasant aesthetics of draining abscesses and purulent nasal discharges.

1. Patient profile. The disease affects horses less than age 5 or 6 years, although all ages can be susceptible. Foals less than 3 months are most often unaffected, probably because of colostal protection.

2. Clinical signs
   a. After an incubation period of 1–3 weeks, affected horses suddenly develop depression, complete anorexia, fever (39.5°C–40.5°C), and serious nasal discharge.
   b. The nasal discharge rapidly becomes copious and purulent, and horses show signs of severe pharyngitis and laryngitis, with reluctance to swallow and a soft, moist cough that appears painful. Possibly as a result of this pain, affected horses often stand with their head and neck extended and may appear dyspneic.
   c. For 3 or 4 days, the lymph nodes of the throat region enlarge and are hot, painful, and initially firm in consistency. The lymph node enlargement may be sufficiently severe to cause obstruction to swallowing, dyspnea, and, in severe cases, death by asphyxia. Within 10 days, the swollen lymph nodes begin to weep serum and develop a soft spot from which they rupture (usually externally) and drain thick yellow material. Occasionally, these nodes rupture and drain internally into the pharynx. Often, the horse shows an improved attitude when the lymph nodes rupture and drain.
   d. Complications
      (1) Most animals recover within 3–6 weeks, but there can be a number of secondary complications. These include pneumonia of aspiration to aspiration with internal rupture or extension into the guttural pouches, causing guttural pouch empyema.
      (2) Very young foals can develop bacteremia or septicemia with joint infections and generalized lymphadenopathy.
      (3) The most common complication is metastatic strangles, or "bastard strangles," in which abscesses can spread to internal organ systems (i.e., lung, mesentery, spleen, brain) and cause subsequent localizing signs. Localized abscesses on the limbs can induce limb edema with the lower limb swelling three or four times its size. Finally, a delayed reaction due to immunologic sensitivity to the streptococcal protein can result in vasculitis, causing purpuric hemorrhagic.

3. Epidemiology
   a. Outbreaks in susceptible animals often occur in cold wet weather, although movement and exposure to infected horses are also factors.
   b. The causative organism is highly resistant in the environment, lasting up to a
month outside the host. Thus, contaminated water or feed buckets, or even objects such as blankets, brushes, and tack can be the source of infection.

c. The organism is known to persist in the pharynx of clinically normal, recovered horses for up to 8 months, and field experience suggests that such horses can be a source for new infections during this time.

4. Etiology and pathogenesis

a. Strangles is caused by a β-hemolytic streptococcus, Streptococcus equi, which is present in the nasal discharges and draining abscesses of affected horses. This organism is not considered part of the normal nasal flora of the horse.

b. The bacteria usually is transmitted by inhalation but can also be ingested. After incubating for 1–3 weeks, the organism causes acute pharyngitis and rhinitis. S. equi has M protein in its capsule, which is antiphagocytic and provides a means of avoiding normal defenses.

c. From the mucosal surfaces, the organism moves by lymph drainage to local lymph nodes (submandibular and retropharyngeal) with subsequent abscessation at these sites.

d. Strong immunity occurs immediately after infection and lasts 6 months to several years.

5. Diagnostic plan and laboratory tests

a. In addition to clinical signs, cultures of nasal swabs or lymph node drainage is key to diagnosis. Although other streptococcal species, such as S. zooepidemicus, also are readily found in nasal swabs; and can give mild upper respiratory signs along with occasional lymph node abscessation, identification of S. equi is important because it requires rigorous control and quarantine measures.

b. Routine hematology reveals neutrophilic leucocytosis, hyperfibrinogenemia, and anemia of chronic infection, but this is not specific to strepangles.

6. Therapeutic plan

a. Horses with early clinical signs (e.g., fever, anorexia, depression, pharyngitis, purulent nasal discharge) require treatment. The drug of choice administered at 22.00 IU/kg, twice daily intramuscularly for 5 days or until all clinical signs are absent.

b. Tetracyclines are also effective but should be avoided because of the risk of inducing colitis.

(3) Trimethoprim-sulfadiazine is an alternative to penicillin treatment, with its advantage being oral administration.

b. Horses with lymph node abscessation require local treatment to enhance maturation and drainage of the abscesses.

(1) Hot packs and poultries can be applied to the area of swellings several times daily. When the abscesses are mature, with the softening of a point of overlying skin, they can be lanced and flushed with 5–5% povodine iodine.

(2) Parenteral antibiotics given after abscessation formation tend to prolong rather than arrest disease. However, some veterinarians suggest treating all affected animals to reduce the risk of more animals becoming ill.

(c) Horses recently exposed to strepangles with yet no clinical signs may benefit from antimicrobial therapy (e.g., benzathine penicillin) at the time of exposure and every 2 days thereafter until the end of the outbreak. This may prevent seeding of lymph nodes with the organism.

d. Treatment for secondary complications

(1) For bastard strangles, long-term penicillin treatment (3–6 months) is required. Oral phenoxymethyl penicillin (100.000 IU/kg every 8 hours) is possible but expensive. Thus, trimethoprim-sulfadiazines usually provide the drug of choice.

(2) Purpura hemorrhagica, being an immunologic disease, requires glucocorticoids (dexamethasone) and supportive treatment such as leg wraps for the limb edema (see Chapter 9).

(3) Cutaneous abscesses are treated locally by flushing the affected pocketholes with saline through the pharyngeal opening, often with the use of indwelling catheters for repeated treatment.

7. Prevention

(a) Management strategies

(1) From the onset of clinical signs, it is critical to isolate affected animals because this highly contagious infection can spread through many of the young stock. Isolation for at least 6 weeks after start of signs is suggested, but based on field experiences, some veterinarians strongly suggest 8 months is a safer time frame. Clearly this latter recommendation must have the full cooperation and understanding of the client.

(2) Because of the highly resistant nature of the organism, it is also imperative to thoroughly clean and disinfect stalls and grooming and feeding equipment, and the bedding from infected animals.

(b) Vaccination against this organism provides only partial protection, with reduced severity and incidence of disease if and when it occurs.

(1) Several commercial products are available and require a minimum of three doses at 2- to 4-week intervals, followed by annual boosters. Some products also induce muscle soreness and possible abscess formation, and for this reason vaccination should be administered in the pectoral muscles.

(2) A vaccine containing a concentrated, purified M-protein extract of S. equi has been shown to reduce the rate of clinical disease by 50%.

8. Laryngeal obstruction in cattle

1. Patient profile and history

(a) Adult cattle

(1) Adult cattle develop acute laryngeal obstruction most commonly as a result of laryngeal necrosis secondary to balling gun and drenching injuries. Laryngeal edema secondary to smoke inhalation also can result in signs of laryngeal obstruction.

(2) Chronic laryngeal obstruction in adults is most often caused by retropharyngeal swelling, either from lymphadenitis or abscess, or a tumor in the throat latch region.

(b) Calves, particularly those between 3 and 18 months, most commonly develop laryngeal obstruction as a result of the calf diphtheria (oral laryngeal necrobacillosis).

2. Clinical signs

(a) Laryngeal obstruction causes characteristic inspiratory dyspnea and stertor. There is also apparent excessive salivation caused by the reduced willingness for the animal to swallow as a result of the dysphagia induced by laryngeal inflammation.

(b) There is anorexia, depression, and fever in calves with diphtheria and adult cattle with balling gun injuries that develop extensive cellulitis. In calves with diphtheria, there is invariably a characteristic foul necrotic odor to the breath.

3. Etiology and pathogenesis

(a) Inflammation of the larynx and pharynx depends on the inciting cause. For example, smoke inhalation may be a combination of chemical and thermal burn and, thus, be diffuse yet affect only the superficial mucosa.

(b) Bailing gun injuries often extend into the interstitial tissues around the larynx and have extensive cellulitis from contaminating oropharyngeal inhabitants.

(c) In calf diphtheria, the initial pharyngeal swelling, either from lymphadenitis or abscess, or a tumor in the throat latch region.

4. Diagnostic plan. The clinical signs and history often are sufficient to provide a working diagnosis.

(a) Oral examination with a speculum or an endoscope via the nares provides ready assessment of most of the structures of the pharynx and larynx.

(b) For bailing gun injuries, radiology of the pharyngeal region also can be valuable with the demonstration of gas or foreign matter in areas swollen with cellulitis.
5. Therapeutic plan
   a. Most conditions benefit from parenteral antibiotics, such as sodium sulfadimidine 150 mg/kg for 2-3 days or procaine penicillin 20,000 IU/kg intramuscularly twice daily for 5 days.
   b. In severely dyspneic animals, a tracheostomy should be performed as asphyxiation may occur before laryngeal swelling subsides.
   c. Those animals that are unable to swallow require parenteral fluid therapy.
   d. In cases of balling gun injury that are recognized at the time of occurrence and presumed to have caused extensive trauma, emergency slaughter should be considered as an economic alternative to treatment.

C. Laryngeal obstruction in sheep, goats, and swine
   1. Patient profile and history. Similar sporadic causes of laryngeal obstruction as found in cattle also can occur in small ruminants. In sheep, there also have been outbreaks of laryngeal obstruction reported resulting from necrotic laryngitis, with Fusobacterium necrophorum isolated from lesions.
   2. Clinical signs include laryngeal stenosis, causing the characteristic inspiratory dyspnea. Also, regional lymphadenopathy and lung abscession are seen.
   3. Therapeutic plan. Treatment is similar to that described for cattle. Laryngeal obstruction in swine is most commonly seen as a result of encroachment of regional subcutaneous abscesses or abscessed lymph nodes.

STUDY QUESTIONS

DIRECTIONS: Each of the numbered items or incomplete statements in this section is followed by answers or by completions of the statement. Select the ONE numbered answer or completion that is BEST in each case.

1. Which one of the following statements regarding viral respiratory disease in horses is true?
   (1) Although equine viral arteritis (EVA) is associated with conjunctivitis, limb edema, and respiratory signs, subclinical infections are most common, with aborting mares acting as the main carriers of the virus.
   (2) Equine rhinopneumonitis virus infection caused by equine herpesvirus-4 (EHV-4) results in fever, conjunctivitis, and cough, whereas equine herpesvirus-1 (EHV-1) is associated with late-gestation abortion and neurologic disease in addition to fever, conjunctivitis, and cough.
   (3) In addition to fever and the acute outbreak of cough, equine influenza virus can cause limb edema, myositis, and conjunctivitis in affected horses.
   (4) Painful submandibular lymph nodes are a characteristic of rhinopneumonia.
   (5) In horses, clinical signs of viral infection are seldom pathognomonic, and finding evidence of seroconversion is the only method of identifying many viruses.

2. A growing pig shows acute nasal discharge and rhinitis, followed by shortening of the snout and turbinate atrophy. Which one of the following statements is true?
   (1) Inclusion body rhinitis is a major factor in the turbinate atrophy noted.
   (2) The changes to the snout are typical of necrotic rhinitis (bullnose).
   (3) Brachyspira bronchiseptica infection followed by Pasteurella multocida infection is the most likely cause of these clinical signs.
   (4) Infection by type A swine influenza virus damaged the nares and was followed by Fusobacterium necrophorum infection of the turbimates.
   (5) Because secondary bacterial involvement was not found, a fungal pathogen should be suspected instead of a primary viral agent.

3. Equine herpesvirus-4 (EHV-4) is recovered from a group of horses on a brood mare farm following an outbreak of upper respiratory disease. Which one of the following statements is correct?
   (1) Vaccination against equine herpesvirus-1 (EHV-1) provides temporary but strong cross protection against EHV-4 infection.
   (2) Most respiratory outbreaks in horses caused by herpesviruses are caused by EHV-4.
   (3) Complications of EHV-4 infection include abortion and hind-end paresis.
   (4) Differentiating EHV-4 infection from EHV-1 infection can be done using currently available serologic tests for specific antibodies.
   (5) Because secondary bacterial involvement was not found, a fungal pathogen should be suspected instead of a primary viral agent.

4. A 3-week-old Arabian foal that has been receiving treatment for pneumonia dies and adenovirus is recovered from the lungs as the only pathogen. Which one of the following statements is true?
   (1) Serum obtained from this foal immediately prior to death can be used to confirm an immunodeficiency in this foal.
   (2) The presence of presuckle immunoglobulin M (IgM) against adenovirus confirms that this infection was present at birth.
   (3) Adenovirus is unlikely to cause significant disease in non-Arabian foals.
   (4) The other foals on the farm of similar ages should be vaccinated against adenovirus.
   (5) Because secondary bacterial involvement was not found, a fungal pathogen should be suspected instead of a primary viral agent.
5. Inspiratory dyspnea and stertor are observed in a 8-month-old Hereford calf. The rectal temperature is 39°C, and the calf is depressed. Furthermore, a foul, necrotic breath odor is detected, and the calf appears reluctant to eat. What is the most likely diagnosis?

1. Calf diphtheria caused by Fusobacterium necrophorum
2. Balling gun injury sustained 24 hours ago
3. Enzootic pneumonia complicated by lung abscession
4. A tooth root abscess with invasion by anaerobic bacteria
5. Papular stomatitis or bovine virus diarrhea (BVD)

6. Absence of passage of air in one nostril is a specific finding that may suggest which one?

1. Enzootic pneumonia in a pig with concomitant bilateral bloody discharge and bulging of the facial bones
2. Papular stomatitis or bovine virus diarrhea (BVD)

7. An owner reports the sudden onset of a harsh cough and bilateral ocular discharge in an 18-month-old steer. Other similarly aged animals in the herd are also similarly affected. Clinical examination reveals a rectal temperature of 40°C, a serious nasal discharge, swollen conjunctiva, and small necrotic plaques in the nares. What advice should be offered to the owner?

1. The affected animals need to be isolated from the breeding herd because they may be a source of an infection that can cause cerebellar hypoplasia or immunotolerance in calves.
2. To halt the spread of infection, an intranasal vaccine can be administered.
3. Signs are typical for Pasteurella pneumoniae; affected calves need to be placed on antibiotics.
4. This is likely an upper respiratory virus but additional tests will be needed to identify it.

5. Treatment should include broad-spectrum antibiotics because lung infection is likely to occur.

8. A yearling Standardbred filly is receiving treatment in a clinic for clinical signs of strangulation. Streptococcus equi is cultured from the affected nare.

1. Throat swab or buccal smear
2. Oesophagus biopsy
3. Inclusion body rhinitis
4. Ethmoid sinusitis
5. Granulomatous pedunculated masses

9. The answer is 2 [II A 3 b (3) (e)]. Equine viral rhinopneumonitis caused by equine herpesvirus-4 (EHV-4) and equine herpesvirus-1 (EHV-1) is associated with fever, conjunctivitis, and cough. In addition, EHV-1 infection may cause late-gestation abortion and neurologic disease. Stallions, rather than aborting mares, are the presumed carriers of the virus that causes equine viral arthritis (EVA). Although equine influenza may be associated with limb edema and myositis, conjunctivitis is not a recognized sign. Equine rhinopneumonia, a viral process, should not induce enlarged and painful lymph nodes; this sign is more consistent with Streptococcus equi infection. Some viruses can be cultured in the early stages; therefore, serology is not the only means of identifying some viruses.

1. The answer is 2 [II A 3 b (2) (b)]. More than 80% of respiratory outbreaks involving equine herpesvirus are associated with equine herpesvirus-4 (EHV-4); EHV-4 cannot be differentiated from EHV-1 using commonly available serologic methods. Equine herpesvirus-1 (EHV-1) has only 20% homology with EHV-4; therefore, vaccination against EHV-1 does not provide much protection against EHV-4. Abortion and hind-end paraparesis are complications associated with EHV-1 infection.

2. The answer is 3 [II D 1 b]. Atrophic rhinitis often occurs after infection of Bordetella bronchiseptica, which causes acute inflammation. B. bronchiseptica infection is then followed by Pasteurella multocida infection. Inclusion body rhinitis can cause acute nasal discharge but is not thought to be a component of atrophic rhinitis. Necrotic rhinitis is a cellulitis in the soft tissues around the snout and is thought to be caused by Fusobacterium necrophorum infection.

3. The answer is 3 [II A 3 b (2) (b)]. More than 80% of respiratory outbreaks involving equine herpesvirus are associated with equine herpesvirus-4 (EHV-4). EHV-4 cannot be differentiated from EHV-1 using commonly available serologic methods. Equine herpesvirus-1 (EHV-1) has only 20% homology with EHV-4; therefore, vaccination against EHV-1 does not provide much protection against EHV-4. Abortion and hind-end paraparesis are complications associated with EHV-1 infection.

4. The answer is 3 [II A 3 e]. Adenovirus only causes severe disease in foals with combined immunodeficiency, such as that observed in some Arabian foals that lack the ability to produce their own immunoglobulins and are severely lymphopenic. The foal has no ability to produce its own immunoglobulin M (IgM); therefore, sera from the foal at the time of age will likely contain immunoglobulins present from the dam's colostrum. The other foals on the farm are unlikely to be at risk for such a severe, overwhelming infection because they probably are not immunodeficient. Adenovirus in this case can be the sole pathogen for pneumonia.

5. The answer is 1 [II B 6 a (5) (d)]. The inspiratory dyspnea, fever, and necrotic breath odor are characteristic signs of calf diphtheria. A balling gun injury sustained 24 hours previously would not usually be likely to produce an extremely necrotic breath odor so quickly. The breath odor of lung abscesses is consistent with that described here, but calves with enzootic pneumonia will not have dyspnea on inspiration. Dental problems would not be characterized by dyspnea. Papules and ulcers, not necrosis, are more likely to be associated with papular stomatitis or bovine viral diarrhea (BVD).

6. The answer is 4 [II B 2 b]. Clinical findings of ethmoid carcinomas in cattle include bulging facial bones and the discharge of blood from the nostrils. The bloody discharge should not have a notable odor. The discharge is not usually blood-tinted; it is blood. Clinical signs are usually minimal.

7. The answer is 2 [II B 6 a (7) (a) (i)]. The signs are typical of infectious bovine rhinotracheitis (IBR). An intranasal vaccine is available that can be used in the face of an outbreak because it stimulates the production of local interferon within 72 hours. The presence of the conjunctivitis and nasal plaques should allow the clinician to rule out bovine virus diarrhea and mucosal disease. An animal with Pasteurella pneumonia would not have the conjunctivitis and would be significantly depressed. In uncomplicated viral infections such as this one, antibiotics are not indicated.

8. The answer is 2 [II A]. Shedding of the organism causes equine strangulates (horse disease). Streptococcus equei, can persist for 6 weeks or longer after the clinical signs of infection resolve; therefore, the filly is at risk of spreading the disease to other horses at the horse establishment. A vaccine is available, but the immunity is not strong and offers only partial protection. S. equei is not part of the normal flora in horses. Immunity does occur with infection but is not permanent.