



# THE MERCK VETERINARY MANUAL



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Hemorrhagic Septicemia

## Overview of Hemorrhagic Septicemia

Hemorrhagic septicemia (HS) is an acute, highly fatal form of pasteurellosis that affects mainly water buffalo, cattle, and bison. It is considered the most economically important bacterial disease of water buffalo and cattle in tropical areas of Asia, particularly in southeast Asia, where water buffalo populations are high. Disease is most devastating to smallholder farmers where husbandry and preventive practices are poor and free-range management is common. HS is also an important disease in Africa and the Middle East, with sporadic outbreaks occurring in southern Europe. The only confirmed outbreaks of HS in the Americas occurred in bison in Yellowstone National Park, most recently from 1965–1967. Natural disease occurs infrequently in pigs, sheep, and goats and has been reported in camels, elephants, horses, donkeys, yaks, and various species of deer and other wild ruminants.

### Etiology

Classical HS as defined by the OIE is caused by *Pasteurella multocida*

serotypes B:2 and E:2 (Carter and Heddleston classification system). Serotype B:2 has been identified in most areas where the disease is endemic, whereas serotype E:2 has been found only in Africa. Septicemic pasteurellosis that is clinically similar to HS can be caused by a wide variety of other *P multocida* serotypes and has been reported worldwide.

## Transmission, Epidemiology, and Pathogenesis

The tonsils of up to 5% of healthy water buffalo and cattle are colonized by small numbers of *P multocida* serotype B:2 or E:2, which can be shed during periods of stress. Common stressors associated with outbreaks include high temperature and humidity, concurrent infection (blood parasites or foot and mouth disease), poor nutrition, or work stress. Although outbreaks can occur at any time, disease is most prevalent during the rainy season. Increased outbreaks associated with high rainfall are most likely due to the multiple stressors present during this time and the moist conditions, which prolong the survival time of the organism in the environment. Infection occurs by contact with infected oral or nasal secretions from either healthy carrier animals or animals with clinical disease, or by ingestion of contaminated feed or water. Infection begins in the tonsil and adjacent nasopharyngeal tissues. Subsequently, bacteremia leads to dissemination and rapid growth of bacteria in various locations, tissue injury, a host cytokine response, and release of lipopolysaccharides that results in a rapidly progressing endotoxemia. Clinical signs can appear 1–3 days after infection, and death can occur within 8–24 hr after the first signs develop. In endemic areas, HS affects older calves and young adults, and morbidity and mortality are variable. In nonendemic areas, epizootics can occur with high morbidity and mortality that can reach 100%. Water buffalo tend to be more susceptible and have more severe clinical disease than cattle. Recovery can stimulate acquired


immunity to homologous and often heterologous strains of *P multocida*, and some of these animals become healthy carriers that can provide a source of infection for future outbreaks.

## Clinical Findings

Many cases of HS are peracute and result in death within 8–24 hr. These animals often have fever, hypersalivation, nasal discharge, and difficult respiration, but because of the short duration of disease these signs may easily be overlooked. Acute disease can persist up to 3 days, and less often 5 days, and is characterized by fever of 104°–106°F (40°–41.1°C), apathy or restlessness and reluctance to move, hypersalivation, lacrimation, nasal discharge that begins as serous and progresses to mucopurulent, subcutaneous swelling in the pharyngeal region that extends to the ventral neck and brisket (and sometimes the forelegs), progressive respiratory difficulty, cyanosis, terminal recumbency, and possibly abdominal pain with diarrhea.

### Lesions

The characteristic lesion of HS is swelling of the subcutis and muscle of the submandibular region, neck, and brisket by clear to blood-tinged edema fluid. Serous to serofibrinous fluid may also be present in the thorax, pericardium, and abdominal cavity. There is typically widespread congestion with petechiae and ecchymoses in tissues and on serosal surfaces. Hemorrhages are often most prominent in the pharyngeal and cervical lymph nodes. Pulmonary congestion and edema, sometimes with interstitial pneumonia, and gastroenteritis may occur in some cases.

 Hemorrhagic septicemia, water buffalo



## Diagnosis

Clinical diagnosis of HS in endemic areas is based on history, lapses in vaccination, environmental conditions, and the characteristic clinical signs and lesions of disease. Although typical outbreaks of HS are not difficult to recognize in endemic regions, acute salmonellosis, anthrax, and noninfectious toxicities should also be considered. Sporadic cases are more difficult to diagnose clinically and could be confused with blackleg, lightning strike, or snakebite. A definitive diagnosis of HS is based on isolation of *P multocida* serotype B:2 or E:2 (or other less common serotypes recognized by the OIE as causing HS) from the blood and tissues of an animal with typical signs. Various other *P multocida* serotypes can cause HS-like disease in cattle and water buffalo, which must be differentiated from classical HS. The passive mouse protection test using specific B:2 and E:2 immune rabbit sera has been used in Asia and Africa to identify these serotypes. More precise tests, such as indirect hemagglutination, coagglutination, counter immunoelectrophoresis, and immunodiffusion tests have also been used in some laboratories. More recently, molecular techniques, including pulsed field gel electrophoresis, southern blots, and PCR-based protocols, have been used to differentiate between capsular and somatic serotypes. The PCR techniques are most feasible for use in endemic areas and can be used with various samples, including blood, tissues, or bacteria from broth or plate cultures.

## Treatment and Prevention

Antimicrobials are effective against HS if administered very early in the disease. However, because HS progresses rapidly, therapy is often unsuccessful. During outbreaks, any animal with a fever should be treated with IV antimicrobials as soon as possible to quickly obtain systemic

bactericidal antimicrobial concentrations. Various sulfonamides, tetracyclines, penicillin, gentamicin, kanamycin, ceftiofur, enrofloxacin, tilmicosin, and chloramphenicol have been used effectively to treat HS. However, plasmid- and chromosomal-mediated multidrug resistance seems to be increasing for some strains of *P multocida*, and resistance to tetracyclines and penicillin has been reported for serotype B:2.







Killed vaccines are most commonly used for prevention and include bacterins, alum-precipitated and aluminum hydroxide gel vaccines, and oil-adjuvant vaccines. In animals >3 yr old, an initial two doses, 1–3 months apart, is recommended, followed by annual or biannual booster vaccinations. The oil-adjuvant vaccine provides protection for 9–12 mo and is given annually. Although it provides the best immunity, it is unpopular in the field because of its viscosity and difficulty of administration. Oil-based vaccines combined with tween 80 or saponin have also been used in attempts to increase the ease of administration or immune protection. The commonly used alum-precipitated and aluminum hydroxide gel vaccines have shorter durations of immunity, and twice yearly booster vaccinations are recommended. It is important that the vaccines are made from the strains of *P multocida* circulating in the regions of intended use to obtain maximal effectiveness. Attenuated or modified-live vaccines have been used with some success. A live avirulent vaccine prepared from a *P multocida* serotype B:3(4) of fallow deer origin seems effective and is recommended for use by the Food and Agricultural Organization of the United Nations (FAO) in southeast Asia. Various modified-live and subunit vaccines made from either purified or recombinant bacterial components have also been investigated experimentally.

## Zoonotic Risk

The *P multocida* serotypes that cause HS have not been recovered from human infections. However, because many serotypes of *P multocida* have the potential to infect people, appropriate precautions should be taken when dealing with suspected cases of HS or HS-like disease.

Last full review/revision May 2014 by Derek A. Mosier, DVM, PhD, DACVP

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