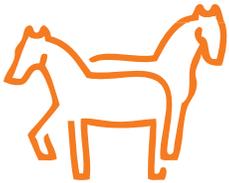


# TECHNICAL BULLETIN

October 2013



## Equine strangles: An update on recommendations for diagnosis, treatment, and prevention including long-term local eradication

**Emily A. Graves, VMD, MS, Dipl. ACVIM**

Zoetis

100 Campus Drive

Florham Park, NJ 07932

One of the most important challenges facing equine practitioners is to effectively control and help prevent highly contagious *Streptococcus equi ssp equi* infections, or strangles. Strangles is not only one of the most frequently diagnosed infectious diseases of horses worldwide,<sup>1-3</sup> but it also has a number of potentially fatal complications and the capability of persistent infection in populations of asymptomatic carrier horses.<sup>4-10</sup> In fact, it is estimated that half of all strangles outbreaks produce at least one carrier horse, creating a reservoir of chronically infected animals that perpetuate the disease in the highly mobile equine population.<sup>6,11</sup> When complications occur, strangles changes from a self-resolving upper respiratory infection to a disseminated disease with a mortality rate as high as 40%.<sup>10</sup> As one expert has noted, strangles is “a highly dangerous disease” and detection of *S. equi ssp equi* in any horse is significant.<sup>6</sup>

While our understanding of *S. equi ssp equi* and the pathogenesis of strangles has grown, the typical approaches to controlling the disease have not advanced appreciably in the past decade. As a result, strangles continues to be widespread, clinically significant, and economically damaging to the equine industry. The National Animal

Health Monitoring System (NAHMS) compiled epidemiologic data from more than 1,000 farms in all regions of the U.S. This extensive survey found that strangles was the cause of approximately 20% of infectious upper respiratory disease in horses (Table 1), the obligatory host species.<sup>12</sup> Other studies have reported much higher morbidity rates in susceptible populations. For example, University of Pennsylvania investigators identified a 1-year old horse as the index case in a strangles outbreak that resulted in confirmed *S. equi ssp equi* infections in 61% (31/51) of horses with clinical strangles.<sup>9</sup> A European study of three protracted strangles outbreaks found that between 29 and 52% of horses had laboratory confirmed *S. equi ssp equi* infections.<sup>11</sup> *S. equi ssp equi* has an estimated 80% contact attack rate in susceptible horses.<sup>6</sup> In addition, morbidity of >90% and mortality of 10% of clinical cases have been reported.<sup>4,13</sup>

Given the imposing epidemiology and pathology of strangles, it is advisable to have a clearly defined strategy for diagnosing the disease, managing outbreaks, and helping prevent their recurrence. Recognizing this, the American College of Veterinary Internal Medicine (ACVIM) developed a definitive Consensus Statement in 2005 that the

**Table 1 – Prevalence of *Streptococcus equi* infection and sequelae in horses**

Clinical or diagnostic event	Prevalence	References
Morbidity	Overall: 20% Range: 1-100%	4,8,11-13,28
Metastatic strangles	20-28% of affected horses	8,10,18,21
Purpura hemorrhagica	20% of affected horses	8,10,21
Myopathies	20% of affected horses	10,21
<i>S. equi</i> carriage in convalescent, asymptomatic horses (confirmed by endoscopy, guttural pouch or nasopharyngeal wash)	Overall: 10% Range: 9-45%	5,9-11,13,18,20,27
Mortality	Up to 10% of cases overall; 40-72% of cases with complications	4,8,10,13,18

American Association of Equine Practitioners (AAEP) considers to be the principal guideline document for managing strangles.<sup>10</sup> Some of the key aspects of the ACVIM Consensus Statement are summarized in this report. These include a suggested protocol for identifying carrier horses, the all-important source of endemic disease and acute outbreaks. Also discussed is an approach for strangles eradication at the local level, a realistic option that can help curtail the widespread economic loss that this disease inflicts on the equine industry.

### **Rapid pathogenesis increases transmission risk**

Histopathology studies show that within 3 hours after oronasal exposure, *S. equi ssp equi* in small numbers invade the tonsillar crypts

and draining lymph nodes. At 48 hours and with the onset of fever, clumps of *S. equi ssp equi* are visible in the lamina propria of the tonsils and in long, extra-cellular chains in the mandibular and retropharyngeal lymph nodes.<sup>14</sup> The rapid, early pathogenesis of *S. equi ssp equi* infection ensures that bacterial shedding occurs before abscessation appears and perhaps even before onset of fever.<sup>6</sup> Thus, considerable dissemination of *S. equi ssp equi* will have occurred by the time clinical disease is apparent.

The implications for the practitioner are that, once clinical signs of *S. equi ssp equi* infection occur, the battle is half lost. Infected, immunologically naïve horses face an often lengthy convalescence and isolation period, and implementing on-site biosecurity measures can be difficult and protracted. Rather than reacting to an outbreak after

the fact, the better strategy is to preempt exposure using such means as temporary isolation and observation of transient horses, immediate quarantine of clinically sick horses, and identification, isolation, and treatment of persistent *S. equi* carriers. Unlike the genetically related *S. equi ssp zooepidemicus*, *S. equi ssp equi* is not a commensal organism of horses. *S. equi ssp zooepidemicus* requires viral or other coinfection for clinical signs to emerge.<sup>4</sup> *S. equi ssp equi*, on the other hand, is present only in cases of active infection or in convalescent carrier horses. Thus, prevention of exposure and de novo infections is a principal goal of strangles control.

## Recognizing strangles: Clinical signs

In susceptible horses, abrupt onset of fever of 103° F or higher is usually the first sign of strangles, occurring 2 or 3 days after exposure and initial infection with *S. equi ssp equi*. This is followed several days later by serous nasal discharge that turns purulent. Suppurative lymphadenopathy is the most prominent feature of uncomplicated strangles. Swollen, painful lymph nodes appear about a week after infection. The submandibular, retropharyngeal, and parotid lymph nodes are most commonly affected (Figures 1 and 2), often asymmetrically. As the lymph nodes enlarge, upper respiratory obstruction accompanied by dyspnea and dysphagia may develop (hence the name “strangles”) along with a resulting loss of appetite. Tracheostomy may be required in severe cases. As the lymph node abscesses increase in size, they may rupture and drain either externally through the skin or internally into the guttural pouches, offering the horse some relief. Recovery from uncomplicated strangles usually takes 3 to 6 weeks.

Older, previously exposed horses generally have a milder, catarrhal form of strangles limited to upper respiratory signs, minimal if any lymph node abscessation, and a rapid recovery. However, the host’s immune status and virulence of the infective *S. equi ssp equi* strain, rather than the horse’s age, are the determinants of susceptibility. Despite experiencing an attenuated disease, infected



**Figure 1.**

Lymphadenopathy and abscessation occurring 7 to 10 days after *S. equi* infection is a classic sign of strangles in immunologically naive horses. Abscessation may be internal or external, as in this case where retropharyngeal and parotid lymph nodes are draining through the skin.

*Photo courtesy of Corinne R. Sweeney, DVM, University of Pennsylvania School of Veterinary Medicine*

horses with some residual immunity will still shed *S. equi ssp equi*, contaminating the environment or infecting susceptible contact horses.<sup>10</sup> An estimated 70-75% of horses develop a durable, post-convalescent *S. equi ssp equi* immunity lasting 5 years or longer.<sup>10</sup> Horses with a less complete immune responses may become susceptible within a few months.<sup>3,10</sup>

A more detailed description of the clinical signs of strangles appears in the ACVIM Consensus Statement.

**Figure 2.**

Parotid and periorbital swelling abscessation is shown in a horse with strangles.



*Photo courtesy of Rob Arnott, DVM*

## Complications of *S. equi* ssp *equi* infection: Source of increased mortality

Strangles is often characterized as a high-morbidity, low-mortality disease. This description is somewhat misleading because of the impact of the estimated 20% rate of non-respiratory complications (Table 1). These include disseminated or metastatic (“bastard”) strangles, purpura hemorrhagica, and myopathies, including muscle infarctions and rhabdomyolysis with progressive atrophy. Horses that develop these sequelae have a much less favorable prognosis. For example, an outbreak described in the ACVIM Consensus Statement resulted in metastatic strangles in 28% (7/25) of the sick horses, 5 of which had to be euthanized.<sup>10,15</sup>

Metastatic strangles, which involves *S. equi* ssp *equi* abscesses that develop in lymphoid tissue at sites other than the respiratory tract (Figure 3), can cause gastrointestinal pain and complications when the mesenteric lymph nodes are affected. Purpura hemorrhagica is perhaps the most dramatic non-respiratory complication of strangles. This immune-

### Figure 3.

Mesenteric lymph node abscessation is a potentially fatal complication known as bastard strangles, whereby *S. equi* infects lymphoid tissue distant from the original site of respiratory tract infection.



Photo copyright 2012, Richard M. Jakowski, Case #103.02041. Reproduced with permission of the author and publisher.<sup>26</sup>

mediated sequela is a necrotizing vasculitis characterized by edema and localized hemorrhage caused by deposition of *S. equi* ssp *equi* M-protein immune complexes in blood vessel walls. A retrospective case analysis of 53 horses with purpura hemorrhagica determined that 17 were infected with or exposed to *S. equi* ssp *equi*.<sup>16</sup> Among the assortment of profound clinical and pathological effects were subcutaneous edema of all four limbs, reluctance to move, hemorrhages on visible mucous membranes, anorexia, fever, tachycardia, colic, lymph node drainage, hematologic and biochemical abnormalities, and 13.6% mortality in *S. equi*-associated cases.<sup>10,16</sup> The horses involved in this study were aged <1 to 19 years of age. Although horses >5 years old tend to be more resistant to strangles,<sup>4</sup> the age range in this study indicates that prior exposure or immune status is not protective once *S. equi* ssp *equi* complications occur.

Muscle infarction is a rare complication which can be associated with purpura hemorrhagica. A severe vasculopathy results in infarction of skeletal muscle, necrosis, muscle stiffness, and a guarded prognosis. *S. equi* ssp *equi*-associated rhabdomyolysis can produce rapid onset of progressive muscle atrophy, lymphocytic vasculitis, and perivascular fibrosis.<sup>10</sup> When recumbency occurs in such cases, rapid deterioration can be expected despite aggressive antimicrobial and anti-inflammatory treatment.<sup>17</sup>

## The *S. equi* ssp *equi* carrier state: Source of endemic disease

The *S. equi* ssp *equi* carrier state is central to the epidemiology of strangles. In most transiently infected horses, nasal shedding of *S. equi* ssp *equi* usually persists for 2 to 4 weeks and seldom lasts longer than 6 weeks.<sup>10</sup> In contrast, persistently infected horses become carriers for periods varying from months to years after full clinical recovery. A study found that asymptomatic carriers were *S. equi* ssp *equi*-positive for an average of 4.5 months after initial infection, with one

horse remaining infectious for 56 months.<sup>13</sup> A 10% incidence is the generally accepted rate of *S. equi* ssp *equi*-persistent infections.<sup>4,18</sup> However, this rate is considerably less than the 28% incidence reported in a 12-month study of horses that recovered from clinical strangles (n = 205), where 57 were found to be persistent *S. equi* ssp *equi* carriers based on guttural pouch endoscopy and culturing.<sup>5</sup> This unexpectedly high rate of persistent infection underscores the importance of repeated testing of all strangles-convalescent horses to confirm a negative *S. equi* ssp *equi* status.

Guttural pouch infection is the usual site of persistent *S. equi* ssp *equi* infection, with the sinuses also involved in some cases.<sup>7,11,19</sup> The guttural pouch becomes infected soon after oronasal infection when the retropharyngeal lymph nodes rupture. Transient empyema in the guttural pouch then occurs. When the purulent exudate persists, as in chronic cases of strangles, chondroids (inspissated or solidified exudate) develop in the guttural pouch. Chondroids harbor and continuously shed large numbers of *S. equi* ssp *equi* and serve as the source of transmission via sporadic coughing or intermittent nasal discharge by carrier horses that are otherwise clinically normal.

Diagnostic evaluation of the guttural pouches of convalescent and recovered horses is critical for identifying asymptomatic carriers. Repeat culturing or PCR analysis of nasopharyngeal or guttural pouch lavage samples is a reliable method for detecting acute *S. equi* ssp *equi* infection from 24 hours to 3 weeks after onset, and is also the diagnostic gold standard for identifying asymptomatic long-term carrier horses. The ACVIM Consensus Statement recommends analysis of 3 nasopharyngeal swabs or lavages obtained at weekly intervals after clinical recovery.<sup>10</sup> However, European investigators found that repeated testing over 2 to 3 months was useful in identifying persistent infection in asymptomatic carrier horses.<sup>7</sup> They caution that guttural pouch cultures may yield negative results for up to 3 months, after which *S. equi* ssp *equi* shedding can resume.<sup>2</sup> Asymptomatic carriers almost

always have guttural pouch abnormalities, such as chondroids or empyema, that can be detected by endoscopy (Figure 4), even when *S. equi* ssp *equi* culturing results are negative.<sup>4,6,20</sup> Culturing and PCR testing of guttural pouch samples concurrently with endoscopy, particularly when visible pathology is absent, are confirmatory of *S. equi* ssp *equi* infection. In contrast, serologic response to infection is not a reliable indicator of carrier status.<sup>20</sup>

#### Figure 4.

Endoscopy of the guttural pouch of strangles-convalescent or recovered horses is critical for identifying asymptomatic carriers, which will have guttural pouch abnormalities such as chondroids or empyema (inset, present as purulent exudate coating the guttural pouch floor). These features of persistent guttural pouch infection are visible by endoscopy, even in cases where *S. equi* ssp *equi* culturing results are negative.



*Photos courtesy of the School of Veterinary Medicine, University of California-Davis and Dr. Bonnie Rush, Kansas State University.*

## When to use anti-infective treatment

### ***Systemic treatment of acute-phase and metastatic strangles***

Systemic anti-infective (AIF) treatment of uncomplicated, acute-phase strangles is somewhat controversial because it can conceivably limit the affected horse's post-infection immune response, leaving it at risk for re-infection. It should be noted that this is not an evidence-based contraindication supported by experimental or clinical data. The ACVIM Consensus Statement recommends that treatment in these cases be limited to rest and supportive care until the disease runs its course.<sup>10</sup> Horses should be monitored to ensure that they are sufficiently pain-free to continue eating and drinking. The Consensus Statement adds that AIF treatment may be of benefit (1) in new cases immediately after onset of fever by preventing abscessation and limiting the spread of strangles to other horses on the premises, and (2) to decrease abscess size and relieve airway obstruction in horses with lymphadenopathy-associated respiratory distress. Systemic penicillin treatment of horses with metastatic strangles, perhaps with other AIFs to increase the spectrum of activity, has been recommended.<sup>21</sup>

A more detailed discussion of strangles AIF treatment and co-therapies appears in the ACVIM Consensus Statement and elsewhere, and is beyond the scope of this report.

### ***Anti-infective treatment of carrier horses***

Treatment of guttural pouch empyema with saline or polyionic fluids and a local penicillin-gelatin mixture is described in the ACVIM Consensus Statement.<sup>10</sup> An alternative protocol consists of penicillin administered locally into the guttural pouch followed by systemic treatment with procaine penicillin G for 7 days, then oral trimethoprim-sulfadiazine (TMS) for 14 days.<sup>13</sup> Treatment of infected guttural pouches should be combined with physical removal of chondroids, which if left in place, will maintain infection and defeat the effects of AIF treatment.

### ***Which anti-infective agent to use***

*S. equi* ssp *equi* is consistently sensitive to penicillin. Although penicillin is generally acknowledged to be the drug of choice for systemic AIF treatment of strangles, ceftiofur's activity against a broad range of *S. equi* ssp *equi* strains has also been confirmed.<sup>22</sup> Most *S. equi* ssp *equi* isolates are also sensitive to TMS, although exceptions have been noted.<sup>4,18,19</sup> One report noted that initial TMS treatment of guttural pouch carriers of *S. equi* ssp *equi* was unsuccessful in 33% (5/15) of cases.<sup>19</sup> When these horses were then treated with penicillin and ceftiofur, guttural pouch infection and inflammation were eliminated. Antimicrobial sensitivity data for North American equine *S. equi* ssp *equi* isolates found that susceptibility was 100%, 97%, and 72% to ceftiofur, penicillin, and TMS, respectively.<sup>23</sup> Widely accepted guidelines list penicillin, ceftiofur, and TMS as the first, second, and third choices respectively for AIF treatment of strangles caused by *S. equi* ssp *equi*.<sup>23</sup> Although not specifically recommended in the ACVIM Consensus Statement, ampicillin is a member of the beta-lactam antimicrobial class along with penicillin and ceftiofur, and has been shown to have activity against *S. equi* ssp *equi* comparable to the other two drugs.<sup>23</sup>

## Local eradication and control

When strangles outbreaks occur at multi-horse sites, the task of eradicating the disease and reestablishing biosecurity is invariably difficult and lengthy. Acutely infected and carrier horses need to be identified, isolated, and in some cases treated, followed by diagnostic monitoring of affected and suspect horses for extended periods. This process becomes more difficult as the mobility and the size of the on-site horse population increase, with premises of >100 horses being at substantially greater risk.<sup>3</sup> Despite these hurdles, local eradication is a realistic possibility. The following recommendations for maintaining an *S. equi* ssp *equi*-free site and eradicating infection following outbreaks summarize the guidelines in the ACVIM Consensus Statement and other sources.

### **Observation and screening of newly arriving horses**

Routine screening of horses that are introduced onto a farm can be difficult, especially at sites with a large, transient population. The ACVIM Consensus Statement recommends quarantining newly arriving horses for 3 weeks and screening them for *S. equi ssp equi* by multiple nasopharyngeal swabs, measures that many horse owners would consider to be onerous.<sup>10</sup> However, to the extent possible, horses entering the premises should be observed for signs of strangles for at least 2 weeks before being mingled with resident horses.<sup>3</sup> Horses that remain clinically asymptomatic after this period are at low risk of transmitting disease. A useful screening method short of diagnostic testing is to take daily (twice-daily is better) rectal temperatures of newly arriving horses for the 2-week holding period. Horses with temperature elevations  $\geq 101.5^\circ$  F should be considered suspect. Because fever usually precedes *S. equi ssp equi* shedding by 24-48 hours, prompt isolation of horses after pyrexia is detected will help prevent transmission to other horses.

### **Prompt identification and isolation of clinically affected or suspect horses**

Clinically affected or *S. equi ssp equi*-positive horses should be quarantined and movement of horses on and off the premises should be stopped until all infected and contact horses are found to be diagnostically negative.

### **Diagnostic monitoring of convalescent or contact horses**

At least 3 post-recovery nasopharyngeal swabs or lavages should be obtained at weekly intervals from convalescent horses for culturing and PCR testing. Horses that test negative on consecutive tests may be safely returned to the non-quarantine area.

### **Detection and monitoring of carrier status**

Asymptomatic convalescent horses should be evaluated by guttural pouch endoscopy and testing of nasopharyngeal or guttural pouch lavage samples to determine if they are *S. equi ssp equi* carriers. As noted earlier

in this report, a minimum of 3 consecutive weekly samples should be tested. Additional, subsequent tests will help provide added assurance of negative carrier status.

### **Treatment of guttural pouch infections and carrier horses**

Topical and systemic AIF treatment should be administered to *S. equi ssp equi* carriers to eliminate guttural pouch infection, and guttural pouch chondroids should be removed.

### **Vaccination**

The AAEP classifies strangles vaccination as risk-based, rather than a core, equine vaccination, meaning that vaccination should be based on a risk-benefit assessment by the attending veterinarian in conjunction with the horse owner. AAEP guidelines recommend vaccination against *S. equi ssp equi* on premises where (1) strangles is a persistent, endemic problem or (2) a high risk of exposure is anticipated. Risks for strangles exposure include high stocking density, frequency of travel, comingling with horses from other locations, and susceptibility due to lack of prior vaccination or natural exposure to *S. equi ssp equi*. Given the prevalence of strangles, its potential for causing debilitating clinical disease and costly treatment, and the presumed benefits of herd immunity, vaccination is advisable for all healthy horses that meet these risk criteria.

Two types of equine *S. equi ssp equi* vaccines are available in the U.S. A parenteral, inactivated *S. equi ssp equi* extract vaccine given in 3 primary doses has been shown to reduce the clinical attack rate by 50% in vaccinated horses.<sup>3,10</sup> A modified-live *S. equi ssp equi* vaccine given by the intranasal (IN) route (Pinnacle<sup>®</sup> I.N., Zoetis Inc.) is administered in two primary doses 2-3 weeks apart followed by annual revaccination (see box). The IN vaccine stimulates mucosal immunity in the nasopharyngeal tract, mimicking the immune response to natural infection.<sup>3</sup>

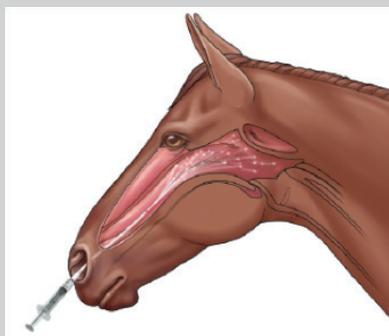
### **Sanitation, hygiene, and pasture management on contaminated premises**

Organic material that infected horses came in contact with should be removed from stables. Contaminated facilities, equipment, and vehicles should be cleaned and disinfected. To effectively disinfect, organic debris should be removed first, followed by washing with mild soap and rinsing. Several types of disinfectants are appropriate for use on *S. equi ssp equi* contaminated premises, including dilute bleach solutions, iodophors (povidone iodine), quaternary ammonium compounds (such Roccal-D), and Virkon. Cost and efficacy will guide the choice of disinfectant. Disinfectant labels will list those

bacteria against which it is effective. Water troughs, a common site of *S. equi ssp equi* transmission, should receive special attention during an outbreak, including once daily cleaning and disinfection. Pastures where infected horses grazed should be rested for 4-6 weeks.<sup>18,21</sup> Personnel handling sick horses should observe barrier precautions, including use of disposable gloves, washing or disposal of outer garments, and use of foot baths. Handlers should perform frequent hand-washing or use of sanitizer before contact with individual horses. Handlers should also work with sick horses after all work with healthy horses has been completed.

### **Protection induced by PINNACLE I.N. helps prevent disease caused by *S. equi ssp equi***

*S. equi ssp equi* produces a surface virulence factor known as the M protein (SeM), unique to streptococci. SeM inhibits phagocytosis by lymphocytes of the host's innate immune system. Horses that recover from *S. equi ssp equi* infection develop both serum and mucosal SeM immunoglobulins. However, mucosal SeM immunoglobulins appear to play a prominent role in protection at the local, nasopharyngeal site of initial *S. equi ssp equi* infection.<sup>10,24</sup> Inactivated, intramuscular *S. equi ssp equi* vaccines stimulate a serum SeM immunoglobulin response but appears not to induce mucosal SeM immunoglobulins.<sup>3,25</sup> This may explain why injectable, inactivated *S. equi ssp equi* vaccines help reduce the *S. equi ssp equi* attack rate by only an estimated 50%.<sup>10</sup> In contrast, IN vaccination with PINNACLE I.N. has been shown to produce both serum and mucosal SeM immunoglobulins,<sup>3</sup> an immune response that mimics what occurs following natural infection.<sup>3,10,24</sup>



Because it is given intranasally, PINNACLE I.N. avoids local, injection-site reactions that can occur with parenteral *S. equi ssp equi* vaccines.<sup>10</sup> A field study of 582 horses given 2 primary doses of PINNACLE I.N. found that 5 of 582 first-time vaccinates had mild local or systemic reactions, and 0 of 582 second-time vaccinates had post-vaccination reactions, a 0.43% incidence (Data on file, Study Report No. 22741, Zoetis Inc.).<sup>29</sup>

***After aseptic rehydration, PINNACLE I.N. is instilled into one nostril with a specially designed cannula. Proper placement of the cannula is important to ensure that vaccine is delivered to the pharyngeal area, where it can elicit a mucosal immune response at the site of natural infection.***

## A biosurveillance opportunity

In most cases, veterinarians diagnose and treat equine respiratory disease empirically in order to respond in a timely manner and to minimize costs. Going to the inconvenience and expense of obtaining a laboratory diagnosis based on blood samples or nasal swabs is the exception in clinical practice. Zoetis offers a no-charge biosurveillance service to aid veterinarians in obtaining a laboratory diagnosis and case resolution for horses with clinical infectious respiratory disease. As data accumulate, the Veterinary Medical Information and Product Support (VMIPS) group at Zoetis monitors epidemiologic trends regionally and nationally. Individual practitioners can benefit by obtaining a free, laboratory-confirmed diagnosis and by monitoring disease incidence on specific farms.

Results of ongoing surveillance programs can yield unexpected results. To illustrate, equine influenza virus (EIV) is the most commonly diagnosed cause of infectious respiratory disease in some equine populations.<sup>30</sup> However, recent 2-year data compiled at the University of California-Davis (UC-D) found that the incidence of equine herpesvirus type 4 was 37% greater than that for EIV (82/761 versus 60/761 clinical respiratory cases).<sup>28</sup> *S. equi ssp equi* was diagnosed in 6.4% (49/761) of cases.

Samples from horses with clinical respiratory disease can be submitted to the Equine Infectious Disease Research Laboratory, University of California-Davis (UC-D) for respiratory panel testing. Details on the biosurveillance program can be obtained from Zoetis by contacting VMIPS at 800-366-5288 or from UC-D at 530-752-7991.

## Summary

Equine veterinarians and horse owners should be vigilant for outbreaks of strangles due to the disease's high prevalence, potentially fatal complications, and the difficulty and cost of recovery, site clean-up, and eradication. Because uncomplicated strangles is self-

limiting, AIF treatment is generally reserved for horses immediately following onset of clinical signs to preempt abscessation and *S. equi ssp equi* transmission to other horses, horses with severe lymphadenopathy that restricts breathing, and horses with guttural pouch infections that are the source of a persistent carrier state.

Effective prevention and control of strangles is heavily dependent on physical isolation and restricted movement of sick or at-risk horses in order to limit *S. equi ssp equi* exposure and transmission. Isolation of affected horses should be accompanied by diagnostic monitoring of infection status based on culturing and PCR testing of nasopharyngeal swabs or lavage samples.

Recovering, asymptomatic horses infected with *S. equi ssp equi* are the source of long-term shedding and endemic disease. Identification of persistently infected horses is accomplished by testing of nasopharyngeal or, preferably, guttural pouch lavage samples for a minimum of 3 weeks after clinical signs have resolved. Endoscopy will further reveal *S. equi ssp equi*-associated guttural-pouch abnormalities such as empyema and chondroids.

Vaccination can be an important component of a strangles control program for horses in an endemic area or in frequent contact with horses of unknown *S. equi ssp equi*-infection status. PINNACLE I.N., a modified-live vaccine given by the IN route, has the advantage of helping confer systemic immunity, as well as mucosal immunity at the nasopharyngeal site of natural infection. IN vaccination also avoids local injection-site reactions.

Long-term eradication of strangles on premises where outbreaks have occurred can be made feasible by implementing a program of diagnostic monitoring to detect and treat *S. equi ssp equi* carrier horses, careful sanitation to eliminate environmental *S. equi ssp equi* contamination, and vaccination of susceptible, at-risk horses.

## Acknowledgment

The authors acknowledge the contribution of Mark Dana of Scientific Communications Services, LLC in the writing and editing of this report.

## References

1. Harris SR, Webb K, Aanensen DM, et al. Diversity, microevolution and within-host niche adaptation of *Streptococcus equi*. *J Equine Sci*. 2012;32:S12.
2. Smith PA. How to eliminate strangles infections caused by silent carriers. *AAEP Proceedings*. 2006;52:101-103.
3. Timoney JR. Equine strangles: 1999. *AAEP Proceedings*. 1999;45:31-37.
4. Ainsworth DM, Hackett RP. Disorders of the Respiratory System. In: Reed SM, Bayly WM, Sellon DC, eds. *Equine Internal Medicine*. St. Louis, MO: Saunders; 2004:289-353.
5. Kemp-Symonds J, Butcher N, Mitchell Z, et al. Epidemiology of *Streptococcus equi* carriers and the development of diagnostic ELISAs for strangles: Preliminary findings. *Proc 10th Intern Cong World Equine Vet Assoc*. 2008;431. Available at: <http://www.ivis.org/proceedings/weva/2008/shortcom1/19.pdf?LA=1>. Accessed August 2, 2013.
6. Knottenbelt DC. Strangles update. *Proc 10th Intern Cong World Equine Vet Assoc*. 2008;65-68. Available at: <http://www.ivis.org/proceedings/weva/2008/mainsession1/8.pdf?LA=1>. Accessed August 3, 2013.
7. Newton JR, Wood JL, Dunn KA, et al. Naturally occurring persistent and asymptomatic infection of the guttural pouches of horses with *Streptococcus equi*. *Vet Rec*. 1997;140:84-90.
8. Sweeney CR, Whitlock RH, Meirs DA, et al. Complications associated with *Streptococcus equi* infection on a horse farm. *J Am Vet Med Assoc*. 1987;191:1446-1448.
9. Sweeney CR, Benson CE, Whitlock RH, et al. Description of an epizootic and persistence of *Streptococcus equi* infections in horses. *J Am Vet Med Assoc*. 1989;194:1281-1286.
10. Sweeney CR, Timoney JF, Newton, JR, Hines MT. *Streptococcus equi* infections in horses: Guidelines for treatment, control, and prevention of strangles. *J Vet Intern Med*. 2005;19:123-134.
11. Newton JR, Verheyen K, Talbot NC, et al. Control of strangles outbreaks by isolation of guttural pouch carriers identified using PCR and culture of *Streptococcus equi*. *Equine Vet J*. 2000;32:515-526.
12. Gross DK, Morley PS, Traub-Dargatz J, et al. A national estimate of acute infectious upper respiratory disease (IURD) and risk factors associated with infection for horses in the United States during 1998-1999. *AAEP Proceedings*. 2000;46:274-276.
13. Holland RE, Harris DG, Monge A. How to control strangles infections on the endemic farm. *AAEP Proceedings*. 2006;52:78-80.
14. Timoney JR, Kumar P. Early pathogenesis of equine *Streptococcus equi* infection (strangles). *Equine Vet J*. 2008;40:637-642.
15. Spoor-makers TJ, Ensink JM, Goehring LS, et al. Brain abscesses as a metastatic manifestation of strangles: symptomatology and the use of magnetic resonance imaging as a diagnostic aid. *Equine Vet J*. 2003;35:146-151.
16. Pusterla N, Watson JL, Affolter VK, et al. Purpura haemorrhadica in 53 horses. *Vet Rec*. 2003;153:118-121.
17. Sponseller BT, Valberg SJ, Tennet-Brown BS, et al. Severe acute rhabdomyolysis associated with *Streptococcus equi* infection. *J Am Vet Med Assoc*. 2005;227:1800-1807.

18. Hines MT. Strangles: Is there a consensus? *Proceedings North Am Vet Conf 2005*. 2005;191-194. Available at: <http://www.ivis.org/proceedings/navc/2005/LA/086.pdf?LA=1>. Accessed August 2, 2013.
19. Verheyen K, Newton JR, Talbot NC, et al. Elimination of guttural pouch infection and inflammation in asymptomatic carriers of *Streptococcus equi*. *Equine Vet J*. 2000;32:527-532.
20. Davidson A, Traub-Dargatz JL, Magnuson R, et al. Lack of correlation between antibody titers to fibrinogen-binding protein of *Streptococcus equi* and persistent carriers of strangles. *J Vet Diagn Invest*. 2008;20:457-462.
21. Ainsworth DM. Caught in the middle of a strangles outbreak – Review of the diagnostic and management procedures. *Proc AAEP Focus on Upper and Lower Respiratory Diseases*. 2010;49-2010.
22. Salmon SA, Watts JL, Yancey RJ Jr. In vitro activity of ceftiofur and its primary metabolite, desfuroylceftiofur, against organisms of veterinary importance. *J Vet Diagn Invest*. 1996;8:332-336.
23. Weese JS, Baptiste KE, Baverud V, et al. Guidelines for antimicrobial use in horses. In: Guardabassi L, Jensen LB, Kruse H, eds. *Guide to Antimicrobial Use in Animals*. Oxford, UK: Blackwell Publishing; 2008:161-182.
24. Hamlen HJ, Timoney JF, Bell RJ. Epidemiologic and immunologic characteristics of *Streptococcus equi* infection in foals. *J Am Vet Med Assoc*. 1994;204:768-775.
25. Sheoran AS, Sponseller BT, Holmes MA, et al. Serum and mucosal antibody isotype responses to M-like protein (SeM) of *Streptococcus equi* in convalescent and vaccinated horses. *Vet Immunol Immunopathol*. 1997;59:239-251.
26. Jakowski RM. *OCW Veterinary Gross Pathology Image Collection*, “Bacterial Disease of the Hemolymphatic System.” Published in Tufts OpenCourseWare (2005-2013). Available at: <http://ocw.tufts.edu/Content/72/imagegallery/1362322/1368983/1377573>. Accessed August 7, 2013. Licensed under the Creative Commons Attribution-Noncommercial-Share Alike 3.0 Unported License.
27. Kelly C, Bugg M, Robinson C, et al. Sequence variation of the SeM gene of *Streptococcus equi* allows discrimination of the source of strangles outbreaks. *J Clin Microbiol*. 2006;44:480-486.
28. Pusterla N, Kass PH, Mapes S, et al. Surveillance programme for important equine infectious respiratory pathogens in the USA. *Vet Rec*. 2011;169:12.
29. Data on file, Study Report No. 22741, Zoetis Inc.
30. Mumford EL, Traub-Dargatz JL, Salma MD, et al. Monitoring and detection of acute viral respiratory tract disease in horses. *J Am Vet Med Assoc*. 1998;213:385-390.

