

Satellite Article

Epidemic disease caused by Equine herpesvirus-1: recommendations for prevention and control

G. P. ALLEN

Department of Veterinary Science, M. H. Gluck Equine Research Center, University of Kentucky, Lexington, Kentucky 40546-0099, USA.

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Introduction

Of all infectious viral diseases recognised in horses, equine herpesvirus-1 (EHV-1) is one of the most prevalent (Allen *et al.* 2002). The most devastating manifestations of infection are large-scale outbreaks of abortion, perinatal foal mortality or myeloencephalopathy (Dixon *et al.* 1978; Hartley and Dixon 1979; Greenwood and Simpson 1980; Mumford *et al.* 1987; McCartan *et al.* 1995). Epidemics of the 3 clinical entities may occur separately or concurrently within a horse population; and each has the potential for huge attendant economic and equine welfare consequences. The risk of spread of EHV-1 infection to new horse groups is high, epidemics spread rapidly, and a large number of clinical cases may reasonably be expected.

Numerous recent outbreaks of EHV-1 abortigenic or paralytic disease associated with high attack rates have led to requests for specific information about the management of such outbreaks and for suggestions on practical and effective ways to prevent or limit the spread of epidemic EHV-1 infection. **This article provides practical recommendations, based on the most current understanding of the epidemiological characteristics of EHV-1, that are intended for use in guiding veterinary and farm management personnel in their attempts to prevent, manage and control epidemic EHV-1 disease.**

Epidemiology

EHV-1 is deeply entrenched in most horse populations. The herpesvirus has evolved to occupy a **unique ecological niche** within the horse that allows viral persistence over the lifetime of the individual animal. **The ultimate epidemiological reservoir** for EHV-1 is the large and globally distributed pool of latently infected, intermittently shedding carrier horses, which may comprise up to half of a given horse population

(Edington *et al.* 1994). Although exhibiting low genotypic diversity and very little antigenic polymorphism, **EHV-1 has the potential for increasing its virulence** and expanding its cellular tropism (Rebhun *et al.* 1988; Smith *et al.* 1999). Three biological sources of the virus may serve as direct origins of natural infection for the susceptible horse:

- **an actively infected horse** releasing progeny EHV-1 into its nasopharyngeal secretions;
- **the fetus, fetal membranes or reproductive tract** secretions of a mare immediately following an EHV-1 abortion;
- **endogenous virus reactivated from its quiescent state** to full infectivity within a latently infected carrier horse.

The constellation of symptomatology caused by EHV-1 - epidemic abortion, myeloencephalopathy and perinatal foal death - are highly contagious viral diseases. The **principal mode of horse-to-horse transmission of EHV-1** is through close, intimate contact with virus-containing secretions (from the nasopharynx, reproductive tract, or an aborted fetus) of another infected horse. The **only receptive portal of entry** for EHV-1 is the mucosal epithelium of the upper respiratory tract through which infection takes place. Evidence also supports the **spread of EHV-1 among horses over limited distances** (e.g. between paddocks, stalls and fields) by fine, airborne droplets of virus-laden secretions generated through the snorting or coughing of actively shedding horses.

Transmission of EHV-1 by contaminated hands, boots and clothing of personnel and by virus-contaminated water and feed may also occur. Iatrogenic spread of equine herpesvirus through the multiple use of **nondisinfected diagnostic utensils** (e.g. endoscopes) is also possible.

The communicability of an EHV-1-infected horse is greatest during the first few days of respiratory tract infection. In young horses experiencing their first infection with EHV-1, this period of maximal contagiousness coincides with the febrile phase of respiratory infection during which the virus is

excreted in high numbers (up to 10^6 infectious particles per swab) into a copious outpouring of nasal discharge. **Nasal shedding** of EHV-1 may occur for as long as 14 consecutive days in immunologically naive animals. In older, **previously infected horses**, both pyrexia and nasal discharge after re-exposure to EHV-1 are minimal, more transient, and often go unnoticed by attending personnel. In such immunologically conditioned horses experiencing clinically inapparent EHV-1 respiratory infection, the levels of virus excreted into the nasal mucus are reduced by several orders of magnitude.

Clinical EHV-1 respiratory disease appears between 3 and 6 days following natural exposure to the virus. The incubation period for EHV-1 abortion ranges from 7 days to several months after respiratory challenge with EHV-1 (Bryans and Allen 1989). Signs of EHV-1-associated **central nervous system disorders**, when present, usually appear during the second week following respiratory tract exposure to the virus. Horses immunosensitised to EHV-1 by prior infection are at greater risk than naive animals for development of neurological manifestations of virus infection.

The cardinal elements of all infectious disease management policies, upon which the procedures recommended herein for controlling EHV-1 epidemic disease are founded, are:

- to **minimise** both exogenous and endogenous introduction of infectious virus into closed groups of animals (**reduce exposure to pathogen**);
- to maintain the highest achievable level of both innate and immunological resistance to the viral pathogen (**minimise clinical effects of incognisant exposure**);
- to prevent the transmission of virus infection from affected to nonaffected groups of animals (**contain the infection**).

Preventative measures to reduce the risks for acquisition of EHV-1 infection

The most effective measures for preventing the initiation of EHV-1 infection in a population of horses are those that minimise exposure of the animals to contact with the 3 known biological sources of infectious virus (**see Epidemiology section**). The preventative strategies addressed in this document include:

- **vaccine maintenance** of a herd-wide, efficacious level of immune-mediated resistance to EHV-1 infection;
- **subdivision and maintenance** of the farm population of horses as smaller, physically separate and epidemiologically isolated subgroups;
- implementation of management practices designed to **minimise the risks of exogenous** introduction of infectious EHV-1 into established groups of horses;
- minimalisation of management practices associated with the risk of **endogenous** introduction of infectious EHV-1 as the result of stress-induced reactivation of latent virus from chronic carrier horses resident within an isolated group (**Fig 1**).

Prophylactic immunisation

This much may be stated dogmatically: severe outbreaks of EHV-1 abortion, perinatal foal death and myeloencephalopathy may occur despite rigorous and regular vaccination with commercial preparations of currently marketed vaccines. Such vaccine breaks have generally been attributed to:

- **exposure to a quantity of virus** inoculum sufficient to overwhelm existing vaccine immunity;
- **exposure to a particularly aggressive** (i.e. hypervirulent) strain of virus;
- **inherently suboptimal performance of the current generation of EHV-1 vaccines.**

Whatever the underlying cause, the ever-present potential for vaccination failure with EHV-1 necessitates integrated, herd infection-control programmes that are not dependent solely on the benefits of vaccination for prevention of epidemic EHV-1 disease.

Notwithstanding this recognised efficacy limitation of present day EHV-1 vaccines, the planned, regular stimulation of the immune system of horses by immunisation with viral antigens remains a major component of the overall defense against EHV-1 epidemic disease (Wilson 1996; Lunn and Townsend 2000). Maximal vaccine effectiveness is achieved when vaccination coverage rates are high enough to realise the added benefits of herd immunity (e.g. reduction in contact transmission).

Careful epidemiological records of EHV-1 abortions kept over the past 40 years among the large Thoroughbred broodmare population of central Kentucky indicate that a combination of vaccination and additional herd management procedures described herein have brought about a significant reduction in both the annual incidence and size of multiple-case abortion storms caused by the herpesvirus (Vickers and Powell 2001).

The contributive effect to this observed decrease in epidemic EHV-1 abortion in Kentucky of vaccines whose principal activity is the induction of circulating, virus-neutralising antibody is thought to be antibody-mediated inactivation of the virus at one of the few obligate extracellular phases of its pathogenic cycle; the transfer of virus reactivated from latently infected lymphocytes to the respiratory epithelium. Such an immune block to the infection of respiratory tract epithelial cells by reactivated EHV-1 present within the underlying connective tissue would prevent virus from reaching the site in which it is able to replicate and release infectious progeny virions into the nasal mucus. This scenario is consistent with that seen over the past 4 decades in Kentucky broodmares; i.e. **an unchanging incidence of single, sporadic abortions due to endogenous infection of the fetus by EHV-1 reactivated from latency within the dam** (i.e. **reactivation disease**), but a dramatic reduction in the number of such virus reactivation events that subsequently progress to the stage of nasal shedding of infectious virus for transmission to pregnant herdmates with consequent storms of abortion (i.e. **dissemination disease**).

Implementation of preventative, herd-management practices

i. The concept of small, epidemiologically isolated groups of horses

The potential for equine losses during an outbreak of EHV-1 disease is related directly to herd size. **No infection control strategy has so great a bearing on the success in controlling the magnitude of epidemic EHV-1 disease as that of the subdivision of the at-risk population of horses into smaller groups and the maintenance of those groups as closed, physically separated units** (Bryans 1981; Bryans and Allen 1986).

For maximal protection, group size should be as small as the physical facilities allow, with each group kept under conditions that limit the transmission of virus between established groups. In the absence of viral transmission between horse groups, the magnitude of an outbreak of EHV-1 abortion, perinatal foal death or myeloencephalopathy is then limited to the number of animals comprising the largest group.

Horses should, if feasible, be segregated into like groups that avoid the mixing of yearlings with older animals, pregnant with nonpregnant mares, horses with donkeys, etc. For pregnant mares, it is important that foaling groups (a) be established early in gestation to allow for the establishment of social hierarchies prior to the increased vulnerability to EHV-1 abortion during late gestation; (b) be assembled on the basis of similar expected delivery dates and (c) do not combine first-foal mares with older broodmares (Powell 1992; Anon 2000; Vickers and Powell 2001).

A large, densely populated group of susceptible horses, in which **no restrictions on movement into and out of the group are in place, provide a setting uniquely conducive to the spread of infectious EHV-1 and carry a great potential for explosive outbreaks of disease.**

ii. Prevention of exogenous introduction of infectious EHV-1 into isolated horse groups

Close contact with another infected horse is by far the most frequent exogenous source of EHV-1 responsible for initiating outbreaks of disease among horses within a closed group. Procedures designed to prevent any deliberate or inadvertent contact with horses outside epidemiologically isolated groups should, therefore, be strictly followed. The risk of exogenous infection by EHV-1 is reduced by a space barrier between paddocks housing different horse groups that is sufficient to prevent aerosol transmission between the groups.

The greatest danger for exogenous infection lies in the introduction of new horses into established groups, particularly those with recent opportunities for exposure to large, intermingled assemblages of horses from diverse sources (e.g. sales, shows, racetracks, competitions, training centres). All horses, even those without overt signs of

disease, must be considered as possible carriers of EHV-1 with the potential for disseminating infectious virus. The addition of new animals, including foster mares, into horse groups should if at all possible be avoided or, alternately, preceded by **a 21-day period of isolation**. A horse temporarily removed from a group for purposes that may involve prolonged transport or its contact with other horses (e.g. breeding, showing, training, veterinary care, sales) should also undergo a 21-day period in isolation before returning to its resident group.

iii. Prevention of endogenous introduction of infectious EHV-1 in isolated horse groups

Because of the endemicity of EHV-1, frequent occurrences of clinical disease are seen in closed groups of horses in which no contact with outside animals can be identified. Most horses become latent, intermittently shedding carriers of the herpesvirus after a primary infection, and nearly every foal is recruited as a new host into this biological life cycle of the virus. **Because EHV-1 latency is a common and lifelong condition, any previously infected horse should be regarded as a potential and permanent source of infection for other horses in its surroundings.** The latently infected horse is usually a silent carrier who, in response to external stressors, sheds infectious EHV-1 into the environment. This section focuses on preventative measures that enable the horse to control the frequency and magnitude of the repeating cycles of reactivation, nasal shedding and subsequent dissemination of EHV-1.

The high prevalence of EHV-1 latency in horses precludes the practice of identifying and separating latent carrier horses from the herd. Measures to control the frequency of reactivation of latent EHV-1 are aimed at minimising stress experienced by horses - stress caused by crowding, poor nutritional state, heavy parasite infestation, lengthy transport, disruption of established social groups, inclement weather, *en masse* weaning, other disease states (Bryans 1981).

The 3 described preventative strategies for EHV-1 epidemics (**vaccination, subdivision with subsequent isolation and stress reduction**) are not isolated actions. Each is an integral part of the overall process of preventing and controlling the magnitude of epidemic EHV-1 disease. The omission of any one of the interrelated procedures may compromise the odds for success.

Implementation of such preventative strategies for EHV-1 adds financial and labour burdens to horse management operations. The extent of the precautions necessary depends upon the nature of the individual operation. The rigour of EHV-1 control policies that are put in place on an equine dwelling involves infection-control decisions which, in the end, must be a compromise between the level of risk reduction desired and the practicability and economic feasibility for the given equine operation to implement the corresponding control procedures.

Outbreak management and control

The priorities for management of an active outbreak of EHV-1 disease are:

- **establishment of an early diagnosis;**
- **prevention of subsequent spread** of the virus from initially infected horses to other members of the group and to other horse groups both on and off the affected dwelling;
- **therapeutic treatment of individual clinical cases** (Anon 1994; Anon 1997; Anon 2000; Donaldson and Sweeney 1997; Wilson 1997).

Early diagnosis

EHV-1 epidemics evolve rapidly and require immediate intervention. Early laboratory identification of EHV-1 as the aetiological agent of an outbreak of epidemic abortion, myeloencephalopathy or neonatal foal mortality is, therefore, necessary for rapid execution of the most appropriate control efforts. Furthermore, the **success of applied control measures is greatest when intervention is initiated early** on the ascending limb of the epidemic curve.

A definitive *antemortem* diagnosis of EHV-1 infection in the horse can best be achieved by laboratory isolation of the virus from either (a) **samples of nasopharyngeal exudate** collected on a dacron-tipped swab and held in a small volume of transport liquid or gel, or (b) **20 ml venous blood collected in anticoagulant** (heparin, EDTA or citrate). **Both specimens for attempted virus isolation should be collected as early as possible after the onset of illness and transported immediately to the diagnostic laboratory in a chilled (not frozen) environment.**

Postmortem diagnosis of EHV-1 can be made by virus isolation, immunohistochemistry, polymerase chain reaction (PCR) or histopathological examination on **appropriate tissues collected during necropsy: lung, liver, spleen and thymus** from aborted fetuses and newborn foals; **spinal cord and brain tissue from cases of neurological disease.** Detailed procedures for diagnosis of EHV-1 infections in the horse have been reported recently (Allen 2000).

While not conclusive and often difficult to interpret in vaccinated horses, **serological evidence** for recent infection by EHV-1 can be obtained by laboratory examination of single collections of serum by complement-fixation (McCarten *et al.* 1995) or recombinant glycoprotein-G ELISA tests (Crabb *et al.* 1995). Retrospective serodiagnosis of EHV-1 infection can be accomplished by demonstration of a 4-fold rise in antiviral titre between acute- and convalescent-phase serum samples.

Prevention of the transmission of EHV-1 infection to other horses

This section describes actions that may be taken for minimising the spread of EHV-1 epidemic disease through implementation

of the practices of isolation, quarantine and disinfection (**Fig 1**). **The underlying goal of outbreak control procedures is to halt the further spread of EHV-1 infection beyond the affected group of horses to surrounding groups both on and off the affected premises.**

i. Isolate the focus of infection

Clinically or subclinically infected horses, including aborted fetuses, serve as the primary source of contagious EHV-1 for spread to other horses. Aborted fetuses and the afterbirth should be **sealed in plastic bags** and transported to a laboratory in a manner that avoids further contamination of the environment with the virus (e.g. dragging of the nonbagged fetus). In a suspected EHV-1 outbreak, aborting mares, sick newborns and any horses exhibiting pyrexia, nasal discharge or gait abnormalities should be removed from their resident group and kept in **physical isolation** away from the remainder of the horse population until all danger of conveying the infection has passed. The building of space, sanitary and immunological barriers around the reservoir of infected animals held in isolation is also important for reliable containment of infection to its original focus.

ii. Interrupt the routes of virus transmission

a) **The creation of space and sanitary barriers** around the isolated focus of infection is important for controlling indirect transfer of the virus by airborne transmission and inanimate objects, respectively. A zone of empty space sufficient to serve as a barrier to the airborne spread of EHV-1 should surround the isolation area.

The objective of building a sanitary environment around infected animals held in isolation is achieved by the **use of chemical disinfectants** to inactivate virus encountered outside the body of the contagious horse on contaminated hands, shoes or clothing of animal care staff; lead ropes; feed or water buckets; bedding; veterinary instruments etc. that may serve as inanimate vehicles for virus spread. **After an abortion caused by EHV-1, the bedding should be disinfected and removed, the stall cleaned and disinfected, and the external genitalia of the aborting mare washed thoroughly before transfer to isolation.**

Personnel caring for animals placed in isolation should be educated about the specific procedures to be used for controlling the further spread of infection. Such staff should wear **protective outer clothing, disposable latex gloves and disinfectant-immersible footwear**, all of which should be removed upon leaving the isolation area. Frequent hand washing and the use of disinfecting footbaths are beneficial in curtailing indirect spread of EHV-1 by way of contaminated fomites.

Ideally, staff handling horses in isolation would be assigned to that group only and to other nonanimal duties. Bedding contaminated with respiratory secretions should be sprayed with disinfectant and bagged for disposal.

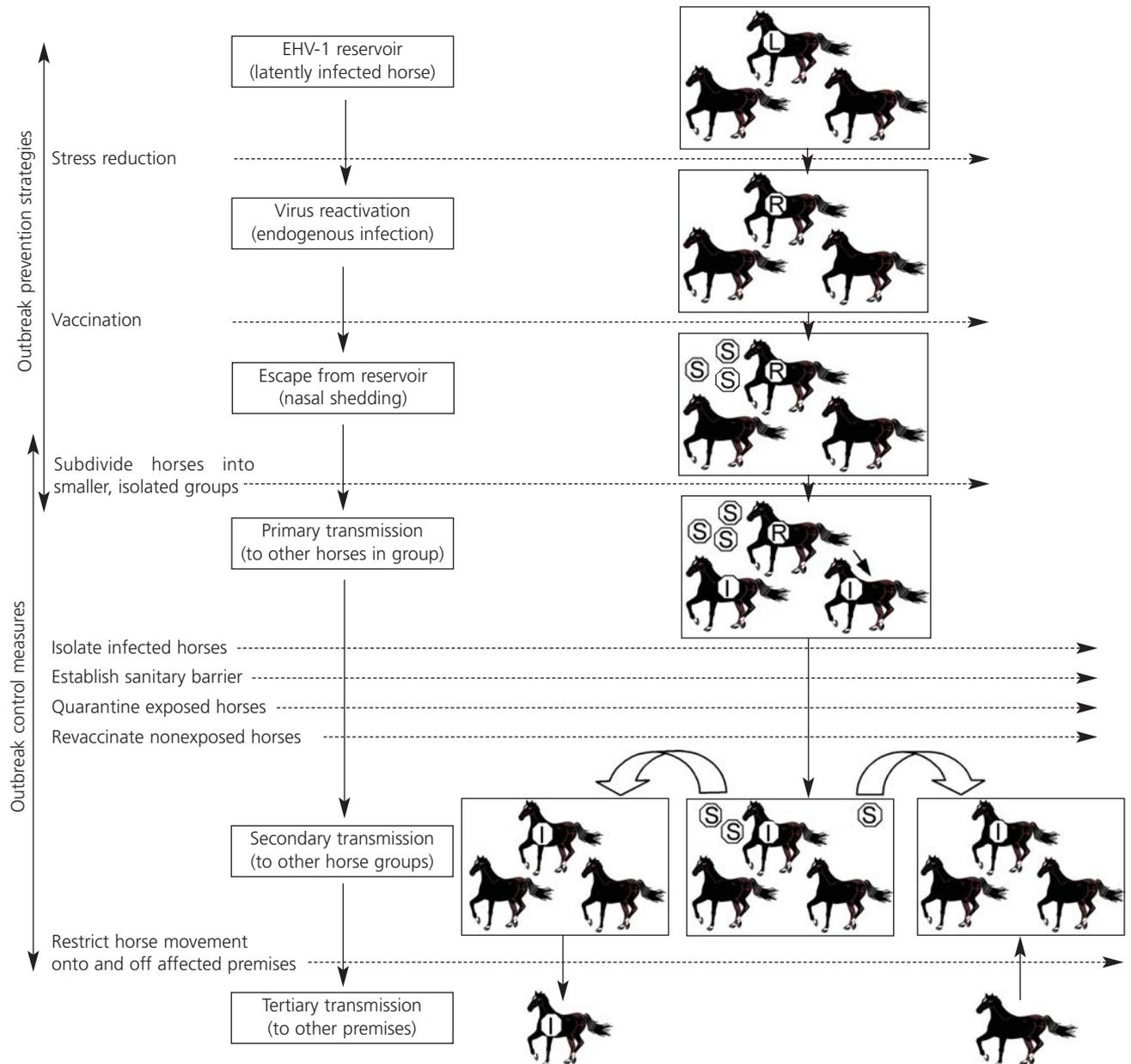


Fig 1: Diagrammatic illustration of the initiation and progression of a typical epidemic of EHV-1 infection (vertical arrows) and locations along the progression cycle at which specific control measures can be applied for interrupting transmission of the virus (horizontal arrows). L = latent virus; R = reactivated virus; S = shed virus; I = infecting virus.

Unnecessary traffic into the isolation area by farm employees, farriers or vehicles should be strictly controlled.

b) All horses in physical contact or sharing facilities with clinically affected animals (including those in adjacent paddocks, stalls, treatment areas, etc.) should be considered as having been exposed to the virus, remain confined to their respective stalls or paddocks and placed under a movement-restricted quarantine.

The relocation and intermingling of in-contact horses during an active outbreak of EHV-1 creates the greatest possible risk for transmitting infectious virus into additional

groups of horses. Daily observation of the quarantined horses for signs of EHV-1 infection (**pyrexia, nasal discharge, ataxia, abortion**) and immediate removal of suspected cases into an isolation area are advisable. If the initial number of in-contact horses is large (and if the facilities so permit), their early subdivision into smaller, physically isolated groups may reduce infection rates within the quarantined horse population.

The mating of in-contact mares placed in quarantine should cease until the epidemic has been brought under control. The breeding of mares aborting from EHV-1 should be delayed until the second oestrus cycle after abortion.

iii. Minimise the number of horses on the premises susceptible to disease

Restraints upon the transport of horses both onto and off EHV-1-affected premises will minimise the number of animals placed at risk of exposure to the circulating, epidemic strain of virus (**quarantine of premises; Fig 1**). If the horse operation is large and widely dispersed, the movement-restricted quarantine may judiciously be limited to only the affected portion of the premises, freeing the remainder of the farm for continuation of its daily activities (e.g. breeding shed activities). Orphaned foals of mares dying as a result of EHV-1 infection should not be fostered to other premises.

A full course of vaccination (i.e. 3 appropriately spaced doses) administered to nonexposed, unvaccinated mares, or a booster dose of vaccine given to nonexposed, previously vaccinated pregnant mares on the EHV-1-affected dwelling, to build an **immune cordon** around the focus of infection, may help to reduce the number of horses susceptible to overt clinical disease following viral exposure and also to reduce the magnitude and duration of nasal shedding of the virus. Because of the possibility of immunisation-induced exacerbation of the severity of EHV-1 neurological signs, **revaccination is not recommended for recent cohorts of horses affected with EHV-1 myeloencephalopathy**.

The success of the collective control procedures in halting the spread of EHV-1 from the primary focus of infection can be assessed by monitoring the epidemic curve for a falling-off in the number of new clinical cases.

Therapeutic management of individual cases

Specific details of the veterinary medical and nursing procedures that may be used for salvaging individual horses affected with clinical EHV-1 disease have been thoroughly described (Paradis 1996; Cutler and MacKay 1997; Donaldson and Sweeney 1997; Goehring and Sloet van Oldruitenborgh-Oosterbaan 2001; Olsen 2001) and will be only briefly summarised here. **The rationale for such treatment regimens is the use of symptomatic and supportive care for maintaining the nutritional needs and reducing distress or the likelihood of death or permanent impairment of the affected horse.**

Antipyretics are given to pyrexemic animals, **fluid and electrolyte replacement therapy** to horses with inappetence, and **antibiotics** given for controlling bacterial superinfection in newborn foals suffering from systemic EHV-1 infection acquired *in utero*. **Nonsteroidal anti-inflammatory** agents are indicated in the management of EHV-1-associated vasculitis, respiratory tract disease or other soft-tissue injuries. The use of antiviral chemotherapy with acyclic nucleoside analogues may have some merit in reducing the mortality rate during outbreaks of EHV-1 perinatal disease (Murray *et al.* 1998).

The administration of dimethylsulphoxide and a **corticosteroid** is a standard treatment for horses with EHV-1-associated paralysis. Horses with bladder wall or sphincter paralysis associated with EHV-1 myeloencephalopathy may

require frequent urinary catheterisation, postcatheter flushing of the bladder with povidoniodine solution, and antibiotic treatment for cystitis. Because mares aborting EHV-1 infected foals remain asymptomatic, treatment is rarely indicated. Horses exhibiting prolonged recumbency from EHV-1 myeloencephalopathy require intensive, round-the-clock nursing care (McConnico *et al.* 1991). Light tranquilisation, thick bedding with frequent repositioning of the patient, daily cleansing and topical care of decubital ulcers and urine scalding and the use of mechanical slings for limited periods for assisting support of recumbent or paretic animals are often indicated.

Euthanasia should be considered in laterally recumbent horses or sling-supported animals failing to show improvement after a few days, or in animals developing severe systemic complications from recumbency. The prognosis for full neurological recovery of EHV-1-infected horses so severely affected as to require prolonged sling-assistance is not generally favourable (van Maanen *et al.* 2001).

Postoutbreak management

Measures to be taken at the **end of an EHV-1 outbreak include** (a) lifting of the quarantine, (b) dispersal of affected horses, (c) final decontamination of the premises and (d) management steps taken to prevent recurrences of epidemic disease.

Release from quarantine and dispersement of recovered horses

An outbreak of EHV-1 disease may reasonably be considered as being over, allowing unrestricted release of horses from established isolation or quarantine restraints, as appropriate, when **thrice the usual shedding period for EHV-1 in mature horses (3 x 7 days = 21 days) has elapsed without the occurrence of any further cases of disease**. In abortion outbreaks, however, pregnant mares should remain on the affected premises until after foaling.

There are no available data to indicate that horses that have recovered from infection caused by epidemic or paralytic strains of EHV-1 pose any greater risk for infection of susceptible horse populations than a random assortment of animals.

Terminal disinfection of premises

Swift decontamination of the environmental surfaces of facilities in which an epidemic of EHV-1 has occurred can be achieved by thorough cleaning with detergent and water followed by rigorous chemical disinfection with **phenolic- or iodophor-type compounds** (Dwyer 1992).

Objects requiring cleaning and terminal disinfection include surfaces of stalls and aiseways; used bedding; tack; lead ropes; containers used for feeding and watering; boots and outer apparel of attendants; treatment instruments; grooming, stall-cleaning and mucking-out utensils; and the interior of horse conveyance vehicles.

Inactivation of EHV-1 in a contaminated environment will also occur naturally with the passage of time. Viability of the virus outside the body of the horse is transient enough that, after a period of 21 days without the presence of horses, the facilities may be considered safe for horse repopulation without risk of infection.

Follow-up control measures

Three important follow-up aspects of infectious disease epidemic management are:

- a **postepidemic assessment** of the efficacy of the control measures used;
- implementation on the affected dwelling of **preventative measures** aimed at avoiding the repetition of similar outbreaks of EHV-1 infection;
- the generation of a **published account** of the outbreak experience for the benefit of others.

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References

- Allen, G.P. (2000) Equine rhinopneumonitis. In: *OIE Manual of Standards for Diagnostic Tests and Vaccines*, 4th edn., Eds: M. Trusczynski, J.E. Pearson, S. Edwards and B. Schmitt, OIE Press, Paris. pp 565-575.
- Allen, G.P., Kydd, J.H., Slater, J.D. and Smith, K.C. (2002) Equid herpesvirus 1 (EHV-1) and equid herpesvirus 4 (EHV-4) infections. In: *Infectious Diseases of Livestock*, Eds: J.A.W Coetzer, G.R Thomson and R.C. Tustin, Oxford University Press, Cape Town. (In Press)
- Anon (1994) *The Code of Practice for the Control of Equine Herpesvirus-1 Disease in Australia*, Victorian Blood Horse Breeders Association, Flemington, Victoria, Australia.
- Anon (1997) *Guidelines for the Control of Equine Herpesvirus Abortion*, Australian Equine Veterinary Association, Artarmon, New South Wales, Australia.
- Anon (2000) *A Common Code of Practice for the Control of Contagious Equine Metritis and other Equine Reproductive Diseases*, Horserace Betting Levy Board (UK), London.
- Bryans, J.T. (1981) Application of management procedures and prophylactic immunization to the control of equine rhinopneumonitis. *Proc. Am. Ass. equine Practns.* **26**, 259-272.
- Bryans, J.T. and Allen, G.P. (1986) Control of abortigenic herpesviral infections. *Curr. Therap. Theriogenol.* **2**, 711-714.
- Bryans, J.T. and Allen, G.P. (1989) Herpesviral diseases of the horse. In: *Herpesvirus Diseases of Cattle, Horses and Pigs*, Ed: G. Wittmann, Kluwer, Boston. pp 176-229.
- Crabb, B.S., MacPherson, C.M., Reubel, G.H., Browning, G.F., Studdert, M.J. and Drummer, H.E. (1995) A type-specific serological test to distinguish antibodies to equine herpesviruses 4 and 1. *Arch. Virol.* **140**, 245-258.
- Cutler, T.J. and MacKay, R.J. (1997) Equine herpesvirus-1 myeloencephalitis. In: *Current Therapy in Equine Medicine*, 4th edn., Ed: N.E. Robinson. W.B. Saunders Co., Philadelphia. pp 333-335.
- Dixon, R.J., Hartley, W.J., Hutchins, R.D., Lephard, E.E., Feilen, C., Jones, R.F., Love, D.N., Sabine, M. and Wells, A.L. (1978) Perinatal foal mortality associated with a herpesvirus. *Aust. vet. J.* **54**, 103-105.
- Donaldson, M.T. and Sweeney, C.R. (1997) Equine herpes myeloencephalopathy. *Comp. cont. Educ. pract. Vet.* **19**, 864-882.
- Dwyer, R.M. (1992) Foal management, disinfection and hygiene. In: *The Health of Horses*, Eds: D.G. Powell and S.G. Jackson, Longman Group, Essex. pp 234-261.
- Edington, N., Welch, H.M. and Griffiths, L. (1994) The prevalence of latent equid herpesviruses in the tissues of 40 abattoir horses. *Equine vet. J.* **26**, 140-142.
- Greenwood, R.E. and Simpson, A.R. (1980) Clinical report of a paralytic syndrome affecting stallions, mares and foals on a Thoroughbred studfarm. *Equine vet. J.* **12**, 113-117.
- Goehring, L.S. and Sloet van Oldruitenborgh-Oosterbaan, M.M. (2001) The mystery of equine herpes myeloencephalopathy. *Equine vet. Educ. IAE* **3**, 53-59.
- Hartley, W.J. and Dixon, R.J. (1979) An outbreak of foal perinatal mortality due to equine herpesvirus type 1. *Equine vet. J.* **11**, 215-218.
- Lunn, D.P. and Townsend, H.G.G. (2000) Equine vaccination. *Vet. Clin. N. Am.: Equine Pract.* **16**, 199-226.
- McCartan, C.G., Russell, M.M., Wood, J.L.N. and Mumford, J.A. (1995) Clinical, serological and virological characteristics of an outbreak of paresis and neonatal foal disease due to equine herpesvirus-1 on a stud farm. *Vet. Rec.* **136**, 7-12.
- McConnico, R.S., Clem, M.F. and DeBowes, R.M. (1991) Supportive medical care of recumbent horses. *Comp. cont. Educ. pract. Vet.* **13**, 1287-1294.
- Mumford, J.A., Rossdale, P.D., Jessett, D.M., Gann, S.J., Ousey, J. and Cook, R.F. (1987) Serological and virological investigations of an equid herpesvirus 1 (EHV-1) abortion storm on a stud farm in 1985. *J. Reprod. Fert., Suppl.* **35**, 509-518.
- Murray, M.J., del Piero, F., Jeffrey, S.C., Davis, M.S., Furr, M.O., Dubovi, E.J. and Mayo, J.A. (1998) Neonatal equine herpesvirus type 1 infection on a thoroughbred breeding farm. *J. vet. Intern. Med.* **12**, 36-41.
- Olsen, T.F. (2001) Equine herpesvirus myeloencephalopathy in a 14-year-old quarter horse stallion. *Can. vet. J.* **42**, 217-220.
- Paradis, M.R. (1996) Equine herpesvirus (rhinopneumonitis). In: *Large Animal Internal Medicine*, 2nd edn., Ed: B.A. Smith, Mosby, St. Louis, Missouri. pp 587-588.
- Powell, D.G. (1992) Prevention, treatment of equine herpesvirus. *Thoroughbred Times* January 31st, 9.
- Rebhun, W.C., Jenkins, D.H., Riis, R.C., Dill, S.G., Dubovi, E.J. and Torres, A. (1988) An epizootic of blindness and encephalitis associated with a herpesvirus indistinguishable from equine herpesvirus 1 in a herd of alpacas and llamas. *J. Am. vet. Ass.* **192**, 953-956.
- Smith, K.C., Mumford, J.A., Hannant, D. and Whitwell, K.E. (1999) A comparison between the pathogenicity of EHV-1 isolates of high and low abortigenic potential in the natural host and in the mouse mode. In: *Equine Infectious Diseases VIII*, Eds: U. Wernery, J.F. Wade, J.A. Mumford and O.-R. Kaaden, R&W Publications, Newmarket. pp 581-582.
- van Maanen, C., Sloet van Oldruitenborgh-Oosterbaan, M.M., Damen, E.A. and Derksen, A.G.P. (2001) Neurological disease associated with EHV-1 infection in a riding school: clinical and virological characteristics. *Equine vet. J.* **33**, 191-196.
- Vickers, M.L. and Powell, D.G. (2001) Equine herpes virus abortions. *Equine Dis. Quart.* **10**, 3-4.
- Wilson, W.D. (1996) Equine vaccination and infectious disease control. Equine herpesviruses (rhinopneumonitis). In: *Large Animal Internal Medicine*, 2nd edn., Ed: B.A. Smith, Mosby, St. Louis, Missouri. pp 1638-1639.
- Wilson, W.D. (1997) Equine herpesvirus 1 myeloencephalopathy. *Vet. Clin. N. Am.: Equine Pract.* **13**, 53-72.