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Equine protozoal myeloencephalitis

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Equine Protozoal Myeloencephalitis (EPM) is currently the most frequently diagnosed neurologic disorder in horses in the US. EPM is caused by the protozoan organism Sarcocystis neurona. Typically, Sarcocystidae have an obligate prey-predator life cycle, where asexual stages can be found in the muscle of prey animals, and sexual stages of their reproduction within the intestine of their specific predator animals. S. neurona uses birds as intermediate and the opossum (idelphis virginiana), a marsupialian animal native to the Americas, as the definitive hosts. The horse is an aberrant host, and can become infected after ingestion of infectious oocysts. The protozoan stages enter the CNS, however, they are unable to mature within the CNS, and therefore are they neither infectious for the opossum, nor for other horses.

With the (American) opossum as the definitive host of S. neurona, EPM is an American problem, however, EPM can occur in horses exported from the US. The clinical signs vary, depending on the location of the parasite within the CNS. The damage is caused by the presence and further multiplication of the protozoan organism, and the associated inflammation. The infection can also be multifocal. An asymmetric ataxia of the limbs, with or without lower motor neuron involvement is the most frequently found complaint at presentation. Seroprevalence among horses varies depending on climate, and the presence of the definitive host, the opossum; seroprevalence can be as high as 60%. However, seroprevalence does not correlate with clinical disease. So far, a vertical infection from the mother to the developing foal has not been demonstrated. The clinical disease seems more frequent in Standardbreds and Thoroughbreds, and least likely in ponies.

EPM is diagnosed ante mortem by the presence of clinical neurologic signs, a positive immunoblot assay showing intrathecally produced antibody against S. neurona, and improvement with therapy. The most established treatment is a combination of sulfadiazine and pyrimethamine, which are administered for an average period of six months. Treatment goals are to treat until the immunoassay on cerebrospinal fluid is cleared from intrathecally produced antibodies. Currently diclazuril and toltrazuril are under clinical evaluation in several field trials.

Current problems with the disease complex "EPM" are the treatment, and its efficacy, as well as the lack of criteria when to discontinue medication. In more than 90% of the horses treated for EPM for at least six months the immunoblot assay on cerebrospinal fluid remains positive. Explanations for this phenomenon may be inefficacy of the therapeutics in use or resistance development of the parasite; chronic reinfection, or persistent infection, as described for Toxoplasma spp. infections in other species. Intensive further research in epidemiology and the mechanism of host-parasite interaction is necessary to clarify the complexity of this disease.

Heart rate and haematological responses in Quarter Horse during a reining competition

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Reining is a classical Western discipline with manoeuvres requiring fast and powerful muscle contractions and motoric skills. It is considered an «anaerobic» discipline, but there are no studies showing the degree of anaerobiosis induced by an American Quarter Horse Association reining pattern. Measurement of heart rate and plasma lactate are established methods in sports medicine to estimate the workload of a sports discipline. The purpose of this study was to estimate the work intensity of a reining pattern and haematological responses of trained Quarter Horses during a reining competition. Twelve Quarter Horses between 4 and 8 years of age were equipped with a heart rate meter (Hippocard®, Isler Bioengineering AG, Zurich) at a National Reining Horse Association approved horse show. Heart rates were continuously recorded from about the last 30 minutes of the warm-up until 2 minutes after leaving the arena. The performance of each participant was video recorded and heart rates subsequently assigned to the different manoeuvres. Blood samples were taken one day before the class for resting levels and 1 minute after completion of the pattern. Plasma samples (S-Monovette®) for lactate measurement were immediately centrifuged and shock frozen in liquid nitrogen until processing. Plasma lactate was determined by a photometric method (Boehringer, Mannheim). Data were analysed by paired t-tests or Wilcoxon signed-rank tests. Average time needed to com-