Cook, W.R: EIPH or AIPE? *Equine Athlete* March/April 1997, p23

EIPH or A.I.P.E?

Brent Kelley D.V.M., provides a good answer to the interesting question from Frederick Spohn M.D. about the effectiveness of Lasix as a treatment for exercise-induced pulmonary hemorrhage in the horse (*Thoroughbred Times; October 26, 1996, p 60).* There was, however, one phrase in his answer that calls for a comment. Dr Kelley correctly points out that Lasix is a well-recognized treatment for pulmonary edema in man but adds that "pulmonary edema...has nothing to do with EIPH" in the horse. With all due respect to Dr Kelley, whose articles I read with pleasure and instruction, I have to say that I disagree. My own research leads me to the conclusion that horses which "bleed" from the lungs at exercise are in fact suffering from exactly this problem, ie., pulmonary edema.

If a *post-mortem* examination is carried out *immediately* on a horse that has died from EIPH, there is abundant evidence of acute pulmonary edema (Refs x 4). Unfortunately, if the *post-mortem* examination is delayed several hours, which is generally the case, this evidence will no longer be present and this explains why the true nature of EIPH has not been recognized.

It is my belief that the condition we call *exercise-induced pulmonary hemorrhage (EIPH)* is a misnomer. A more accurate name, I suggest, is *asphyxia-induced pulmonary edema (AIPE)*. The discharge that we have mistakenly called a "hemorrhage" is not blood but heavily blood-stained edema fluid. The post-mortem lesions that we have called "exercise-induced" are not specific to exercise alone, for they can also be seen in a horse that has not been recently exercised. The factor that is common to both occurrences is not exercise but asphyxia. Clinical signs and *post-mortem* lesions characteristic of so-called EIPH occur in horses that have experienced an obstruction to the upper airway (ie., an obstruction at any point between the nostrils and the windpipe at the level of the first rib), regardless of whether this occurs in the standing or the galloping horse.

Dr Kelley correctly points out that "as many as 95% of horses in training show evidence of EIPH on endoscopic evidence". Readers may well protest that surely it cannot be true that 95% of racehorses suffer from asphyxia during a race. This may *seem* improbable but, sadly, it is not so. Those that doubt this should remember the adage "today's nonsense is tomorrow's common sense". Causes of airway obstruction in racehorses are legion and quite common enough to explain the prevalence of EIPH/AIPE. First, any bit pressure that results in head flexion of the slightest degree results in airway obstruction at the level of the throat (nasopharynx). Secondly, there are many defects of conformation that are sufficiently common to represent serious sources of upper airway obstruction. One can list narrow jaws (and therefore narrow throats and larynges) and the much-commoner-than-realized deformities of the windpipe as two examples but there are others. Finally, obstruction of the airway is a primary feature of any upper airway disease.

One disease that occurs, to varying degrees, in over 95% of horses is recurrent laryngeal neuropathy (RLN). In the live animal this can be shown to be present by an objective test of nerve function, using a relatively new electro-diagnostic method known as

electrolaryngeography (ELG). In the dead animal it can be shown to be present by an examination of the laryngeal muscles and nerves.

The reason that RLN is not more often recognised at necropsy examinations is that the requisite examinations are not being carried out. If an examination of the larynx was made part of the regular *post-mortem* examination protocol, the evidence would be found. Examination of the larynx is time-consuming but it is by no means impossible. The examination comes well within Peter Medawar's definition of science as "the art of the soluble". A project that may well be insoluble is to devise a method for testing the relationship between either AIPE or furosemide and racing performance. There are so many variables involved that the influence of any one factor is probably impossible to show. How to "measure" AIPE is only one problem but, paradoxically, the most difficult parameter to measure may be performance itself, especially so in a sport where the difference between success and failure can be as small as one fifth of a second. No wonder that no statistically significant correlations have been reported.

But absence of evidence is not evidence of absence. Because furosemide "cannot be proven to improve performance" we should not assume, therefore, that it has no effect. Similarly, though there is surely proof enough that AIPE affects performance when a horse dies on the racetrack, it is probably impossible to show its influence when the degree of AIPE is less than fatal. We should certainly not comfort ourselves with the notion that AIPE is "normal". It is true to say that, within the racing Thoroughbred population AIPE is statistically common and might therefore be considered statistically to be the norm, or average, but is not true to say that it is biologically normal for a horse to develop pulmonary edema at exercise. The presence of blood-stained edema fluid in the airway is not physiological in any mammal. The air sacs of the lung should be filled with air not fluid. Although I cannot show any "scientific evidence" of the sort that perhaps Dr Spohn is seeking (eg., from a research project designed to test the null hypothesis that AIPE has no effect on performance), I believe it is reasonable to argue that any athlete that develops AIPE is likely to be a less successful performer than one with a healthy pair of lungs. On these grounds, I believe we should assume that AIPE does affect performance.

AIPE has been described as "unavoidable consequence of competitive training and racing". Though I would agree that it is difficult to avoid, I do not think that we should make no effort to avoid it. Once it is recognized that the cause of some racetrack accidents and some deaths is asphyxia, then an important step will have been taken towards a limitation if not an outright elimination of the problem.