ANHIDROSIS

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The last several weeks at Brazos Valley Equine Hospital – Navasota have seen an increase in horses presenting for evaluation of clinical signs consistent with or with a history of non-sweating. Despite first being recognized by the British in the early 20s, interest in the sweat response of horses did not appear in the literature until the early 50s, and the term anhidrosis first appears in the early 80s. Despite over 50 years of study, the cause and a consistent treatment for this condition continues to eludes clinicians.

Anhidrosis is defined as the inability of horses to sweat in response to appropriate stimulation. Hypohydrosis is a reduced sweat production in response to appropriate stimulation. Frequently, the term anhidrosis is used to include all degrees of hypohydrosis. Anhidrosis is a common problem in the tropics, sub-tropics and most of the southern United States. It is thought to develop from poor acclimation of horses to hot and humid conditions, however the condition will spontaneously develop in horses that are native to the area, as often as imported horses. A handful of epidemiologic studies done in Florida showed a prevalence of ~6%, although others have suggested incidence as high as 20%. Anhidrosis is seen more commonly in older horses than in young horses and is less common in pregnant mares. In addition to age, pregnancy, and degree of exercise, the prevalence likely depends on the severity of the summer weather. There is

no proof of a genetic or dietary influence, although both are frequently debated as modifying factors.

Sweating is necessary to transfer heat away from the body. Equine sweat glands are apocrine and have a very extensive capillary network. Sweating in the horse is controlled by both endocrine and neural mechanisms. The equine sweat glands are under beta 2 adrenergic control, through both direct stimulation and regulation of the capillary beds.

There is very little understood about the pathogenesis of anhidrosis, but the cause is failure of the gland to produce sweat secondary to a stimulus. It is thought that desensitization and down regulation of the beta 2 receptors occurs from excessive stimulation. This could be from internal factors (e.g. endocrine/neural mechanisms) or external factors (e.g. chronic clenbuterol use). While a complete pathogenesis is not known there is little evidence to implicate the pituitary, loss of cutaneous innervation, adrenal dysfunction, hypothyroidism, electrolyte abnormalities, inflammation, or immune-mediated processes as a cause of anhidrosis.

Clinical signs in horses suffering from abnormal sweating include: poor performance, prolonged recovery times, tachypnea, hyperthermia, alopecia, dry hair coat, lethargy, playing in water buckets, and lack of normal sweat production. It is important to note that some horses may continue to sweat under the mane and under the saddle. Tachypnea is the most impressive clinical sign and many horses often present for evaluation of respiratory disease. Tachypnea resolves quickly with a water bath and placement under a fan. Recognition of the symptoms early seems to have the best chance at managing the condition successfully. Recognition of the condition late, risks a heat related illness and significant stress to the horse.

The diagnosis of anhidrosis is based primarily on clinical signs and response to an intra-dermal sweat test with epinephrine or terbutaline. Epinephrine is frequently used, but it is both an alpha and beta-2 adrenergic agonist. Use of terbutaline is a more specific beta-2 agonist and should be used when available. One tenth of a ml of varying dilutions of either stimulant are injected intra-dermally over the side of the neck using a 25 gauge needle. Saline is used as a control. We prefers to clip the hair over the injection sites to help identify a partial response. Epinephrine is available in most veterinary practices in a 1:1000 dilution. Using saline, dilutions of 1:10,000, 1:100,000 and 1:1,000,000 can be made.

Each injection is made 5 to 10 cm apart. Within 10 minutes some sweating can be observed, but the response should be judged at 30 minutes. A quantitative test has also been developed using absorbent pads to weigh the amount of sweat produced in a standard length of time.

Treatment of anhidrosis remains a challenge. There are a multitude of supplements that claim to “cure” anhidrosis along with multiple anecdotal therapies. Moving to a cooler climate is the only known cure. In most cases, horses are managed to try to prevent heat related injuries. Working horses in the coolest part of the day, keeping them under fans and misters, or moving to a cooler climate will help avoid the risk of inducing a heat related injury. The author has listed 23 of the most common treatments proposed for anhidrosis in publications, proceedings, and by clients. Of these, use of beta-2 agonists (clenbuterol) is unlikely to be successful because the condition is caused by overstimulation and down regulation of the sweat gland receptors. There is evidence in other species that thyroid hormones may improve beta 2 receptor sensitivity. Similarly glucocorticoids may enhance the receptor response to beta 2 agonists. Use of electrolyte and trace mineral supplement has not been critically tested, but makes logical sense

and is not likely to cause harm. Methyldopa is a vasodilator that has been tried based on the theory that it reduces central sympathetic activity and overstimulation of the sweat glands. While it has been successful anecdotally, it currently lacks any reported efficacy in clinical trials.

Ultimately the prognosis is dependent on how severely affected the horse is and how the anhidrosis is affecting the horse's ability to perform. Once the weather cools, most horses will return to normal performance.

LIST OF TREATMENTS CLIENTS HAVE TRIED COLLECTED BY THE AUTHOR – NO CLAIM IS MADE FOR ANY TREATMENTS ON THE LIST.

1. Electrolyte supplements
2. Trace mineral supplements
3. Dark beer
4. Acupuncture
5. Coffee grounds/lite salt/Gatorade
6. Methyldopa
7. Clenbuterol
8. Air conditioner
9. Transdemeral electrolyte patches
10. Corticosteroids (both continuous and pulse therapy)
11. Restricting beet pulp in the diet
12. Iodine supplementation
13. Vitamin E supplementation
14. ACTH injections
15. Alpha 2 agonists
16. Prostaglandin injections
17. Progesterone supplementation
18. Antihistamines
19. Caffeine
20. Restricting hay in the diet
21. Restricting protein in the diet
22. Levothyroxine
23. Mast cell stabilizers