CALIFORNIA HORSE RACING BOARD
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MEDICATION AND TRACK SAFETY COMMITTEE MEETING

of the **California Horse Racing Board** will be held on **Wednesday, May 19, 2010,** commencing at **2:00 p.m.,** in the **Directors Room at Golden Gate Fields, 1100 Eastshore Hwy, Albany,** California. Non-committee Board members attending the committee meeting may not participate in the public discussion, official committee vote or committee closed session.

The agenda for the meeting will consist of the following matters:

1. Report and discussion regarding the **Racing Medication and Testing Consortium (RMTC) April 12, 2010 meeting** including laboratory accreditation, withdrawal time research and proposed changes to non-steroidal anti-inflammatory drug substances (NSAID) levels.

2. Update and discussion regarding the **CHRB track safety standards program.**

3. Report and discussion regarding the **current injury monitoring programs, CHRB/University of California at Davis – California Animal Health and Food Safety (CAHFS) necropsy program and the Equine Injury Database.**

4. Discussion and update of the **CHRB's drug testing programs.**

5. Discussion and action regarding the **proposed amendment to CHRB Rule 1844, Authorized Medication,** to modify the levels permitted for procaine subsequent to procaine penicillin administration.

6. Discussion regarding the **transparency and transferability of veterinary medical records.**

7. Discussion and action regarding the **proposed amendment to CHRB Rule 1846.5, Postmortem Examination,** to require training and veterinary medical records on all horses dying within a CHRB inclosure.

Additional information regarding this meeting may be obtained from Jacqueline Wagner at the CHRB Administrative Office, 1010 Hurley Way, Suite 300, Sacramento, CA 95825; telephone (916) 263-6000; fax (916) 263-6042. A copy of this notice can be located on the CHRB website at www.chrb.ca.gov. *Information for requesting disability related accommodation for persons with a disability who require aids or services in order to participate in this public meeting, should contact Jacqueline Wagner.

SAFTEY COMMITTEE
Commissioner Bo Derek, Chairman
Commissioner John C. Harris, Member
Kirk E. Breed, Executive Director

Date of Notice: May 7, 2010

STAFF ANALYSIS
REPORT AND DISCUSSION REGARDING
THE RACING MEDICATION AND TESTING CONSORTIUM (RMTC) APRIL 12, 2010 MEETING INCLUDING LABORATORY ACCREDITATION, WITHDRAWAL TIME RESEARCH AND PROPOSED CHANGES TO NON-STEROIDAL ANTI-INFLAMMATORY DRUG SUBSTANCES (NSAID) LEVELS

Medication and Track Safety Committee Meeting
May 19, 2010

BACKGROUND

The attached information explains the basis for the RMTC's action relative to phenylbutazone. The RMTC Board voted to approve the recommendation of the RMTC Scientific Advisory Committee to reduce the Model Rule NSAID level to 2ug/ml. There were a few other minor changes to clean up language. The only other substantive change was to lower the secondary level of PBZ to 0.5ug/ml.

The key evidence was the levels of phenylbuatzone and flunixin levels found at pre-race exam time in KY & CA combined with Soma's conclusions from his PBZ review. The exam time NSAID levels were obtained by examining veterinarians in CA & KY at the time the examining veterinarians performed pre-race examinations. There were no changes recommended for flunixin. The RMTC level is 20ng/ml; the CHRB level is 50ng/ml. To be in compliance with ARCI & RMTC guidelines the CHRB levels would need to be reduced. Back to 20ng/ml as it was previously.

The way a change in the ARCI works is the full ARCI will address this recommendation at their July meeting if it is approved by the ARCI's Drug Testing, Standards and Practices committee & their Model Rules committees. After that individual states will adopt the recommendations or not. There will be some impetus for many states to move forward as NTRA accreditation is contingent on adopting RMTC & ARCI model rules.

The RMTC's Scientific Advisory Committee is comprised of Tom Brokken, Scott Stanley, George Maylin, Rick Sams, Larry Soma, Don Catlin, Tom Tobin, Ted Hill, Al Kind, Bobby Lewis; I chair the committee.

Not available at the time of the RMTC Scientific Advisory Committee's review was fatality rate information from both Iowa and Virginia. Both states have documented increases in fatality rates subsequent to increasing permitted phenylbutazone levels from 2ug/ml to 5ug/ml. Virginia was so convinced their increase in fatality rates was associated with the increase in permitted phenylbutazone levels from 2ug/ml to 5ug/ml they reduced the phenylbutazone level back to 2ug/ml and immediately saw a drop in racing fatality rates.

RECOMMENDATION

This item is presented for committee discussion.

TO: RMTC Board of Directors

FROM: Dr. Rick Arthur, Scientific Advisory Committee chairman

DATE: 4/12/10

SUBJECT: Background NSAID Recommendation

Last spring the ARCI Regulatory Veterinarian's committee submitted a letter to the RMTC and ARCI outlining their concern that current medication regulations were compromising pre-race examinations (attached Dr. David letter). Subsequently the RMTC Sci Adv committee asked Dr. Larry Soma to do a review of the scientific literature on phenylbutazone (PBZ). All members of the Sci Adv committee were encouraged to provide any comments to Dr. Soma. Attached is the final version after committee input (attached Soma review). Note the reviews conclusion:

> This review presented an historical prospective and examined the information presented in 4 different models used to determine the effects of NSAIDs, especially PBZ. They included naturally occurring lameness, reversible induced lameness, and indirect plasma and tissue models studying the suppression of the release of arachidonic-derived mediators of inflammation. The majority of studies suggest an effect of PBZ at 24 hours at a dose of 4.4 mg/kg. This reflects and substantiates the opinion of many clinical veterinarians, many of whom will not examine a horse for a pre-purchase lameness examination unless the horse is shown to be free of NSAIDs and glucocorticoids. This remains the opinion of many Commission Veterinarians in that they wish to examine a horse pre-race without the possibility of a NSAID or corticosteroid interfering with the examination and masking a possible musclo-skeletal condition. Based on scientific reports and the impression of clinical veterinarians, residual effects of PBZ remain at 24 hours. The impact of this sustained effect on the health and welfare of the horse remains problematic.

To obtain relevant data the blood samples were obtained from horses in CA & KY at exam time by CA & KY examining veterinarians (attached CA & KY Exam time samples).

The Sci Adv committee discussed all the information and the best options for PBZ regulation for the racing industry. IFHA rules, 48hr rules and other alternatives were discussed. The consensus was the best option was to revisit the 2ug/ml rule that was in place in many jurisdictions before the 5ug/ml rule was adopted. While this does not solve the problem identified by the ARCI Regulatory Veterinarian relative to pre-race examination, the conclusion was the 2ug/ml rule is a move in the right direction. There is a wealth of experience administering the 2ug/ml rule and until corticosteroids can be properly regulated, any more stringent PBZ rule will simply encourage more corticosteroid use. The general consensus was the latter would be more detrimental to horse health and welfare.

RMTC Scientific Advisory Committee
Phenylbutazone Review
Lawrence R Soma, VMD
University of Pennsylvania
School of Veterinary Medicine
New Bolton Center Campus

Initial opinions on the effects of phenylbutazone (PBZ) and historical prospective.

Phenylbutazone was introduced into veterinary medical practice in the 1950s (Tobin et al., 1986) and still remains one of the more commonly used non-steroidal anti-inflammatory drugs (NSAID) in the horse. In the late 1970s, the use of PBZ came under scrutiny which resulted in the publication of the book "The Misuse of Drugs in Horse Racing: a Survey of Authoritative Information on Medication of Race Horses" by the Illinois Hooved Humane Society. This publication stirred controversy on the use of PBZ especially on race day. In 1977 the National Association of State Racing Commissioners Veterinary-Chemist Advisory Committee concluded that "phenylbutazone is a safe, effective nonsteroidal anti-inflammatory drug (NSAID) with antipyretic and analgesic activity. In recommended doses, there is no evidence that it changes a horse's innate ability to race, except to make a horse perform more nearly normal with pain due to inflammation of part of the musculoskeletal system" (Gabel et al., 1977).

It was also the opinion of many veterinarians, at that time, that PBZ would allow a horse to compete with mild chronic arthritic changes, but did not possess sufficient analgesic activity to allow a horse with a serious injury to compete. The NSAIDs can be used to restore normal performance in a horse affected by some minor injury to joints, tendons, or muscle by providing relief of inflammatory pain. Some consider the use of NSAIDs justified in jumpers, and exceptions are made in racing by some jurisdictions. Many veterinarians agreed that the use of anti-inflammatory drugs could mask unsoundness in horses being examined in a pre-purchase examination for soundness (Sanford, 1983).

Results from performance studies suggested that PBZ had no clear effect on the performance of normal, healthy horses (Sanford, 1974). This impression was confirmed by exercise studies performed in the equine. Plasma concentration of prostaglandins are increased in man (Demers et al., 1981) and equine during exercise (Birks et al., 1991; Mitten et al., 1995). These exercise-induced increases in cyclooxygenase activity was inhibited by the administration of PBZ, but PBZ did not produce detectable changes in systemic hemodynamic or acid-base variables in either standing or running horses (Hinchcliff et al., 1994). In a exercising horses the effect of inhibition of cyclooxygenase activity on the hemodynamic response was examined. Administration of PBZ abolished the exertion-induced increases in plasma 6-ketoprostaglandin F_1 alpha and thromboxane B_2. Phenylbutazone treatment resulted in significantly higher heart rates and right atrial pressures than control.

There was no effect of PBZ on carotid or pulmonary arterial pressures, oxygen consumption, carbon dioxide production, blood lactate concentrations, or plasma volume during exertion. These results suggest that cyclooxygenase products likely mediate or modulate some of the systemic hemodynamic responses to exertion in horses (Mitten et al., 1996), but there is no evidence that the administration of PBZ and/or the suppression of cyclooxygenase products alters performance.

Based on these opinions and observations, investigators conducted a number of studies to determine the plasma PBZ concentrations 24 hours following various dosing schedules, formulations, and dosages (Soma et al., 1983; Chay et al., 1984; Houston et al., 1985; Soma et al., 1985). Following completion of these studies the recommended dosing schedule was as follows: oral administration of 4.4 mg/kg (2 g) for 3 - 4 days followed by a single IV dose of 4.4mg/kg 24 h prior to racing. If these dosing recommendations are followed, plasma PBZ concentrations on race day should not exceed 5 μg/ml. However, these studies did not attempt to determine the pharmacological effect of PBZ at 5 μg/mL or the contribution by its pharmacologically-active metabolite, oxyphenbutazone (OPB).

The initial regulatory race day plasma concentrations suggested to all Commissions was 2 μg/mL. This conservative concentration was based on the opinion of many analysts that the higher concentrations of PBZ were associated with masking or interfering with drug testing in urine (Gabel et al., 1977). This was subsequently reviewed and it was concluded that this concentration could be increased to 5 μg/mL which is the regulatory limit now accepted by most jurisdictions in the United States (see footnote in reference section).

Effects of Phenylbutazone on nociception (pain perception).

Pain is difficult to assess in the horse as it is influenced by many factors. Equally difficult to assess is the alteration of pain by analgesic drugs. The latency to onset of movement of a limb in response to a noxious thermal stimulus has been used as a nociceptive end-point for analgesic studies in many species (Kamerling et al., 1985). Thermal-evoked skin-twitch reflex and thermal evoked hoof withdrawal reflex have been used to compare analgesic activity of procaine, mepivicaine and phenylbutazone. Compared to procaine and mepivicaine, phenylbutazone failed to alter pain thresholds over a 36 hours post-administration period (Kimmerling et al., 1985). This type of stimulation produces an acute pain response and can be used to objectively compare the duration of anesthetic agents regionally administered and other drugs used to reduce the perception of pain. In the horse as in other species, PBZ is not an effective analgesic drug when used to block thermal pain and specific nociceptive pain stimuli.

Central nervous system effects and crossing the 'blood brain barrier'.

Phenylbutazone has no known spinal or central nervous system effects that are involved in the suppression of pain. The effects are primarily thought to be peripheral in action with no central nervous system action or any noticeable sedation. To exert a central effect, NSAIDs have to cross the blood brain barrier

(BBB) and numerous studies have shown that they do cross the BBB (Bannwarth et al., 1989). The presence of NSAIDs in the brain may explain the antipyretic properties and some side effects of the NSAIDs. Concentrations of oxyphenbutazone (OPB) in spinal fluid are similar to corresponding concentrations of unbound free OPB in plasma, which is approximately 5% of the total concentration of OPB in plasma (Gaucher et al., 1983a). Similarly, cerebral spinal fluid concentrations of ketoprofen reflect the unbound plasma ketoprofen concentrations and were in equilibrium with the plasma concentration from 2 to 13 hours after administration (Netter et al., 1985). Ibuprofen, flurbiprofen, and indomethacin rapidly cross the BBB. Plasma protein binding limits the driving force for uptake of NSAIDs into the brain by reducing the free fraction of NSAIDs in plasma (Parepally et al., 2006). The observation that long-term treatment of patients with ibuprofen results in a reduced risk and delayed onset of Alzheimer's disease suggests that it crosses the BBB, has a central effect, and reduces inflammation in the Alzheimer's diseased brain (Dokmeci and Dokmeci, 2004). Attempts to correlate the CFS concentrations of indomethacin with its regional inflammatory suppression and analgesic activity have not been successful (Bannwarth et al., 1989). The statement that the NSAID do not have central effects and all actions are peripheral in nature is certainly not substantiated by a number of studies.

Synovial fluid concentrations of NSAIDs

There is no barrier to the diffusion of NSAIDs into or out of the joint cavity and the efficacy of lNSAIDs in rheumatic diseases may depend on their concentrations within the joint. Piroxicam concentrations in plasma and synovial fluid after a single oral dose of 20 mg, in humans were 2.51 and 1.31 μg/ml, respectively. The elimination from synovial fluid was longer than from plasma (Bannwart et al., 2001). Concentration of OPB in synovial fluid were 57.1% of the corresponding plasma concentration. The OPB concentration was higher in patients with severe inflammation than in those with no or little inflammation (Gaucher et al., 1983a). Concentrations of diclofenac and the diffusion of aspirin and its metabolites through the synovial membrane have also been reported (Gaucher et al., 1983b; Bannwarth et al., 1985). In horses with no joint disease, equilibrium between synovial fluid and plasma concentrations was attained in 8 hours following the IV administration of naproxen; this was followed by a parallel decline in plasma and synovial fluid concentrations for up to 36 hours (Soma et al., 1995). In the horse, ketoprofen was no longer detectable in synovial fluid after 5 h whereas synovial fluid carprofen concentrations did not peak until 12 h and were still detectable at 48 h (Armstrong et al., 1999).

Many authors have suggested that the plasma concentrations of NSAIDs do not correlate well with assessments of therapeutic response. This may reflect weaknesses in experimental design, capability of determining the changes in pain levels and inflammation, and in clinical studies the variability in the diseases being studied. It may be that concentrations in plasma bear only a distant relationship to those in the inflamed tissues where NSAID presumably act (Famaey, 1985; Grennan et al., 1985; Simkin, 1988). Compared to the

CNS, NSAIDs readily penetrate into the joint and the concentration is not limited to the unbound fraction. The concentrations reported have been lower than in plasma. The peak concentrations and elimination from the joint are related to the pharmacokinetics and characteristics of the drug, dose and route of administration.

Post-operative pain and phenylbutazone

Post-operative pain can be considered primarily nociceptive pain produced by trauma to tissues by direct intervention and disruption of these tissues. Inflammation is a part of the pain response due to surgical intervention and the use of NSAIDs has been promoted for this purpose post-operatively. Minimal differences were noted between PBZ and placebo administrations in a group of horses undergoing arthroscopic surgery (Raekallio et al., 1997).

In a similar post-operative study, flunixin, phenylbutazone or carprofen were administered intra-operatively just prior to the end of anesthesia. The time following surgery when additional analgesic drugs were required post-operatively were; 8.4 hours, 11.7 hours and 12.8 hours for the PBZ, carprofen, and flunixin, respectively. Horses receiving the opioid, butorphanol, during surgery needed significantly fewer analgesic agents post-operatively (Johnson et al., 1993).

In a double-blind, randomized, prospective study of human patients undergoing arthroscopic surgery, those receiving a prostaglandin inhibitor (naproxen sodium) had significantly less pain, less synovitis, less effusion and faster recovery (Ogilvie-Harris et al., 1985; Rasmussen et al., 1993) than those without. In equally as large a prospective study, no advantages were observed over control group of patients when compared to physical therapy and administration of the NSAID, diclofenac (Birch et al., 1993).

Effects of Phenylbutazone on naturally occurring osteoarthritis.

In a randomized controlled clinical trial, efficacy and safety of paste formulations of firocoxib (Equioxx®) and PBZ in horses with naturally occurring osteoarthritis were compared. Horses were treated with firocoxib (0.1 mg/kg, orally every 24 h) or phenylbutazone (4.4 mg/kg, orally every 24 h) for 14 days. Clinical improvement was defined as a reduction of at least 1 lameness score grade or a combined reduction of at least 3 points in scores for pain during manipulation or palpation, joint swelling, joint circumference, and range of motion. Results suggested some greater improvement in some categories tested following firocoxib, but overall clinical efficacy of firocoxib and PBZ in horses were comparable (Doucet et al., 2008).

Horses with naturally occurring forelimb and hind limb lameness were exercised on a treadmill and the degree of lameness evaluated by the use of kinematic analysis while horses were trotting on the treadmill. Horses entered into the study were judged to have AAEP lameness scores of 1 to 3 based on a scale of upper severity score of 5. In a cross-over study, PBZ paste was administered at 2.2 mg/kg (orally every 12 h for 5 days), alone or in combination with flunixin meglumine administered at 1.1 mg/kg, (IV every 12 h for 5 days). Lameness evaluations were performed before and 12 hours after administration of two NSAID treatment

regimens. Administration of a combination of the two NSAIDs alleviated lameness more effectively than did oral administration of phenylbutazone alone. Base on the authors' conclusion when evaluating all 28 horses, there was a significant clinical improvement after the administration of both drugs in all horses except 5 with forelimb lameness. PBZ alone did not result in significant clinical improvement in all horses. This study suggested that the use of combinations of NSAIDs (stacking) did have a better effect at 12 hours and would have a greater effect at 24 hours and the "stacking of drugs" should be a real concern. (Keegan et al., 2008).

The analgesic effects of phenylbutazone in 9 horses with chronic forelimb lameness were studied. The horses were administered saline control or PBZ at doses of 4.4 and 8.8 mg/kg IV daily for 4 days. Peak vertical force (force plate) was measured and AAEP clinical lameness scores were assigned before initiation of each treatment. All horses were evaluated 6, 12, and 24 hours after the final dose. The vertical force was significantly increased at all post-treatment evaluation times after PBZ compared to control horses. Clinical lameness scores were significantly decreased 6 and 12 hours at both doses but were only significantly decreased 24 hours after treatment with the higher dose (Hu et al., 2005).

Force plate analysis and the AAEP lameness scoring system were used to evaluate the analgesic efficacies of flunixin (1.1 mg/kg), PBZ (4.4 mg/kg), or physiologic saline solution administered IV in 12 horses with navicular syndrome. Medications were administered once daily for 4 days with a 14-day washout period between treatments. At 6, 12, and 24 hours after the fourth treatment, AAEP lameness evaluations and force plate data indicated significant improvement in lameness from baseline values in horses treated with flunixin or phenylbutazone, compared with saline controls. The effect of flunixin or phenylbutazone was maintained for at least 24 hours but no differences from control were noted at 30 hours. Flunixin meglumine and PBZ appear to have similar analgesic effects in horses with navicular syndrome (Erkert et al., 2005).

The analgesic effects of the nonsteroidal anti-inflammatory drugs, ketoprofen at 2.2 and 3.63 mg/kg and PBZ at 4.4 mg/kg were compared in 7 horses with bilateral forelimb chronic laminitis. Hoof pain was quantified objectively by means of an electronic hoof tester and lameness was subjectively graded on a modified Obel scale. Ketoprofen administered at a dose of 3.63 mg/kg (equimolar to a dose of 4.4 mg/kg of phenylbutazone) reduced hoof pain and lameness score to a greater extent than the 2.2 mg/kg dose of ketoprofen or the 4.4 mg/kg dose of PBZ. These data suggested that ketoprofen, at the dose of 3.63 mg/kg, was more potent than phenylbutazone in alleviating chronic pain and lameness in horses. PBZ and both doses of ketoprofen were still effective at 24 h (Owens et al., 1995).

Effects of phenylbutazone on an induced lameness model.

The objective was to test the hypothesis that PBZ alleviates lameness in an adjustable heart bar shoe model of equine foot pain following a single IV dose of 4.4 mg/kg. Heart rate and lameness score (1-5) were assessed every 20 min for 2 h and then hourly through 9 h. A lameness grade of 4 was produced for the study

and no lameness was observed following the study when the set screw was removed. In the PBZ-treated horses the lameness score was lowest between 4-5 h post-treatment when the score was reduced from 4 to 1.5 compared to control horses. PBZ was efficacious in alleviating lameness in this model. The PBZ plasma concentrations were approximately 15 and 7 μg/ml at 4 and 8 hours, respectively. The study period did not include observations beyond 9 hours, but the lameness score had not recovered to baseline values at that time (Foreman et al., 2008).

Indirect assessment of duration of NSAID effects

Vane in 1971 suggested that the mechanism of the action of aspirin-like compounds was a direct inhibition of prostaglandin synthetase, thereby preventing prostaglandin biosynthesis (Vane, 1971; Moncada et al., 1974; Moncada et al., 1975; Vane and Botting, 1987).

The NSAIDs are potent inhibitor of the conversion of arachidonic acid to arachidonic acid-derived mediators of inflammation. The NSAID's site of action is the cyclooxygenase pathway, therefore, blocking the synthesis and release of prostacyclin, thromboxane, PGE_2, and PGF_2. All NSAIDs have similar modes of action accounting for both their therapeutic and toxic effects (Lees and Higgins, 1985). There have been considerable advances in the development of pharmacodynamic/pharmacokinetic models in veterinary and human medicine and investigators are studying the effects of the drug and concurrent changes in plasma or tissue concentrations of inflammatory mediators. Modern PK/PD studies link the effect(s) of the drug to its corresponding concentration in plasma. Pharmacokinetic (PK) studies determine the effects of the horse on the drug whereas pharmacodynamic (PD) studies determine the effects of the drug on the horse. (Lees, 2004; Lees et al., 2004a; Lees et al., 2004b; Toutain and Lees, 2004). General PD/PK models have been developed for describing drug actions on various active metabolites and hormones (Krzyzanski and Jusko, 2001; Puchalski et al., 2001).

A number of studies have used the reduction in the metabolic products of inflammation as indirect models of the actions of PBZ and other NSAIDs at the molecular level on the degree and duration of action. Three types of models have been used:

1. Suppression of the release of inflammatory mediators in clotted blood samples. A number of PK/PD models have been developed using this technique (Lees et al., 1987a; Soma et al., 1992; Lees et al., 2004b; Lees et al., 2004c).
2. Suppression of the release of inflammatory mediators in tissue cage and sponge models in which a sterile carrageenan solution is injected into the cage or sterile carrageenin-soaked polyester sponge strips are inserted subcutaneously. Both are based on the creation of a mild, reproducible and reversible inflammatory reaction that causes minimal distress to the experimental animals. The acute inflammatory exudates have been shown to contain proteins, white blood cells, and eicosanoids all as a

result of the inflammatory reaction (Higgins and Lees, 1984; Lees and Higgins, 1984; Higgins et al., 1987a; Lees et al., 1987b).

3. More recently, models in humans have used flow through methods to harvest inflammatory exudates. In-vivo human bioassay can be used to study human volunteers and patients. Samples are collected from pertinent tissue sites such as the skin via aseptically inserted micro dialysis catheters. These experiments measured inflammatory substances in interstitial fluid collected from non-inflamed and experimentally inflamed skin (Angst et al., 2008a; Angst et al., 2008b).

Indirect Plasma Models

This study involved the inhibitory actions of NSAIDs on TXB_2 following a single dose of flunixin (1.1 mg/kg) or PBZ (4.4 mg/kg) to determine the duration of action of these drugs. Flunixin and PBZ produced similar degrees of reversible inhibition of TXB_2 at 4 (98% and 88%), 8 (77% and76 %), and 24 (63 and 50 %) hours, respectively. At 48 hours, inhibition of TXB_2 was no longer apparent (Lees et al., 1987a).

In a similar study the concurrent administration of flunixin meglumine (1.1 mg/kg, IV) and PBZ (2.2 mg/kg, IV) on the pharmacokinetics of each drug indicated that the pharmacokinetic variables calculated for each drug when administered alone and in combination were similar. Serum TXB_2 production was significantly suppressed for 8, 12, and 24 hours after administration of flunixin and phenylbutazone in combination. When these drugs were administered alone, the TXB_2 concentrations were not significantly different from control values at 24 h. Note in this study that the dose of PBZ was 2.2 mg/kg. (Semrad et al., 1993).

Indirect Tissue Models.

Distribution of PBZ and its active metabolite OPB into tissue fluids was studied by measuring concentrations in plasma, tissue-cage fluid, peritoneal fluid and acute inflammatory exudate harvested from a polyester sponge model of inflammation in ponies. PBZ and OPB readily penetrated into inflammatory sites. After six hours, the concentration of PBZ was higher in exudate than in plasma and remained so at 24 hours. Mean concentrations of OPB in all fluids were lower than those of PBZ at all times, but OPB readily entered body fluids, especially into inflammatory exudate; suggesting that OPB may contribute to the anti-inflammatory effect. The estimated elimination half-life of PBZ from exudate was 24 h compared to 5 h from plasma. These authors suggested that the persistence of PBZ and OPB in tissues exudates extended the duration of PBZ effectiveness (Lees et al., 1986).

Other studies have shown that flunixin was also cleared more slowly from equine tissue inflammatory exudate than from plasma (Higgins et al., 1987b).

Acute inflammation was induced in 7 ponies by subcutaneous implantation of sterile carrageenin-soaked polyester sponge strips. Treatment comprised a single therapeutic dose of 4.4 mg/kg of PBZ administered intravenously at the time of sponge implantation. Exudates were harvested at 6, 12 and 24 hours and examined

for leukocyte and erythrocyte numbers. Leukocyte numbers were significantly increased from 6-hour values at 12 and 24 hours in both control and PBZ-treated animals but differences between control and treated ponies were not significant. The administration of PBZ produced highly significant reductions in exudate concentrations of PGE_2 and 6-keto-$PGF_1\alpha$ 6 hours. Significant reductions in these eicosanoid concentrations were maintained in treated animals for 12 and 24 hours. Concentrations of TXB_2 were reduced in treated animals at 6 and 12 hours but these changes were not significant. Study results suggested an effect at 24 hours based on the two measured eicosanoids, PGE_2 and 6-keto-$PGF_1\alpha$ (Higgins et al., 1984).

In a 12-day treatment schedule, 5 ponies were administered an oral paste formulation of PBZ and 5 matched ponies were given equivalent doses of a placebo paste. On day 12, a mild, non-immune inflammatory reaction was induced subcutaneously. Exudate was collected at 4, 8, 12, and 24 hours. There were no significant differences in exudate protein concentration and leukocyte numbers between the treatment groups, but exudate concentrations of 6-ketoF1α were reduced at 4, 8, and 12 hours and those of TXB_2 at 8, 12, and 24 h in the PBZ treatment group. The increases in surface skin temperature were significantly less in PBZ-treated than in placebo-treated ponies between 4 and 24 hours (Lees and Higgins, 1986).

Leucocyte and erythrocyte accumulation in exudate were not affected in any of the tissue cage and exudate studies. The hypothesis of prostaglandin synthetase inhibition is the most widely accepted mode of action for the NSAIDs, but other actions of NSAIDs including leukocyte migration, neutrophil aggregation, lysosomal enzyme release, and superoxide radical generation have been demonstrated. In-vitro studies have shown that flunixin, PBZ, OPB, and indomethacin suppress leukocyte migration. Flunixin was the most potent of the 4 drugs studied (Dawson et al., 1987).

Conclusions

This review presented an historical prospective and examined the information presented in 4 different models used to determine the effects of NSAIDs, especially PBZ. They included naturally occurring lameness, reversible induced lameness, and indirect plasma and tissue models studying the suppression of the release of arachidonic-derived mediators of inflammation. The majority of studies suggest an effect of PBZ at 24 hours at a dose of 4.4 mg/kg. This reflects and substantiates the opinion of many clinical veterinarians, many of whom will not examine a horse for a pre-purchase lameness examination unless the horse is shown to be free of NSAIDs and glucocorticoids. This remains the opinion of many Commission Veterinarians in that they wish to examine a horse pre-race without the possibility of a NSAID or corticosteroid interfering with the examination and masking a possible musclo-skeletal condition. Based on scientific reports and the impression of clinical veterinarians, residual effects of PBZ remain at 24 hours. The impact of this sustained effect on the health and welfare of the horse remains problematic.

Footnote: Memo to Dr Robert Gowen, October 21, 1991 from Tom Tobin; Short overview on phenylbutazone and its regulations.

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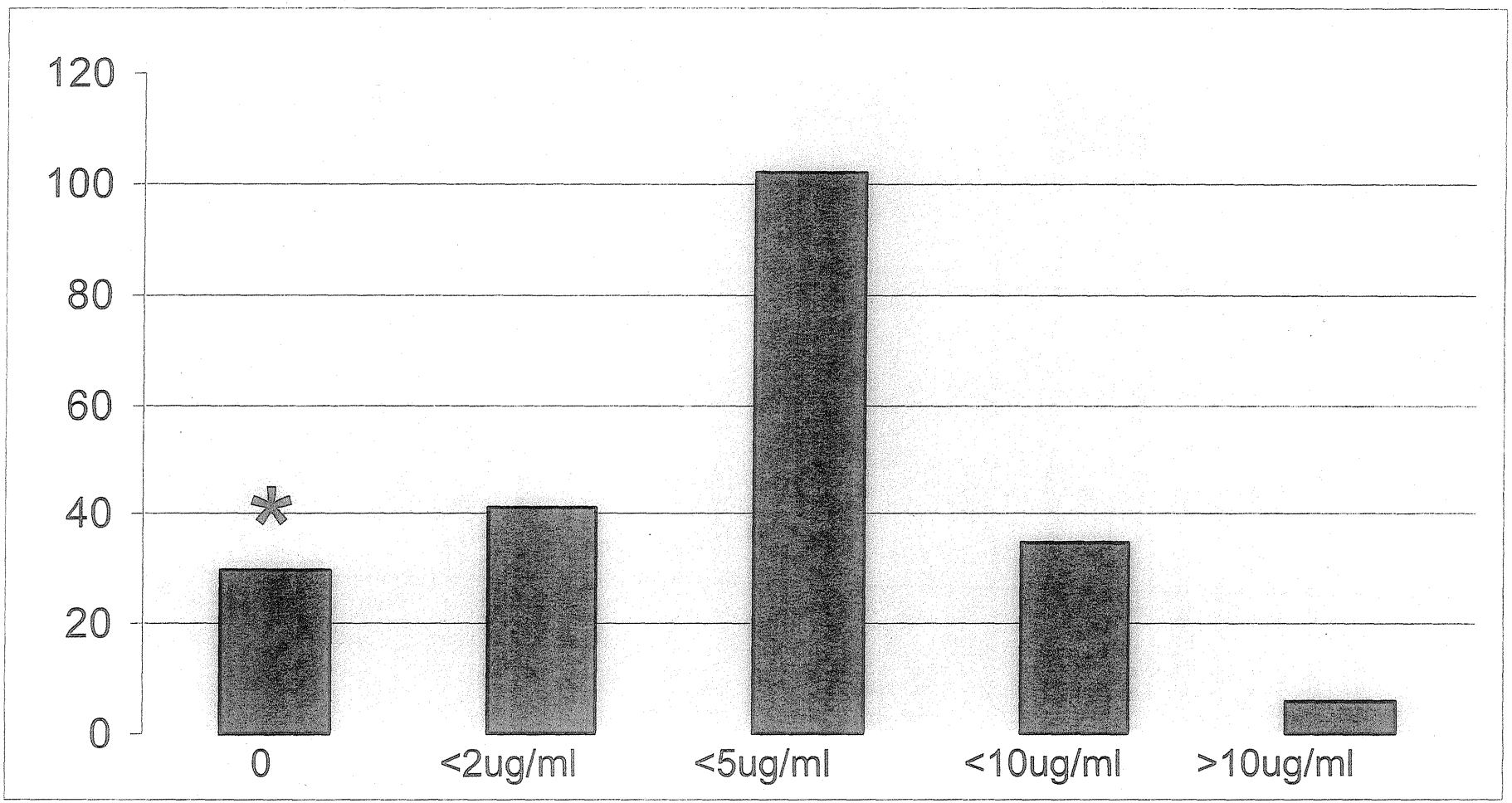
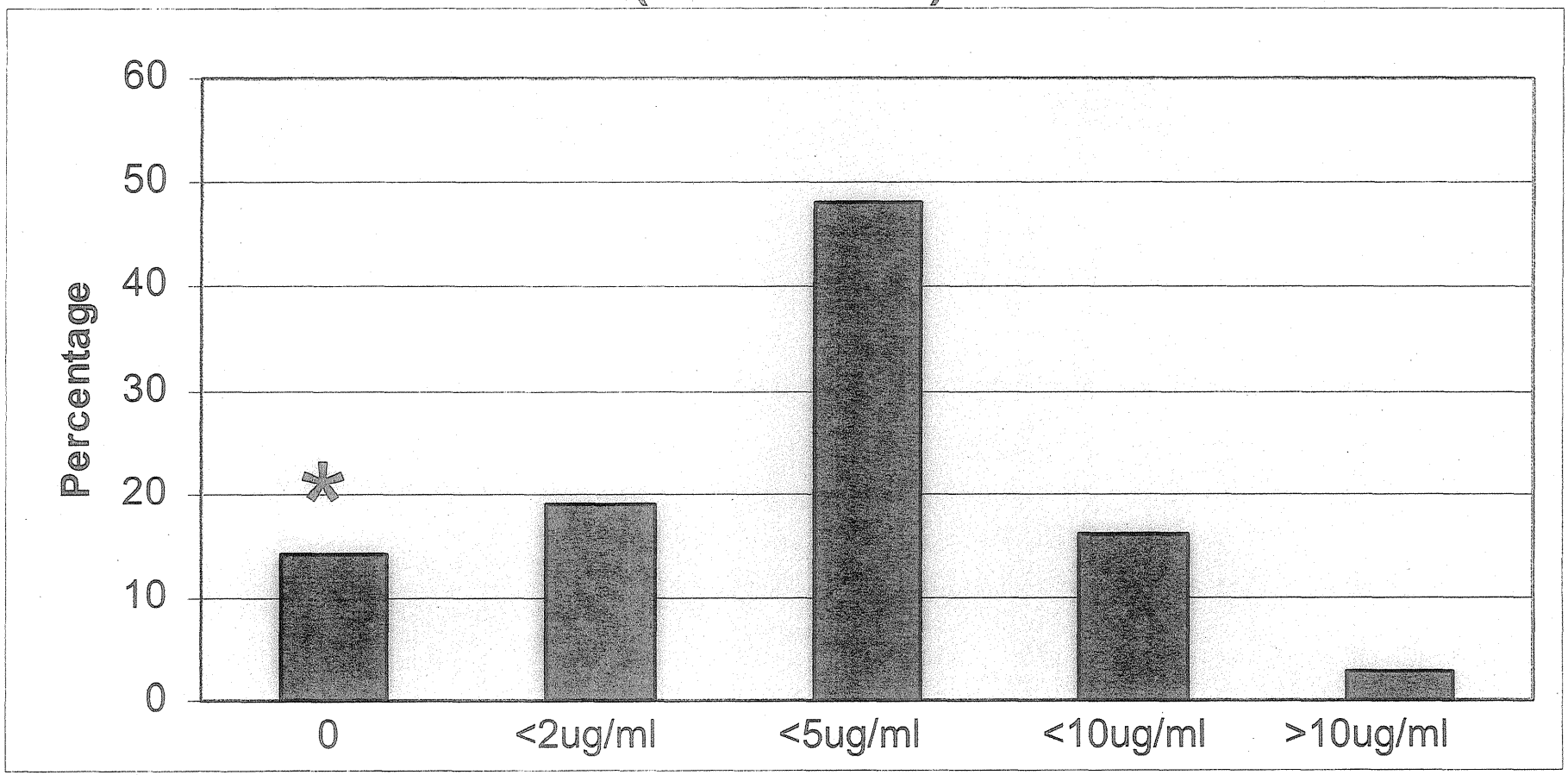


* Includes 10 samples with flunixin in excess of regulatory threshold

CA & KY Exam Time Samples (n= 214)



* Includes 10 samples with flunixin in excess of regulatory threshold

RMTC Recommends New Phenylbutazone Threshold Level, Announces Launch of Recent Rulings... 4/15/10

RMTC
NEWS RELEASE
April 15, 2010
Contact: Hallie Lewis (859) 224-2848

RMTC RECOMMENDS NEW PHENYLBUTAZONE THRESHOLD LEVEL, ANNOUNCES LAUNCH OF RECENT RULINGS DATABASE

Based on the recommendation of its Scientific Advisory Committee, the Racing Medication and Testing Consortium (RMTC) board of directors has revised its recommended threshold for the non-steroidal anti-inflammatory drug (NSAID) phenylbutazone from 5 micrograms per milliliter to 2 micrograms per milliliter of plasma or serum. Under the current model rule, one of three approved NSAIDs is permitted at least 24 hours prior to racing. The permitted NSAIDs are phenylbutazone, flunixin and ketoprofen.

"This action was taken in response to concerns expressed by the regulatory veterinary community regarding the effects of NSAID administration on pre-race examinations," said Dr. Rick Arthur, chair of the RMTC Scientific Advisory Committee and equine medical director for the California Horse Racing Board (CHRB). "The committee felt that the regulatory veterinarians' concerns that NSAID levels may compromise pre-race examinations were justified. This adjustment in permitted phenylbutazone level is measured and appropriate." The Scientific Advisory Committee will continue to research whether further restrictions on NSAID use are warranted.

The board made this recommendation at its meeting in Lexington, Ky., on April 12, 2010, held in conjunction with the Association of Racing Commissioners International 2010 Annual Conference.
Dr. Tom David, equine medical director for the Louisiana State Racing Commission, applauded the RMTC board's action.

"We are very pleased with this step and appreciate the RMTC's willingness to address the concerns of the regulatory veterinarians," said Dr. David. "We look forward to a continuing dialogue on the subject."
Also during the board meeting, RMTC Director of Communications Hallie Lewis announced the creation of a Recent Rulings Database to go live immediately on rmtcnet.com.

"I am excited about this project because I believe it fulfills a need in the industry," said RMTC Executive Director Dr. Scot Waterman. "Members of HANA (Horseplayers Association of North America), racing fans and participants have voiced their desire for transparency and easy-to-access data on drug-related rulings. To our knowledge, this is the only database in existence that allows viewers to see not only the infractions but also a description of the possible uses and effects of the drug in the horse. We hope that this will begin the process of better clarifying the difference between medication management mistakes and more serious drug violations."

Dr. Rick Sams of the University of Florida Racing Laboratory presented an update on RMTC-funded research to determine thresholds and withdrawal times for 13 medications often used by practicing veterinarians to treat horses in race training. This research is being conducted at the University of Florida, University of Pennsylvania New Bolton Center, Pennsylvania Equine Toxicology and Research Laboratory, Iowa State University and the University of California-Davis.

"My colleagues and I expect to publish withdrawal time recommendations on acepromazine, boldenone, butorphanol, detomidine, firocoxib, fluphenazine, testosterone, nandrolone, stanozolol and glycopyrrolate in the next 90 to 120 days," Dr. Sams told RMTC board members.

Dr. Waterman announced that negotiations are close to completion to begin the External Quality Assurance Program through an independent third-party provider. Once initiated, the program will provide proficiency, masked and double-masked urine and blood samples for participating testing laboratories to analyze for the presence and quantity of prohibited and regulated drugs.

According to Dr. Waterman, "This is the first step in implementing the RMTC Drug Testing Initiative. We expect to have a full external quality assurance program up and running in 2011."

In the interim, the Testing Integrity Program Inc. and the Interstate Drug Testing Alliance will continue their single- and double-masked sample programs with RMTC subsidization.

Dr. Scott Stanley, director of the University of California-Davis Kenneth L. Maddy Laboratory, presented a report on the CHRB frozen sample and retrospective testing program for growth hormone. This program, based on methods published by LCH, the French racing laboratory, retrospectively tested 2,000 samples that have been frozen over the past two years under the CHRB's drug testing program. The growth hormone analysis of the CHRB's frozen sample program was made possible by funds provided by The Jockey Club. In other RMTC business:

◦ A strategic plan was adopted that outlines research, reorganization and regulatory communications as key areas of focus in the organization's future.

◦ An update was given on the current research strategies involving the intra-articular administration of corticosteroids. ◦ An update on the efforts toward international harmonization of racing medications was given, including the steps undertaken to exchange research data.

◦ RMTC will create a committee to confront regulation issues and encourage uniformity across racing jurisdictions regarding policy language on out-of-competition testing.

◦ Current RMTC officers Dr. Scot Waterman, Dr. Robert Lewis, Alan Foreman, Chris Scherf, Dr. Rick Arthur and Tom Charters were re-elected to serve another term.

◦ It was announced that RMTC CEO Dan Fick has stepped down to focus on his new position as a steward for the Indiana Racing Commission.

"On behalf of the board of directors, I would like to give a heartfelt thank-you to Dan for his tireless efforts to move this organization forward over the last several years. His commitment and dedication were truly remarkable and we wish him the best in his future endeavors," said RMTC Chairman Dr. Robert Lewis.
The RMTC consists of 25 racing industry stakeholders and organizations that represent Thoroughbred, Standardbred, American Quarter Horse and Arabian racing. The organization works to develop and promote uniform rules, policies and testing standards at the national level; coordinate research and educational programs that seek to ensure the integrity of racing and the health and welfare of racehorses and participants; and protect the interests of the racing public.

For additional information, visit the RMTC website at rmtcnet.com or contact Hallie Lewis, RMTC director of communications, at (859) 224-2848.

CALIFORNIA HORSE RACING BOARD

MAY 19, 2010
MEDICATION AND
TRACK SAFETY MEETING

There is no board package material for Item 2

CALIFORNIA HORSE RACING BOARD

MAY 19, 2010
MEDICATION AND
TRACK SAFETY MEETING

There is no board package material for Item 3

CALIFORNIA HORSE RACING BOARD

MAY 19, 2010
MEDICATION AND
TRACK SAFETY MEETING

There is no board package material for Item 4

STAFF ANALYSIS
DISCUSSION AND ACTION REGARDING THE PROPOSED AMENDMENT TO RULE 1844, AUTHORIZED MEDICATION, TO MODIFY THE LEVELS PERMITTED FOR PROCAINE SUBSEQUENT TO PROCAINE PENICILLIN ADMINISTRATION

Medication and Track Safety Committee Meeting
May 19, 2010

BACKGROUND

Business and Professions Code section 19440 provides that the Board shall have all powers necessary and proper to enable it to carry out fully and effectually the purposes of this chapter. Responsibilities of the Board shall include adopting rules and regulations for the protection of the public and the control of horse racing and pari-mutuel wagering. Business and Professions Code section 19562 states the Board may prescribe rules, regulations and conditions under which all horse races with wagering on their results shall be conducted in California. Business and Professions Code section 19580 requires the Board to adopt regulations to establish policies, guidelines, and penalties relating to equine medication to preserve and enhance the integrity of horse racing in California. Business and Professions Code section 19581 provides that no substance of any kind shall be administered by any means to a horse after it has been entered to race, unless the Board has, by regulation, specifically authorized the use of the substance and the quantity and composition thereof. Board Rule 1844, Authorized Medication, names drug substances and medications authorized by the Board that may be administered to safeguard the health of the horse entered to race. The rule lists the drug substances that may be found in official test samples and the level at which such drugs may occur.

ANALYSIS

The proposed amendment to Rule 1844 would increase the amount of procaine that may be present in an official urine test sample from 10 nanograms per milliliter to 50 nanograms per milliliter. The proposed amendment also adds a new subsection 1844(h), which states procaine, following administration of procaine penicillin, is an authorized medication. There may be no more than 25 nanograms per milliliter in the official blood test sample, and the procaine penicillin administrations have to have been reported pursuant to Rule 1842, Veterinarian Report. In addition, the procaine penicillin can not have been administered after entry to race, and the horse has to have been under surveillance for a minimum of six hours prior to racing. The required surveillance would likely be provided and billed in a manner similar to current detention barn surveillance. All expenses related to surveillance and testing for procaine shall be paid by the owner of the horse. The owners will pay the costs because the program requires special test tubes and unique testing. The proposed amendment will allow the use of an inexpensive and effective antibiotic: procaine penicillin, which will save owners money.

RECOMMENDATION

This item is presented for committee discussion and action.

CALIFORNIA HORSE RACING BOARD
TITLE 4. CALIFORNIA CODE OF REGULATIONS
ARTICLE 15. VETERINARY PRACTICES
PROPOSED AMENDMENT OF
RULE 1844. AUTHORIZED MEDICATION

Medication and Track Safety Committee Meting
May 19, 2010

1844. Authorized Medication.

Consistent with the intent of these rules, drug substances and medications authorized by the Board for use may be administered to safeguard the health of the horse entered to race provided that:

(a) No person shall administer a drug substance to any horse entered to race except upon authorization of the official veterinarian in conformance with these rules.

(b) No drug substance, other than authorized bleeder medication, shall be administered to a horse entered to race within 24 hours of the race in which entered.

(c) Not more than one approved non-steroidal anti-inflammatory drug substance (NSAID) may be administered to a horse that is entered to race and shall be only one of the following authorized drug substances:

(1) Phenylbutazone in a dosage amount that the test sample shall contain not more than 5 micrograms of the drug substance per milliliter of blood plasma or serum.

(2) Flunixin in a dosage amount that the test sample shall contain not more than 50 nanograms of the drug substance per milliliter of blood plasma or serum.

(3) Ketoprofen in a dosage amount that the test sample shall contain not more than 10 nanograms of the drug substance per milliliter of blood plasma or serum.

(4) Metabolites or analogues of approved NSAIDs may be present in post race test samples.

(d) If the official chemist reports that a blood test sample contains an authorized NSAID in excess of the limit for that drug substance under this rule, the official

veterinarian shall, in conjunction with the veterinarian who administered or prescribed the authorized drug substance, establish a dosage amount or time of administration of the drug substance that will comply with the limits under this rule; or the official veterinarian may, if in his/her judgment no such reduced dosage amount or amendment to time of administration will result in a test sample level within the limits of this rule, withdraw authorization for the use of any one NSAID.

(e) Official urine test samples may contain one of the following drug substances, their metabolites or analogs, in an amount that does not exceed the specified levels:

(1) Acepromazine; 25 nanograms per milliliter

(2) Mepivacaine; 10 nanograms per milliliter

(3) Promazine; 25 nanograms per milliliter

(4) Albuterol; 1 nanograms per milliliter

(5) Atropine; 10 nanograms per milliliter

(6) Benzocaine; 50 nanograms per milliliter

(7) Procaine; ~~10~~ 50 nanograms per milliliter

(8) Salicylates; 750 micrograms per milliliter

(9) Clenbuterol; 5 nanograms per milliliter

(10) Stanazolol; 1 nanograms per milliliter

(11) Nandrolone; 1 nanograms per milliliter for geldings, fillies and mares; 45 nanograms for males other than geldings.

(12) Boldenone; 15 nanograms per milliliter in males other than geldings.

(13) Testosterone; 20 nanograms per milliliter in geldings.

(A) Testosterone at any level in males other than geldings is not a violation of this regulation.

(14) Testosterone; 55 nanograms per milliliter in fillies or mares

(f) Official blood test samples may contain clenbuterol in an amount not to exceed 25 picograms per milliliter of serum or plasma.

(g) Official blood test samples shall not contain any of the drug substances, or their metabolites or analogs listed in subsection (e)(1)-(8), and (e)(10)-(14).

(h) Procaine, following administration of procaine penicillin, is an authorized medication provided:

(1) Official blood test samples shall not contain any procaine, or its metabolites of analogs listed in excess of 25 nanograms per milliliter.

(2) all procaine penicillin administrations have been reported pursuant to Rule 1842 of this division,

(3) procaine penicillin was not administered after entry to race,

(4) the horse was under surveillance for a minimum of six hours prior to racing.

(~~h~~i) All expenses related to surveillance and testing for procaine under subsection (h) of this regulation shall be paid by the owner of the horse.

Authority: Sections 19440 and 19562,
Business and Professions Code.

Reference: Sections 19580 and 19581,
Business and Professions Code.

A Review of Postmortem Findings in Cases of Pneumonia in California Racehorses

Cynthia Kollias-Baker, DVM, PhD and Bill Johnson, DVM

Pneumonia is an important cause of mortality and morbidity in racehorses. Thoroughbred racehorses, in particular, appear to be at an increased risk of developing clinical pneumonia. While *Streptococcus spp.* and *Actinobacillus spp.* were the most commonly isolated organisms from pneumonia cases, mixed infections of aerobic and anaerobic organisms were not uncommon. Almost one-third of the cases where pneumonia was found on postmortem examination apparently had not been diagnosed antemortem. Authors' address: California Veterinary Diagnostic Laboratory System, School of Veterinary Medicine, University of California, Davis, CA 95617-1770. © 1999 AAEP.

1. Introduction

Since 1990 the California Veterinary Diagnostic Laboratory System has performed a complete necropsy on every horse that dies or is euthanized on racetracks or training facilities governed by the California Horse Racing Board (CHRB). This program provides a unique opportunity to study causes of mortality in California racehorses. Through this program, a great deal has been learned regarding musculoskeletal injuries in racehorses, but to date the significance of respiratory disease in this population has not been determined. The objective of this study was to evaluate the case reports from the CHRB Postmortem Program to determine whether there are specific aspects to pneumonia in racehorses which may contribute to the morbidity and mortality in this population.

2. Materials and Methods

For this study, CHRB postmortem case reports from 1990 to 1998, where pneumonia and/or pleuritis were diagnosed on the basis of postmortem findings, were reviewed. The reason for submission, clinical and therapeutic histories, results of bacterial cultures, antibiotic sensitivity testing, and other pertinent information was recorded.

3. Results

From February 1990 to December 1998, a total of 71 horses was submitted to CVDLS through the CHRB postmortem program with a primary complaint of pneumonia and/or pleuritis. On postmortem examination, however, pneumonia and/or pleuritis were found in an additional 30 horses that were submitted for nonrespiratory causes. As shown in Fig. 1, the number of horses submitted each year with a primary complaint of pneumonia or pleuritis varied from a low of 5 in 1990 and 1991 to a high of 11 in 1993. The total number of pneumonia and/or pleuritis cases diagnosed on postmortem examination per year varied as well, from a low of 8 in 1990 and 1991 to a high of 16 in 1993 (Fig. 1). Of the total 101 cases, more than 80% were Thoroughbreds (n = 85); the remaining cases were Quarter Horses (n = 8),

NOTES

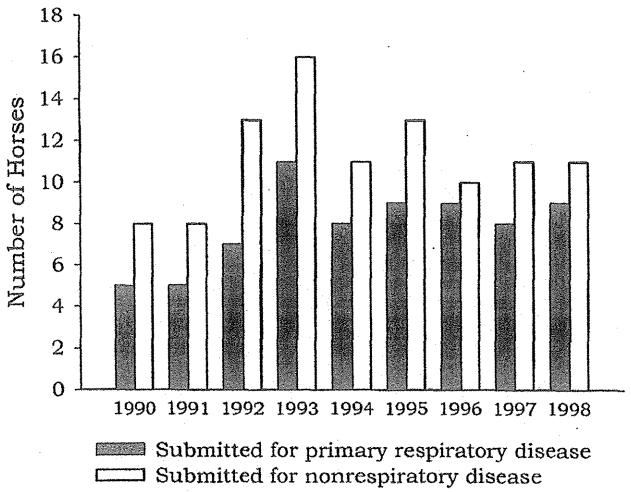


Fig. 1. Number of racehorses submitted for necropsy each year to the CVDLS in which pneumonia and/or pleuritis were diagnosed on postmortem examination.

Standardbreds (n = 4), Appaloosas (n = 3), and an Arabian. In addition, more than half of the submitted horses were either 2 or 3 years of age (n = 59), but cases included horses up to 7 years of age. Finally, in the total 101 cases, there were twice as many geldings and stallions as mares.

Aerobic bacterial isolates were obtained in the majority of cases where cultures were attempted (n = 103 total isolates). As shown in Table 1, multiple bacterial species were isolated from these cases. *Streptococcus equi* subsp. *zooepidemicus* and *Actinobacillus suis*–like *spp.* were isolated from 38 and 17 of the cases, respectively. Overall, single isolates were rare, with most cultures yielding multiple organisms. *Streptococcus equi* subsp. *zooepidemicus* was, however, the single aerobic isolate from 13 cultures. In addition, *Actinobacillus, Pasteurella, Klebsiella,* and *Pseudomonas* were also rarely cultured as single isolates. Anaerobic cultures usually yielded multiple isolates, but *Fusobacterium spp.* and *Bacteroides spp.* were the single anaerobic isolates in 8 and 3 cases, respectively.

Table 1. Frequency of Isolation of Bacteria from 68 Horses with Pneumonia (103 Isolates).

Bacteria	Isolates No.	Isolates %
Gram-positive organisms		
Streptococcus equi		
subsp. *zooepidemicus*	38	36.9
subsp. *equi*	1	0.9
Actinomyces pyogenes	1	0.9
Gram-negative organisms		
Actinobacillus suis-like *spp.*	17	16.5
E. coli	7	6.7
Pasteurella	7	6.7
Pseudomonas	3	2.9
Klebsiella	2	1.9
Misc./Mixed	10	9.7
Anaerobic organisms		
Fusobacterium spp.	8	7.8
Bacteroids spp.	3	2.9
Mixed Anaerobes	5	4.8
Mycotic	1	0.9

Antibiotic sensitivity testing on the isolated bacteria was not routinely performed (n = 18). The results of antibiotic sensitivity testing on *Streptococcus spp.* isolates, however, demonstrated a fairly consistent pattern. All of the isolates (15/15) were either resistant or had intermediate sensitivity to tetracycline, and one-third (5/15) were either resistant or had intermediate sensitivity to sulfonamides. Occasionally, *Streptococcus spp.* isolates were also resistant to trimethoprim/sulfonamide combinations (2/15) and enrofloxacin (4/15). All of the *Streptococcus spp.* isolates, however, were sensitive to ceftiofur, erythromycin, and penicillin.

The *Actinobacillus spp.* isolates demonstrated a fairly variable sensitivity pattern. Isolates were consistently sensitive to ceftiofur and gentamycin and resistant to sulfonamides and beta-lactam agents.

A history of specific antibiotic therapy was provided in 20 cases submitted with a primary complaint of pneumonia. Penicillin was used as the sole antibiotic therapy in 5 cases and was used in combination with other agents in 4 cases. The combination of penicillin and gentamycin was only used in 2 cases. Chloramphenicol was used as the sole antibiotic agent in 3 cases and in combination with other agents in 6 cases. Finally, metronidazole was not used alone, but was used in combination with other agents in 6 cases. Other antibiotic agents listed included tetracycline, sulfonamides, ceftiofur and vancomycin.

One or more coexisting diseases, in addition to pneumonia, were found in numerous cases. In cases that were submitted with a primary complaint of pneumonia and/or pleuritis (n = 71), laminitis was present in 6 horses and colitis in 4. In horses submitted for nonrespiratory disease, in which pneumonia and/or pleuritis was diagnosed on postmortem examination, colitis (n = 9), laminitis (n = 5), and severe musculoskeletal injuries (n = 5) were the most common reasons for submission. Other reasons for submissions included exercise-induced pulmonary hemorrhage, neurologic disease, and sudden death.

4. Discussion

A review of cases submitted through the CHRB Postmortem Program to the CVDLS indicates that pneumonia is a significant cause of mortality in California racehorses. Every year on California racetracks anywhere from 3 to 11 horses die or are euthanized because of pneumonia or its complications, which include pleuritis, colitis, and laminitis. The average number of horses submitted through the CHRB Postmortem Program each year from

1990 to 1998 was 237 ± 19 SD. Pneumonia and its complications, therefore, account for approximately 3% of the mortality in California racehorses (n = 8 ± 2.4 horses/year). The average incidence of pneumonia in racehorses submitted to CVDLS, which includes cases submitted for nonrespiratory disease, was over 4% (n = 11 ± 2.5 horses/year). The total morbidity due to pneumonia, however, is likely significantly higher, as these numbers do not include horses suffering from pneumonia that were subsequently shipped from the track environment.

The profile of the horse most likely to be submitted with a primary complaint of pneumonia is a 2- or 3-year-old Thoroughbred gelding or stallion. This profile is not surprising, as it would also describe the largest proportion of California racehorses. Nevertheless, it would appear that the Thoroughbred is most at risk for developing significant lower respiratory disease.

Based on the findings of this study, the organism most likely to be isolated from racehorses suffering from pneumonia is *Streptococcus equi* subsp. *zooepidemicus,* which was isolated from nearly 40% of all of the cultures. In previous studies, *Streptococcus spp.* were generally among the most common isolates.[1–3] *Actinobacillus suis*–like *spp.* were isolated from 16% of the cases, often in conjunction with *Streptococcus spp.* This finding is also consistent with the results of other studies.[1,4] Other mixed Gram-negative and Gram-positive cultures and single Gram-negative organisms were isolated in 27% of the cases. Single and mixed anaerobic isolates were obtained in 17% of the cases. In addition, although cultures were negative, anaerobic infections were suspected in a number of cases based on post mortem findings.

The history of antibiotic therapy was provided in 20 cases submitted with a primary complaint of pneumonia. As expected, penicillin, either alone or in combination, was one of the most common agents used in these cases. The culture and antibiotic sensitivity testing results obtained in these cases support the use of penicillin as an empiric treatment for pneumonia in horses. All of the *Streptococcus spp.* that were tested were sensitive to penicillin. Nevertheless, in the majority of cases, organisms, such as *Actinobacillus spp.* and other Gram-negative aerobics, which are relatively resistant to penicillin, were also present.[5] Therefore, a broad-spectrum approach would appear to be warranted in most equine pneumonia cases. The combination of penicillin and gentamycin, however, was listed in only two cases. As synergy between aminoglycosides and beta-lactam antibiotics has been well-documented, it is surprising that this combination was not more widely reported.

Chloramphenicol was also commonly used, either alone or in combination with other agents in these pneumonia cases. Chloramphenicol is considered a broad-spectrum agent with efficacy against aerobic and anaerobic organisms and with a wide distribution due to its high lipophilicity. Nevertheless, it is a bacteriostatic agent, and therefore, its use in life-threatening infections, such as acute and/or severe pneumonia, is questionable. It is unclear from the histories, however, whether chloramphenicol was used after treatment with other agents was deemed unsuccessful. In addition, chloramphenicol rarely has been shown to cause irreversible bone marrow suppression in humans, and so its use also presents a human health hazard for personnel handling the drug.

Commonly, metronidazole was also used in these cases. Considering the fairly common isolation of anaerobic organisms in this study, the inclusion of this agent would appear to be prudent therapy.

The results of this study also demonstrate that co-existing diseases are commonly found in the presence of pneumonia. In cases that were submitted for primary respiratory disease, laminitis and/or colitis were frequently reported to have developed subsequent to the pneumonia. In addition, numerous horses that were submitted for primary complaints of colitis or laminitis were also found to have pneumonia on postmortem examination. In many of these cases, it is unknown which condition was present initially. Finally, postmortem examination revealed the presence of pneumonia in a number of cases that had been submitted for nonrespiratory diseases. These findings indicate that clinically significant cases of pneumonia in racehorses can remain undiagnosed for some time.

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Plasma concentration and local anesthetic activity of procaine hydrochloride following subcutaneous administration to horses

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Objective—To determine the durations of the local anesthetic effect and plasma procaine concentrations associated with 5- and 10-mg doses of procaine hydrochloride (with or without 100 µg of epinephrine) administered SC over the lateral palmar digital nerves of horses.

Animals—6 healthy adult horses.

Procedures—The hoof withdrawal reflex latency (HWRL) period was determined by use of a focused heat lamp before and after administration of procaine with and without epinephrine. Blood samples were collected immediately before determination of each HWRL period to assess plasma concentrations of procaine via liquid chromatography–mass spectrometry–mass spectrometry (LC-MS-MS).

Results—10 but not 5 mg of procaine alone and 5 and 10 mg of procaine administered with epinephrine significantly prolonged the HWRL period (mean durations of effect, 5, 120 and 180 minutes, respectively), compared with baseline values. Plasma procaine concentrations did not correlate well with local anesthetic activity; for example, although the HWRL was prolonged to the maximum permitted duration of 20 seconds at 60 to 180 minutes following administration of the 5-mg dose of procaine with epinephrine in certain horses, plasma procaine concentrations were less than the limit of quantitation of the LC-MS-MS assay.

Conclusions and Clinical Relevance—Small doses of procaine coadministered with epinephrine provided long-lasting local analgesia and resulted in plasma procaine concentrations that were not always detectable via LC-MS-MS. On the basis of these results, the use of regulatory limits or thresholds for procaine concentration in equine plasma samples obtained after racing should be seriously reconsidered. (*Am J Vet Res* 2007;68:495–500)

Pain is a perception that is perhaps shared by all species within the animal kingdom. Pain or nociception is a physiologic response that has been defined in animals as "an aversive sensory experience caused by actual or potential injury that elicits progressive motor and vegetative reactions, results in learned avoidance behavior, and may modify species specific behavior, including social behavior."[1] Nociceptive models of acute pain in animals are often classified by the type of stimulus used to evoke the sensation, and most fall into 1 of 4 categories: electrical, thermal, mechanical, or chemical. An ideal stimulus must be quantifiable, reproducible, and noninvasive.[2] Thermal models have been used to study acute pain in horses for many years.[3] Specifically, the efficacies of numerous analgesic agents commonly administered to horses have been studied by use of a focused radiant light-heat source directed onto the proximal interphalangeal region (pastern) of horses to elicit the classic flexion-withdrawal reflex.[4] In that heat latency model, the acute stimulation of primary afferent fibers (Aδ and C) evokes somatic and autonomic reflexes, which result in an organized escape behavior (hoof withdrawal) that is proportional to the intensity of the stimulus.[5]

Procaine hydrochloride is a relatively weak ester-type local anesthetic agent that has a potency of approximately one fourth that of lidocaine and a short duration of effect in most species. For example, by use of a thermal nociception model, doses of procaine as high as 320 mg reportedly induced local anesthesia in

Abbreviations

LC-MS-MS	Liquid chromatography–mass spectrometry–mass spectrometry
HWRL	Hoof withdrawal reflex latency
d10-procaine	Deuterated procaine hydrochloride
m/z	Mass-to-charge ratio
LOQ	Limit of quantitation

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horses for approximately 60 minutes.[6,7] In those same studies, however, the duration of effect of procaine was significantly prolonged by coadministration of epinephrine (1:100,000 or 100 µg). Some horseracing jurisdictions in North America have adopted regulatory limits for procaine concentration in plasma samples obtained after racing, with no penalty assessed if the plasma procaine concentration in a sample is less than that limit.[8] However, plasma concentrations associated with small but locally effective doses of procaine have not been determined in horses. The purpose of the study reported here was to determine the durations of the local anesthetic effects and plasma procaine concentrations associated with 5- and 10-mg doses of procaine (with and without 100 µg of epinephrine) administered SC over the lateral palmar digital nerves of horses. Horses were administered small doses of the local anesthetic, and a heat latency model was used to evaluate the local anesthetic effects; additionally, an extremely sensitive LC-MS-MS method was developed and validated to quantify procaine concentration in plasma samples from those horses.

Materials and Methods

Horses—Six university-owned Thoroughbreds (3 mares and 3 geldings) that were 3 to 8 years old and weighed 447 to 594 kg were used for this study. The study was approved by the University of Florida Institutional Animal Use and Care Committee. All horses were fed a commercially available grain mixture and hay twice daily and had free access to water at all times. Experiments were conducted in a climate-controlled space under artificial lighting.

Determination of the HWRL period—A heat latency model (previously described in detail by Kamerling et al[4]) was used in the study. Briefly, a focused radiant light–heat lamp (500-W incandescent projection lamp) was directed onto the pastern region of each horse as a noxious stimulus to elicit the classic hoof withdrawal reflex. To ensure that the heat absorption from the lamp was consistent for all horses, the hair on the dorsal and lateral sides of the forelimb pastern region selected for experimentation that day was clipped and the skin blackened with stamp pad ink. Both forelimbs were used during the study, but the treatment limb was chosen at random at the time of each experiment. In addition, the lateral side of the pastern region was marked with white ink in 6 distinct but adjacent sites to indicate where the heat lamp would be directed; this prevented undue tissue damage and ensured that the entire area of the pastern region was evaluated for nociception.

The HWRL period was defined as the interval between initiation of lamp illumination and withdrawal of the hoof. This interval was recorded (in seconds) by use of an electronic timer that was controlled by the lamp activation switch. A secondary unfocused light beam (sham light) illuminated randomly prior to and throughout testing was used to prevent response to visual cues rather than the thermal stimulus. The heat lamp intensity was adjusted for each horse to produce an HWRL period of 5 to 7 seconds' duration under untreated control conditions. Exposure of the skin to the heat stimulus was limited to 20 seconds to avoid induction of thermal injury.

The limb to be tested was randomly selected, and following skin preparation, the baseline HWRL period was determined just prior to the administration of each treatment by calculating the mean duration of 3 consecutively measured HWRL periods. The administration of treatment was recorded as time 0 minutes. During measurement of each HWRL period, the focused heat was directed onto 1 of the 6 sites marked with white ink. After administration of 1 of the 4 treatments, durations of subsequent HWRL periods were determined in the same manner.

The 4 treatments consisted of 5 and 10 mg of procaine (10 mg/mL) administered SC over the lateral palmar digital nerve at the level of the mid-lateral sesamoid bone in the presence and absence of epinephrine (1:100,000 or 100 µg). Each horse was administered each treatment in a random crossover fashion with at least 1 week between each administration. The volume administered via each injection was standardized at 1 mL; therefore, the 5-mg dose of procaine was diluted with 0.5 mL of sterile water. Following administration of 5 and 10 mg of procaine in the absence of epinephrine, HWRL periods were determined at 5, 10, 20, 30, 40, 50, 60, and 75 minutes after application and then every 15 minutes until the last recorded HWRL period duration was within ± 10% of the baseline period duration. Following administration of 5 and 10 mg of procaine combined with epinephrine, HWRL periods were recorded at 5, 10, and 30 minutes after application and then every 30 minutes until the HWRL period duration was within ± 10% of the baseline period duration.

Plasma samples—Blood samples were obtained before each drug administration and at time points corresponding to the HWRL period determinations. Blood samples were collected in duplicate via jugular venipuncture into partially evacuated plastic tubes containing a concentration of 2 mg of NaF/mL. Each one of the tubes was filled to capacity (6 mL) and was then centrifuged at 1,380 × g for 5 minutes immediately after collection. Three mL of each plasma sample was then immediately transferred into a new tube containing 2 mg of NaF/mL. The resulting final concentration of NaF in the plasma samples was approximately 6 mg/mL. The samples were then frozen at –20°C until analyzed via LC-MS-MS.

Detection of procaine in plasma via LC-MS-MS—Procaine concentrations in equine plasma samples were determined via LC-MS-MS. Deuterated procaine hydrochloride[a] for use as an internal standard and procaine hydrochloride[b] were purchased. Standard solutions of procaine and d10-procaine were prepared by dissolving the hydrochloride salt in high-performance liquid chromatography–grade methanol to a concentration of 0.1 mg/mL. Working solutions were prepared by diluting the standard solutions in high-performance liquid chromatography–grade methanol to concentrations of 10, 0.1, and 0.01 µg/mL. Working solutions of drug and internal standards were prepared fresh daily and stored in a refrigerator (3°C) when not in use. Calibration and

positive and negative control samples were prepared in drug-free equine plasma. Calibration samples were prepared from the working solutions of procaine at concentrations of 0.1, 0.2, 0.5, 1.0, 1.5, 3.0, 6.0, 10.0, 20.0, and 40.0 ng/mL. Positive control samples were prepared from the working solutions of procaine in duplicate at concentrations of 0.5, 3.0, and 10.0 ng/mL in drug-free equine plasma. To prevent degradation of procaine, 250 µL of the NaF stock solution (30 µg/µL) was added to the calibration and positive and negative control sample tubes. Frozen plasma samples were allowed to thaw at room temperature (approx 22°C), and the internal standard (d10-procaine) was added to 1-mL aliquots of each of the samples at a volume sufficient to provide a final concentration of 50 ng of d10-procaine/mL. Two milliliters of a 1M bicarbonate-carbonate buffer solution (pH, 11) was then added to each tube, and the samples were vortex mixed. Five milliliters of dichloromethane was then added, and the tubes were mixed by inversion for 10 minutes. The samples were centrifuged, and the organic supernatant was removed and evaporated in a water bath (40°C) under a continuous stream of nitrogen. The resulting residue was reconstituted in 50 µL of deionized water and analyzed via LC-MS-MS.

Both the procaine and the d10-procaine internal standard had a mean retention time of 4.32 minutes. The retention times of the sample peaks were required to be within ± 0.2 minutes of the corresponding retention times for the standard and calibrator peaks to be considered acceptable for analysis. The molecular ions for procaine and d10-procaine were isolated and fragmented, and the resultant full MS-MS spectra were collected. Product ions at m/z 100.0, 119.9, and 163.9 were used for the qualitative identification of procaine. Product ions at m/z 110.0, 119.9, and 163.9 were used for the qualitative identification of d10-procaine. The m/z 100.0 and 110.0 product ions from procaine and d10-procaine, respectively, were used for quantitation. Data analysis was carried out by use of computer software.[c]

In addition to the requirement for retention times of the sample peaks to be within ± 0.2 minutes of the corresponding retention times for the standard and calibrator peaks, the retention times for each peak were not acceptable for analysis if they differed from the mean of all the peaks by more than ± 5% over the course of the run. Positive control samples, comprising duplicates at 2 concentrations, were analyzed before and after each set of test samples. To be acceptable for analysis, the ratios of the retention times of the ions used for quantitation could not differ by more than ± 20% between 2 positive control samples. Those positive control samples were dispersed among the test samples. In addition, the ratios of the retention times of the ions used for quantitation of the calibration samples could not differ by more than ± 20% from the positive control samples. Samples that produced area ratios that differed by more than ± 20% were excluded from data analysis. Positive control samples that produced area ratios that differed by more than ± 20% from the calibration samples provoked a repeat analysis of the sample. Regression analysis of the calibration curve was required to produce a correlation coefficient (r^2) of 0.98 for the curve to be acceptable for data analysis. The LOQ was defined as the lowest concentration that had a precision varying less than ± 20%. The LOQ for this study was determined to be 0.2 ng/mL.

Statistical analysis—Data are presented as mean ± SD. Effects of each of the 2 doses of procaine (5 and 10 mg) with and without coadministration of epinephrine were compared with the times for maximum local anesthetic effect that followed each administration for all 6 horses (n = 24) by use of a 2-way ANOVA. A least squares means test followed as a post hoc correction. Significance was set at a value of $P < 0.05$.

Results

In the present study, the mean HWRL period (an indication of a local anesthetic response) after administration of 5 mg of procaine without epinephrine SC over the lateral palmar digital nerves in horses did not differ significantly from the mean HWRL period measured before treatment because of the large degree of variation in the HWRL responses of the horses (**Figure 1**). For example, the HWRL period was prolonged to the maximum permitted duration of 20 seconds in only 4 of the 6 horses following administration of 5 mg of procaine. In contrast, 10-mg doses of procaine without epinephrine produced a maximum mean prolongation of the HWRL period to 19 ± 2 seconds 5 minutes after administration in 6 of 6 horses (**Figure 2**). Although the effects after administration of the 10-mg dose of procaine did last longer in individual horses, for the group overall, this effect dissipated by 30 minutes when the mean HWRL period was no longer significantly different from the mean baseline HWRL period.

Initially, changes in plasma procaine concentrations paralleled the local anesthetic effects. For example, following administration of the 5-mg dose of procaine without epinephrine, the maximum mean peak plasma concentration of procaine (2.6 ± 2.3 ng/mL) and the mean maximum duration of the HWRL period occurred simultaneously 10 minutes after injection (Figure 1). Following the administration of 10 mg of procaine without epinephrine, the onset of local anesthetic

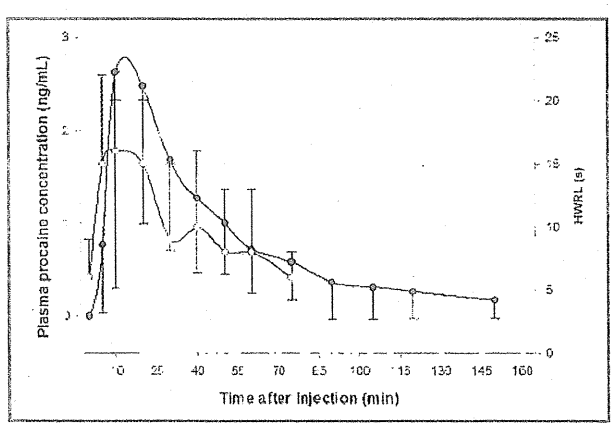

Figure 1—Plasma procaine concentrations (black circles) and duration of HWRL periods (white circles) at intervals before (baseline) and after administration (at time 0) of a 5-mg dose of procaine SC over the lateral palmar digital nerves of 6 horses. Values represent mean ± SD.

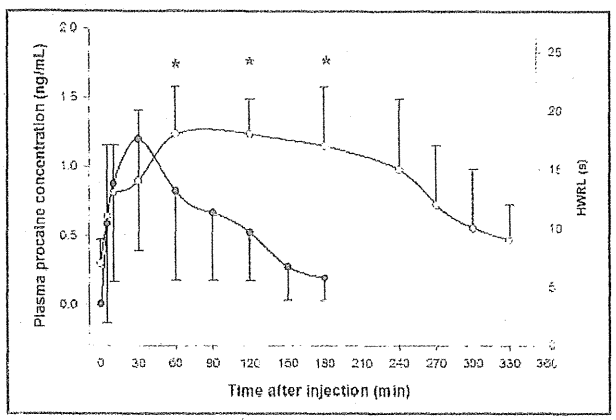

Figure 2—Plasma procaine concentrations (black circles) and duration of HWRL periods (white circles) at intervals before (baseline) and after administration (at time 0) of a 10-mg dose of procaine SC over the lateral palmar digital nerves of 6 horses. Values represent mean ± SD. *Duration of HWRL period at this time point was significantly ($P < 0.05$) different from baseline period.

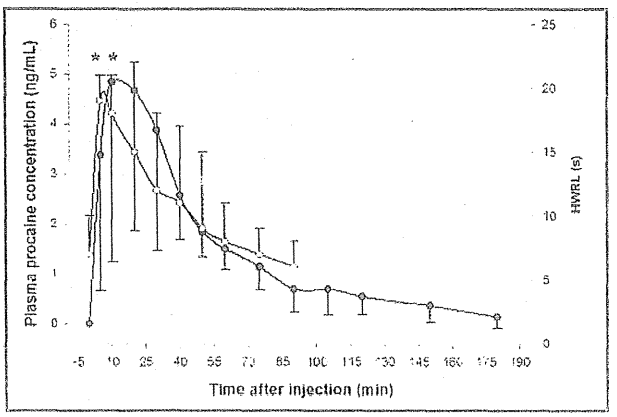

Figure 3—Plasma procaine concentrations (black circles) and duration of HWRL periods (white circles) at intervals before (baseline) and after administration (at time 0) of a 5-mg dose of procaine with epinephrine (10 µL/mL) SC over the lateral palmar digital nerves of 6 horses. Values represent mean ± SD. *See* Figure 2 for key.

activity (as determined by significant prolongation of the HWRL period, compared with the baseline HWRL period) and the mean peak plasma concentration of procaine (4.8 ± 3.6 ng/mL) occurred simultaneously 5 minutes after injection (Figure 2). After determination of the peak value, mean plasma procaine concentration decreased rapidly and was correlated closely with the decrease in the duration of the HWRL period. However, the mean plasma procaine concentration remained higher than the LOQ of the assay (0.2 ng/mL) for approximately 120 and 150 minutes after administration of 5 and 10 mg of procaine, respectively. This was far longer than the mean duration of effect for the 10-mg dose of procaine, which was only 5 minutes. Although the 5-mg dose of procaine did not reliably induce local anesthesia in all of the study horses, it did nevertheless result in plasma procaine concentrations that were readily detectable by the analytic method used in the study.

Compared with findings following injection of procaine alone, administration of 5- and 10-mg doses of procaine with epinephrine SC over the lateral palmar digital nerves of horses resulted in local anesthetic effects that were slower in onset but much longer in duration. In addition, changes in plasma procaine concentrations did not correlate with the local anesthetic effects. For example, when a 5-mg dose of procaine was administered with epinephrine, the peak plasma procaine concentration (1.2 ± 0.8 ng/mL) was detected 30 minutes after administration, but the HWRL period was not significantly different from the baseline period until 60 minutes after injection (**Figure 3**). In a similar manner, the peak plasma procaine concentration following administration of 10 mg of procaine with epinephrine was also detected 30 minutes after administration (4.5 ± 1.8 ng/mL), but the duration of the HWRL period was not significantly different from the baseline period until 60 minutes after injection (**Figure 4**). The mean durations of effect following the administration of 5- and 10-mg doses of procaine with epinephrine were approximately 120 and 180 minutes, respectively; these values were significantly longer than the durations of effect in the absence of epinephrine.

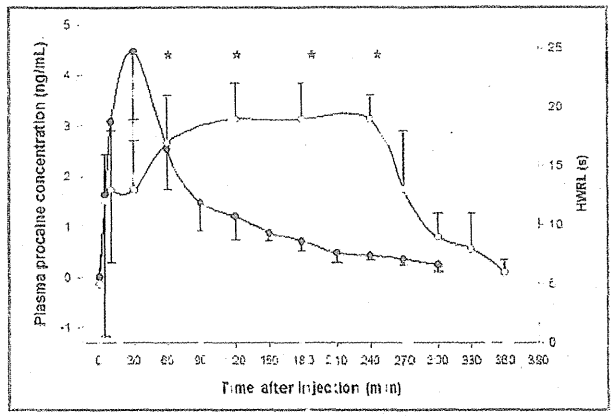

Figure 4—Plasma procaine concentrations (black circles) and duration of HWRL periods (white circles) at intervals before (baseline) and after administration (at time 0) of a 10-mg dose of procaine with epinephrine (10 µL/mL) SC over the lateral palmar digital nerves of 6 horses. Values represent mean ± SD. *See* Figure 2 for key.

When either the 5- or 10-mg dose of procaine was coadministered with epinephrine, the HWRL period remained significantly greater than the baseline period for extended intervals, despite rapid decreases in plasma procaine concentrations (Figures 3 and 4). In 3 of the 6 horses, the local anesthetic effect persisted from 60 to 180 minutes after administration of the 5-mg dose of procaine with epinephrine, despite plasma procaine concentrations that were less than the LOQ of the analytic method used in the study. In contrast, administration of a 10-mg dose of procaine with epinephrine resulted in mean plasma procaine concentrations that were greater than the LOQ of the analytic method for 300 minutes after injection, whereas the mean duration of the HWRL period was significantly greater than the baseline period from 60 to 240 minutes after injection. In addition, plasma procaine concentrations were greater than the assay LOQ at all time points at which a local anesthetic effect was evident as a significant prolongation in HWRL period, compared with the baseline HWRL period, in all of the horses after treatment with 10 mg of procaine with epinephrine.

Discussion

The results of the present study in horses indicated that procaine is a potent local anesthetic agent and that the duration of the drug's anesthetic effects can be quite prolonged in the presence of epinephrine. Although 5 mg of procaine alone did not result in a significant prolongation of the mean HWRL period (compared with the baseline period), that same dose of procaine in the presence of epinephrine significantly increased the duration of the HWRL period for 120 minutes beginning 60 minutes after injection. In a similar manner, the addition of epinephrine increased the mean duration of effect of 10 mg of procaine from 5 to 180 minutes after injection, compared with the baseline value.

The results of the present study also indicated that in both the absence and presence of epinephrine, plasma procaine concentrations do not correlate well with the local anesthetic activity of the drug in horses. In the absence of epinephrine, plasma procaine concentrations following administration of the 5- and 10-mg doses of procaine remained greater than the LOQ of the assay (0.2 ng/mL) long after the HWRL period duration had returned to baseline value. In contrast, when procaine and epinephrine were administered together, local anesthetic activity persisted in some horses even when plasma procaine concentrations were less than the LOQ of the analytic method. It has been hypothesized that epinephrine prolongs the duration of effect of local anesthetics by causing local vasoconstriction, which decreases the elimination rate of the drug from the tissues.[9] However, epinephrine is also known to enhance the potency of lidocaine, and both pharmacodynamic and pharmacokinetic mechanisms have been proposed for this effect.[10] The results of the study reported here confirm that the effect of epinephrine on the duration of local anesthetic activity can be profound.

In the present study, the effect of 5 but not 10 mg of procaine in the absence of epinephrine was similar to that determined in a previous study[7] in horses. For example, the results of that investigation and our study indicated that administration of a 5-mg dose of procaine SC over the lateral palmar digital nerve of horses did not significantly increase the mean duration of the HWRL period from baseline.[7] However, this finding may be misleading because the HWRL period did increase to the maximum permitted duration of 20 seconds in 4 of the horses in the present study; the mean HWRL period was not significantly different from the baseline value because of the high degree of variation in the responses among the horses. This variation was not surprising given the small dose (5 mg) and volume (1.0 mL) that were administered via SC injection that was guided only by digital palpation of the nerves. There may also be some variation in the sensitivity of individual horses to the local anesthetic itself. In our study, the administration of a 10-mg dose of procaine resulted in a mean duration of effect of only 5 minutes. In contrast, in the previous study,[7] the mean duration of effect of 10 mg of procaine was 15 minutes. The reason for the comparatively shorter duration of effect detected in the present study is unclear because the experimental protocols in the 2 studies were identical to the extent that could be achieved feasibly. However, given the relatively small doses of procaine administered and the high degree of interhorse variability in our study, the difference is not surprising.

Our data have important ramifications for some racing jurisdictions in North America. Several jurisdictions in Canada and the United States have adopted regulatory limits for procaine concentrations in plasma samples obtained from horses after racing, with no penalty levied if the procaine concentration in a plasma sample is less than that limit. In Canada, the limit is 25 ng of procaine/mL of plasma in samples obtained after racing, but this plasma concentration is not related to any administered dose of procaine or specific withdrawal period.[8] In fact, prior to completion of the present study, plasma concentrations associated with small but locally effective doses of procaine had not been determined because of the lack of a suitably sensitive analytic method.[7] The use of an LC-MS-MS method with an LOQ of 0.2 ng/mL in the study reported here resulted in detection of procaine for as long as 300 minutes following administration of 10 mg of procaine with epinephrine. Of critical regulatory importance, however, was the finding that a 5-mg dose of procaine with epinephrine prolonged the HWRL period to the maximum permitted duration of 20 seconds, although the plasma procaine concentrations were less than the LOQ of the assay. Because the detection of exogenously administered epinephrine would be extremely difficult, it would not be possible to determine whether procaine had been administered with or without epinephrine. In addition, plasma concentrations of procaine in our study did not exceed 10 ng/mL, which is much less than the concentration permitted by some racing jurisdictions. Altogether, these data have indicated that procaine at any concentration in plasma could be associated with a clinically important local anesthetic effect in horses. Therefore, given these findings, the continued use of regulatory limits set for procaine concentrations in plasma samples obtained from horses after racing has to be questioned.

a. Deuterated procaine hydrochloride, Neogen Corp, Lansing, Mich.
b. Procaine hydrochloride, Sigma Chemical Co, St Louis, Mo.
c. Finnigan Xcalibur qualitative analysis software, Thermo Fisher Scientific, Waltham, Mass.

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CALIFORNIA HORSE RACING BOARD

MAY 19, 2010
MEDICATION AND
TRACK SAFETY MEETING

There is no board package material for Item 6

STAFF ANALYSIS
DISCUSSION AND ACTION REGARDING THE PROPOSED AMENDMENT OF CHRB RULE RULE 1846.5 POSTMORTEM EXAMINATION TO REQUIRE TRAINING AND VETERINARY MEDICAL RECORDS ON ALL HORSES DYING WITHIN A CHRB INCLOSURE

Medication and Track Safety Committee Meeting
May 19, 2010

BACKGROUND

Business and Professions Code section 19440 states that the Board shall have all powers necessary and proper to enable it to carry out fully and effectually the purposes of horse racing law. Business and Professions Code section 19444 provides that in performing its responsibilities the Board may conduct research to determine more fully the cause and prevention of horse racing accidents, the effects of drug substances on race horses, and the means for detection of foreign drug substances. Board Rule 1846.5, Postmortem Examination, requires that every horse which suffers a fatal injury on the racetrack, or which dies or is euthanized within an area under the jurisdiction of the Board, shall undergo a postmortem examination. The regulation requires that a written report of the postmortem examination be filed with the Executive Director, Equine Medical Director and the official veterinarian. It does not state if the owner, trainer or attending veterinarian may receive a copy of the report. In addition, the regulation is silent on whether a copy of the horse's veterinary and training records may be requested by the Board. Such records may provide valuable information and insight in conjunction with the postmortem examination.

ANALYSIS

The proposed amendment to Rule 1846.5 provides that a copy of the postmortem examination shall be provided to the owner, trainer or attending veterinarian upon submission of a written request to the CHRB headquarters office. The written request will guarantee the CHRB has documentation should questions arise regarding dissemination of the postmortem. The proposed amendment also adds a new subsection 1846.5(h), which requires the owner to provide the veterinary medical history of any horse that dies or is euthanized on the grounds of a facility under the jurisdiction of the Board. The new subsection also requires trainers to provide a report of the training history of any horse that dies or is euthanized at a facility under the jurisdiction of the Board. The documents will allow for the correlation of postmortem data with medical records to look for causal relationships. Under the proposed amendment the documents would be considered confidential and their contents shall not be disclosed except as specified under subsection 1846.5(h)(1).

RECOMMENDATION

This item is presented for committee discussion and action.

CALIFORNIA HORSE RACING BOARD
TITLE 4. CALIFORNIA CODE OF REGULATIONS
ARTICLE 15. VETERINARY PRACTICES
RULE 1846.5. POSTMORTEM EXAMINATION

Medication and Track Safety Committee Meeting
May 19, 2010

1846.5. Postmortem Examination.

(a) Every horse which suffers a fatal injury on the racetrack in training or in competition, or which dies or is euthanized within an area under the jurisdiction of the Board, shall undergo a postmortem examination at a diagnostic laboratory which is under contract with the Board to determine the injury or sickness which resulted in euthanasia or natural death.

(b) Test samples may be obtained from the carcass upon which the postmortem examination is to be conducted and sent to the diagnostic laboratory for testing for foreign substances or their metabolites, and natural substances at abnormal levels. When practical, test samples shall be procured prior to euthanasia.

(c) The costs associated with transportation to the diagnostic laboratory of any horse which has died under the provisions of subparagraph (a) shall be the responsibility of the racing association conducting the meeting where the death occurred or the training center or racetrack where death occurred when no meeting is in progress. The services of the official veterinarian and the laboratory testing of postmortem samples for standard necropsy and special equine necropsy examinations shall be made available by the Board without charge to the owner. The cost of any additional necropsy examination(s) requested by the owner or trainer are the responsibility of the requesting individual.

(d) Requests for each postmortem shall be filed with the official veterinarian by the

owner's or trainer's veterinarian within one hour of the death and shall be submitted on a Necropsy Submission Form, CHRB-72, (Rev. 6/04), hereby incorporated by reference, and which is available at all official veterinarian offices. The trainer is co-responsible to supply all information to complete CHRB-72.

(e) If the official veterinarian is not available, the owner's or trainer's veterinarian must phone the diagnostic laboratory within one hour of the death and fax CHRB-72 to the laboratory as notification that the horse is due for necropsy. On the official veterinarian's next scheduled work day, the owner's or trainer's veterinarian shall give the original CHRB-72 to the official veterinarian.

(f) The racing association, racetrack or training center will notify the transporter within one hour of death to have the horse conveyed to the designated laboratory for necropsy.

(g) Upon completion of the postmortem examination the diagnostic laboratory shall file a written report with the Executive Director, the Equine Medical Director and the official veterinarian. The owner, trainer or attending veterinarian shall be provided a copy of the postmortem examination upon submission of a written request to the CHRB headquarters office.

(h) Every owner shall provide to the Board the veterinary medical history of any horse that dies or is euthanized at a facility under the jurisdiction of the Board. Every trainer shall provide to the Board a report of the training history of any horse that dies or is euthanized at a facility under the jurisdiction of the Board.

(1) Any veterinary medical histories and any reports of training history provided to the Board shall be considered confidential and their contents shall not be disclosed except in a proceeding before the stewards or the Board, in exercise of the Board's jurisdiction or

in the analysis of injuries and illnesses causing fatalities as approved by the Executive Director and the Equine Medical Director.

~~(h)~~(i) Each owner and trainer accepts responsibility for the postmortem examination provided herein as a requisite for maintaining an occupational license.

Authority: Section 19440,
Business and Professions Code.

Reference: Section 19444(c),
Business and Professions Code.