Scientific Publications In Support of My Testimony

I. Effect Of Furosemide On Performance Of Thoroughbreds Racing In The United States And Canada. Gross DK, Morley PS, Hinchcliff KW, Wittum TE.

II. Furosemide Reduces Accumulated Oxygen Deficit In Horses During Brief Intense Exertion. K. W. Hinchcliff, K. H. McKeever, W. W. Muir, and R. A. Sams

III. Furosemide-Induced Changes In Plasma And Blood Volume Of Horses. K. W. Hinchcliff, K. H. McKeever, W. W. Muir III


V. Review Of Furosemide In Horse Racing: Its Effects And Regulation. L.R. Soma1, C.E. Uboh2

VI. Hemoconcentration and Oxygen Carrying Capacity Alteration in Race Horses Following Administration of Furosemide Prior to Speed Work, A Pilot Study. Sheila Lyons DVM, FACVSMR

VII. The Use of Blood Doping as an Ergogenic Aid. Sawka, Michael N. Ph.D., FACSM, (Chair); Joyner, Michael J. M.D.; Miles, D. S. Ph.D., FACSM; Robertson, Robert J. Ph.D., FACSM; Spriet, Lawrence L. Ph.D., FACSM; Young, Andrew J. Ph.D., FACSM

VIII. Fracture Risk In Patients Treated With Loop Diuretics. L. Rejnmark, P. Vestergaard, L. Mosekilde

IX. Soft Palate Problems And Bleeding In Racehorses? The Answer Is On The Tip Of The Horse’s Tongue. Robert Cook FRCVS, PhD

X. An Endoscopic Test For Bit-Induced Nasopharyngeal Asphyxia As A Cause Of Exercise-Induced Pulmonary Haemorrhage In The Horse. Robert Cook FRCVS, PhD

Appendix of Scientific Publications By Subject

1) Lasix is Performance-Enhancing: I, II, III, V, VI

2) Lasix is Harmful to the Health and Safety of the Horse in Racing: III, IV, VI, VIII, IX

3) Lasix Use Has Not Ended the Occurrence of Exercise Induced Pulmonary Hemorrhage: V, IX

4) Lasix Increases Risk of Fracture: VIII

5) Conflicts of Interest Affecting Safety In Horse Racing: XI

6) Injury Masking Drug Use and Fatal Breakdowns in Racehorses: XI

7) Regulatory Failure of the Current Horse Racing Industry: XI
OBJECTIVE: To determine the effect of furosemide on performance of Thoroughbreds racing on dirt surfaces at tracks in the United States and Canada.

DESIGN: Cross-sectional study.

ANIMALS: All Thoroughbreds (n = 22,589) that finished a race on dirt surfaces at tracks in the United States and Canada between June 28 and July 13, 1997 in jurisdictions that allowed the use of furosemide.

PROCEDURE: Race records were analyzed by use of multivariable ANOVA procedures and logistic regression analyses to determine the effect of furosemide on estimated 6-furlong race time, estimated racing speed, race earnings, and finish position. Principal component analysis was used to create orthogonal scores from multiple collinear variables for inclusion in the models.

RESULTS: Furosemide was administered to 16,761 (74.2%) horses. Horses that received furosemide raced faster, earned more money, and were more likely to win or finish in the top 3 positions than horses that did not. The magnitude of the effect of furosemide on estimated 6-furlong race time varied with sex, with the greatest effect in males. When comparing horses of the same sex, horses receiving furosemide had an estimated 6-furlong race time that ranged from 0.56 +/- 0.04 seconds (least-squares mean +/- SE) to 1.09 +/- 0.07 seconds less than that for horses not receiving furosemide, a difference equivalent to 3 to 5.5 lengths.

CONCLUSIONS AND CLINICAL RELEVANCE: Because of the pervasive use of furosemide and its apparent association with superior performance in Thoroughbred racehorses, further consideration of the use of furosemide and investigation of its effects in horses is warranted.

PMID: 10476714 [PubMed - indexed for MEDLINE]
Furosemide reduces accumulated oxygen deficit in horses during brief intense exertion

K. W. Hinchcliff,
K. H. McKeever,
W. W. Muir, and
R. A. Sams

Abstract


Furosemide reduces accumulated oxygen deficit in horses during brief intense exertion. J. Appl. Physiol. 81(4): 1550–1554, 1996.—We theorized that furosemide-induced weight reduction would reduce the contribution of anaerobic metabolism to energy expenditure of horses during intense exertion. The effects of furosemide on accumulated O2 deficit and plasma lactate concentration of horses during high-intensity exercise were examined in a three-way balance randomized crossover study. Nine horses completed each of three trials: 1) a control (C) trial, 2) a furosemide-unloaded (FU) trial in which the horse received furosemide 4 h before running, and 3) a furosemide weight-loaded (FL) trial during which the horse received furosemide and carried weight equal to the weight lost after furosemide administration. Horses ran for 2 min at ~120% maximal O2 consumption. Furosemide (FU) increased O2 consumption (ml · 2 min−1 · kg−1) compared with C (268 ± 9 and 257 ± 9, P < 0.05), whereas FL was not different from C (252 ± 8). Accumulated O2 deficit (ml O2 equivalents/kg) was significantly (P < 0.05) lower during FU (81.2 ± 12.5), but not during FL (96.9 ± 12.4), than during C (91.4 ± 11.5). Rate of increase in blood lactate concentration (mmol · 2 min−1 · kg−1) after FU (0.058 ± 0.001), but not after FL (0.061 ± 0.001), was significantly (P < 0.05) lower than after C (0.061 ± 0.001). Furosemide decreased the accumulated O2 deficit and rate of increase in blood lactate concentration of horses during brief high-intensity exertion. The reduction in accumulated O2 deficit in FU-treated horses was attributable to an increase in the mass-specific rate of O2 consumption during the high-intensity exercise test.

Footnotes

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This study was supported by a grant from the Grayson-Jockey Club Research Foundation. Present address of K. H. McKeever: Dept. of Animal Science, Cook College, Rutgers University, Piscataway, NJ 08855.
Furosemide-induced changes in plasma and blood volume of horses

K. W. HINCHCLIFF, K. H. McKEEVER, W. W. MUIR III

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Abstract

The effect of furosemide administration (1mg/kg body weight, i.v.) on plasma and blood volumes in 6 intact and 4 splenectomized horses was measured using Evans blue dye dilution, hematocrit, and hemoglobin and plasma total solids concentrations. Body weight decreased by 33.6±3.3 and 33.7±0.8g/kg 4h after furosemide administration to intact and splenectomized mares, respectively. Plasma volume, estimated by Evans blue dye dilution, was reduced by 8.3±3.3% (mean±SE) 4h after furosemide administration. The reduction in plasma volume was first detectable 5-10 min after furosemide administration and was greatest 15-30 min (13.0±0.8%) after dosing. This study demonstrates that furosemide produces significant and rapid reductions in plasma volume in horses. These decreases in plasma volume only partially resolve 4h after furosemide administration.
IV

Effects of dehydration on thermoregulatory responses of horses during low-intensity exercise

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Abstract
Effects of dehydration on thermoregulatory and metabolic responses were studied in six horses during 40 min of exercise eliciting approximately 40% of maximal O2 consumption and for 30 min after exercise. Horses were exercised while euhydrated (C), 4 h after administration of furosemide (FDH; 1.0 mg/kg i.v.) to induce isotonic dehydration, and after 30 h without water (DDH) to induce hypertonic dehydration. Cardiac output was significantly lower in FDH (144.1 +/- 8.0 l/min) and in DDH (156.6 +/- 6.9 l/min) than in C (173.1 +/- 6.2 l/min) after 30 min of exercise. When DDH, FDH, and C values were compared, dehydration resulted in higher temperatures in the middle gluteal muscle (41.9 +/- 0.3, 41.1 +/- 0.2, and 40.6 +/- 0.2 degrees C, respectively) and pulmonary artery (40.8 +/- 0.3, 40.1 +/- 0.2, and 39.7 +/- 0.2 degrees C, respectively). Temperatures in the superficial thoracic vein and subcutaneous sites on the neck and back and peak sweating rates on the neck and back were not significantly different in DDH and C. In view of higher core temperatures during exercise after dehydration and decrease in cardiac output without concomitant increases in peripheral temperatures or reduced sweating rates, we conclude that the impairment of thermoregulation was primarily due to decreased transfer of heat from core to periphery.

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Review of furosemide in horse racing: its effects and regulation†

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Abstract
Furosemide has been used empirically and has been legally approved for many years by the US racing industry for the control of exercise-induced pulmonary haemorrhage (EIPH) or bleeding. Its use in horses for this purpose is highly controversial and has been criticized by organizations outside and inside of the racing industry. This review concentrates on its renal and extra-renal actions and the possible relationship of these actions to the modification of EIPH and changes in performance of horses. The existing literature references suggest that furosemide has the potential of increasing performance in horses without significantly changing the bleeding status. The pulmonary capillary transmural pressure in the exercising horse is estimated to be over 100 mmHg. The pressure reduction produced by the administration of furosemide is not of sufficient magnitude to reduce transmural pressures within the capillaries to a level where pressures resulting in rupture of the capillaries, and thus haemorrhage, would be completely prevented. This is substantiated by
clinical observations that the administration of furosemide to horses with EIPH may reduce haemorrhage but does not completely stop it. The unanswered question is whether the improvement of racing times which have been shown in a number of studies are due to the reduction in bleeding or to other actions of furosemide. This review also discusses the difficulties encountered in furosemide regulation, in view of its diuretic actions and potential for the reduction in the ability of forensic laboratories to detect drugs and medications administered to a horse within days or hours before a race. Interactions between nonsteroidal anti-inflammatory drugs (NSAIDs) and furosemide have also been examined, and the results suggest that the effects of prior administration of NSAID may partially mitigate the renal and extra-renal effects which may contribute to the effects of furosemide on EIPH.
An unpublished pilot study was conducted to test the hypothesis that furosemide administration causes dehydration and increases the hematocrit in racehorses. This pilot study is being used for validation of my application to carry out a statistically meaningful test of this hypothesis and in the collection of other measurable physiological parameters involving more than a thousand horses under racing conditions in the USA and in multiple racing jurisdictions.

Author Sheila Lyons DVM, FACVSMR

Hemoconcentration and Oxygen Carrying Capacity Alteration in Race Horses Following Administration of Furosemide Prior to Speed Work

ABSTRACT: The measurement of packed red blood cell volume (PCV, Hct or hematocrit) and plasma osmolality immediately preceding and then four hours after intravenous administration of 250mg furosemide in 12 race horses was performed in order to assess the level of dehydration caused by this diuretic. The World Anti-Doping Agency (WADA) has established blood testing parameters for the indication of performance enhancement due to the artificially enhanced oxygen carrying capacity secondary to hemoconcentration in human athletes. Diuretics such as furosemide are banned by the WADA but artificial hemoconcentration has been achieved through the illegal use of EPO, the practice of blood doping, and other banned methods and practices. Since horse racing permits the use of furosemide, this pilot study was conducted to test the theory that the horse racing performance enhancement effect, which has been evidenced in the scientific literature for this drug, may be due to dehydration and improved oxygen carrying capacity achieved through hemoconcentration. The results were an increase in PCV of 6-18% with a nonlinear increase in plasma osmolality in each of the 12 horses tested in this pilot study. The WADA has established the hemoconcentration effect of EPO to be in the range of 6-11% which is considered performance enhancement in human athletics. Therefore, it appears through this pilot study that the administration of furosemide at the dosages used for horse racing supports a theory of performance enhancement through artificially enhanced oxygen carrying capacity due to hemoconcentration. A further study involving the testing of several thousand racehorses entered in races in multiple racing jurisdictions is planned by this investigator and warranted in the interest of fairness in horse racing.
Abstract
Blood doping has been achieved by either infusing red blood cells or by administering the drug erythropoietin to artificially increase red blood cell mass. Blood doping can improve an athlete's ability to perform submaximal and maximal endurance exercise. In addition, blood doping can help reduce physiologic strain during exercise in the heat and perhaps at altitude. Conversely, blood doping is associated with risks that can be serious and impair athletic performance. These known risks are amplified by improper medical controls, as well as the interaction between dehydration with exercise and environmental stress. Finally, the medical risks associated with blood doping have been estimated from carefully controlled research studies, and the medically unsupervised use of blood doping will increase these risks. It is the position of the American College of Sports Medicine that any blood doping procedure used in an attempt to improve athletic performance is unethical, unfair, and exposes the athlete to unwarranted and potentially serious health risks.
Abstract.

Background. Loop diuretics (LD) increase renal calcium excretion. Discrepant results on associations between LD and fracture risk have been reported.

Objective. To assess the fracture risk in users of LD.

Design and subjects. A population-based pharmaco-epidemiological case–control design with fracture in year 2000 as outcome and use of LD during the previous 5 years as exposure variable. We used nationwide computerized registers to assess individual use of LD and related these data to individual fracture data and information on potential confounders. We compared 64,699 cases aged 40 years or more who sustained a fracture during year 2000 with 194,111 age- and gender-matched controls.

Results. A total of 44,001 subjects used LD. Ever use of LD was associated with a crude 51% (OR 1.51; 95% CI 1.48–1.55) increased risk of any fracture and a 72% (OR 1.72; 95% CI 1.64–1.81) increased risk of hip fracture. The risk estimates were reduced after confounder adjustment, i.e. adjusted risk of any fracture was increased by 4% (OR 1.04; 95% CI 1.01–1.07) and risk of hip fracture by 16% (OR 1.16; 95% CI 1.10–1.23). In current users, a tendency towards a decreased fracture risk with increased dose was observed, whereas in former users risk of fracture increased with increased dose. Use of furosemide was associated with higher risk estimates than use of bumetanide.

Conclusion. Treatment with LD affects fracture risk. Special attention should be paid to patients in whom treatment with LD is initiated or stopped, as they may be at an increased risk of fracture.
What Causes Soft Palate Problems And Bleeding In Racehorses?

The answer is on the tip of the horse’s tongue

Robert Cook FRCVS, PhD

- In March 2011, the RCI (Association of Racing Commissioners International) urged U.S. racing’s administrators, within the next five years, to phase out bleeding medication on race days.
- In May 2011, Senator Udall introduced the Interstate Horseracing Improvement Act of 2011 in the Senate, “to end the use of performance-enhancing drugs in the sport of horse racing.”

The RCI informs racing’s administrators that it considers the use of bleeding medication to be unacceptable. I agree. Furthermore, it seems that if racing’s administrators do not ban race-day drugs, the government will intervene. Perhaps the threat of federal regulation will provide the necessary impetus to action. As Samuel Johnson observed, “When a man knows he is to be hanged in a fortnight, it concentrates his mind wonderfully.” Sadly, as judged by published reports from the summit meeting at Belmont Park on June 13 and 14th, 2011, only baby steps forward were taken.

Yet there is a simple solution. Most other countries have solved the problem - race-day drugs are banned. I have to wonder why racing’s administrators in the U.S. are so puzzled by the problem. Why, in this country, do they support a pharmaceutical approach to bleeding (Salix) when it is evident that the therapy fails to cure and, in the attempt, only succeeds in harming the horse and damaging the sport?

Our addiction to drugs for bleeding might be justifiable if the ‘treatment’ was rational and based, as all treatments should be, on removal of the cause. Failure to focus attention on the cause is, I believe, a source of confusion in our thinking about the problem. So-called exercise-induced pulmonary hemorrhage (EIPH) remains an unresolved problem because removal of the cause is a prerequisite of treatment and the only consensus in racing on this vital question is that the cause is supposedly unknown. As explained below, it is I believe no coincidence that dorsal displacement of the soft palate (DDSP) and EIPH, two serious problems of the racehorse are both common and of unknown cause. The two problems are related. Elevation of the soft palate, with or without DSSP, is the major cause of EIPH.

This article is offered in the hope of clarifying the question of cause and thereby emphasizing the need to discontinue ineffectual and unnecessary race-day drugs. Bleeding is a management problem. It requires a management not a pharmaceutical solution. This is something for racing’s administrators to solve, not racetrack veterinarians. In the U.S. discontinuation of race-day drugs is
the first priority. Worldwide, racing’s administrators should focus on the second priority – another management change. In my opinion, a rule update (approval of the crossunder bitless bridle for racing) would not only significantly reduce the prevalence of bleeding but would virtually eliminate dorsal displacement of the soft palate and many other disease and behavioural problems. At the same time it would reduce accidents and vastly improve the welfare of the horse. As though this was not sufficient inducement for change, performance would be improved. The bit is a handicap to performance. For both the athletes involved, racing would become healthier, happier and safer and – for other stakeholders - more pleasurable, honourable and profitable.

This opinion is given by a veterinary researcher who was the first to publish evidence indicating that racehorses with ‘nosebleeds’ were bleeding from the lungs (Cook 1974) and someone who has retained an intense interest in the cause of bleeding ever since. I realise that I am promoting a minority opinion on cause but, in my defence, science in general depends for progress on tenable minority opinions. Science advances by a process of disagreement. I am disagreeing with the status quo on cause.

There was much regret expressed, in the early reports from the Belmont Summit of the lack of consensus. But advance in science does not depend on a majority vote. It depends on evidence. All it needs is for racing’s administrators to consider the evidence. Let’s remember that the consistency of a hypothesis depends, not so much on the evidence to support it, but on the inability to refute it. A scientific hypothesis that cannot be refuted deserves to stand, at the present state of knowledge. Down the road, any hypothesis may have to be adjusted in the light of new evidence or even abandoned altogether. But until such time as conflicting evidence is discovered, an unrefuted hypothesis constitutes the best guide to action. This said, my working hypothesis is that pulmonary ‘bleeding’ in the racehorse is caused by any upper airway obstruction. I have tried diligently to refute this hypothesis and failed. As far as I am aware, so have others, as no publications have appeared to provide contrary evidence.

The syndrome that became known as EIPH has been written about extensively in the veterinary literature. Most of the research articles have focused on its supposed alleviation with a diuretic (Salix). Relatively little has been published on its cause. Two competing explanations have been proposed for the cause. Both focus on the air/blood barrier of the lung. The word ‘barrier’ in this context does not carry the meaning of ‘impassable.’ It refers to the infinitely delicate lining membrane of the lung’s air sacs that separate the dense network of small blood vessels in the lung from the air sacs of the lung. The membrane can be thought of as the lung’s highly specialised ‘skin’ exposed to the atmosphere. When the membrane works correctly (in health), it allows for the exchange of oxygen and carbon dioxide. Under normal conditions, it is thin enough to allow for gas exchange and thick enough to prevent the escape of fluids. The balance is critical.

I will call the two possible causes ‘A’ for air and ‘B’ for blood. ‘A’ stands for the pressure of air in the air sacs when the horse breathes in and ‘B’ for the pressure of blood in the capillaries. The ‘A’ supporters believe that ‘bleeding’ is caused by an abnormally low air pressure. The ‘B’ supporters believe that it is caused by an abnormally high blood pressure. ‘A’ implies too much suction force on the air side (the outside) and ‘B’ too much fluid force on the blood side (the inside). Though I promoted the ‘A’ explanation, the ‘B’ explanation has held sway over the years and been the most popular. The unsuccessful attempts to eliminate EIPH by reducing blood pressure are based on the B explanation.
Some ‘B’ supporters have suggested that high blood pressure during racing is an inherent part of the Thoroughbred’s make-up and that bleeding is inevitable or ‘normal.’ I find this unacceptable, because it is not consistent with equine physiology. Airways are for air, not blood. EIPH is not an accurate name for the syndrome. It is neither exclusively ‘exercise-induced’ nor a true ‘hemorrhage.’ So-called EIPH can occur in the stable when a non-exercising horse is accidentally asphyxiated. The fluid is not blood but edema fluid, albeit heavily blood-stained. Bleeding is not a problem exclusive to the discipline of racing and, in considering the cause, it is helpful to keep this in mind.

The ‘A’ explanation is consistent with the known facts about bleeding. An abnormally negative pressure in the small airways results from any obstruction of the upper airway, i.e., anywhere from nostril to first rib. A rational treatment based on this explanation requires removal of the airway obstruction. The benefits that such a step would bring racing are enormous. They extend far beyond ‘bleeding.’

Breathing is a suck/blow process. Air is sucked into the lungs during the negative pressure of inspiration as the diaphragm flattens. It is blown out again on expiration, under positive pressure, when the diaphragm relaxes. Too great a suction pressure when breathing-in affects the ‘skin’ of the horse’s lung in the same way as a ‘hickey’ on human skin. The only difference is that, in man, the effect is a subcutaneous bruise whereas in the horse, as the ‘skin’ of the air sac is fifteen times thinner than a sheet of airmail paper, red blood cells are sucked straight through the pores of the membrane, accompanied by edema fluid. The lung’s ‘skin’ leaks. It can be thought of as ‘sweating blood.’ This blood-stained fluid forms a puddle in the windpipe at the entrance to the chest. After a race, when the horse drops its head to drink, it may drain out at the nostrils and the horse appears to have had a nosebleed. In my opinion, the basic pathology of ‘bleeding’ in the Thoroughbred (or any other breed – for EIPH occurs in all disciplines) is water-logging of the lung (pulmonary edema). The cause is strangulation.

99% of racehorses ‘bleed’ when they run. Whatever causes EIPH has to be very common. Initially, I taught that paralysis or partial paralysis of the voice box (recurrent laryngeal neuropathy - RLN) was the major cause. Regretfully, RLN, commonly known as ‘roaring’ is common enough to be considered as the culprit. I still maintain that RLN plays a part, especially when the neuropathy is advanced. However, in the last 13 years I have discovered that there is an even more common and serious cause of upper airway obstruction. It has been staring man in the face for 5000 years. I refer to the horse’s bit. The good news is that whereas RLN is untreatable (and in my opinion inherited), the bit could be removed if the rules of racing were updated. After 5000 years of bit usage such a proposition will sound like heresy or even madness, “yet there is method in’t.” I repeat, bitless racing and training would be safer for horse and rider, accidents would be reduced, performance enhanced and the horse’s quality of life improved.

Some readers may question how a bit in the mouth could possibly obstruct the airway. It must be remembered that though the bit can only lie on the tip of the tongue which, together with the body of this large and muscular sense organ are indisputably in the mouth, the long root of the tongue lies in the throat. Indirectly, the bit grabs a horse in the throat. When a horse avoids the bit by withdrawing the tip of its tongue (a common evasion) the bulky root of the tongue bulges upwards in the throat. This in turn elevates the soft palate (which lies on the tongue’s root) and obstructs the airway. In England, trainers describe an attack of suffocation from such a cause as a horse
‘swallowing its tongue’ and ‘choking up.’ There are many other mechanisms whereby a bit obstructs the airway, including bit-induced poll flexion (see ‘Further Reading’).

I declare a conflict of interest but without apology. I know that by removing the bit, horsemen can do much for the horse, themselves and the reputation of racing. EIPH would not be entirely eliminated (as RLN and a few uncommon sources of airway obstruction would still occur) but I predict that its frequency would be significantly reduced. A significant bonus is that there would be a major reduction in the occurrence of dorsal displacement of the soft palate (DDSP) which, like the even more common elevation of the soft palate, is almost exclusively caused by the bit. DDSP and EIPH are both predominantly management problems. For the same reason, there would be a major reduction in the incidence of epiglottal entrapment. Until the bit is removed, we shall never know how much this might also reduce the occurrence of catastrophic musculo-skeletal accidents and breakdowns caused by bit-induced pathophysiology, pain and fatigue.

In closing, I make two recommendations. First, I propose that a more accurate name for EIPH would be ‘Negative Pressure Pulmonary Edema (NPPE).’ Secondly, I recommend that readers carry out an Internet search for a relatively uncommon but life-threatening disease in man by this name. Readers will find that so-called EIPH in the horse is analogous to NPPE in man. They will also be able to read many descriptions of the mechanism whereby this occurs. Though upper airway obstruction is the primary cause, a secondary effect of upper airway obstruction is to raise pulmonary blood pressure. ‘A’ and ‘B’ mechanisms are both involved.

Administrators of racing and other stakeholders will have much to think about as a result of the RCI press release. If I can help by answering questions I would be glad to do so in writing or by telephone.

Further reading: An asterisk after the reference indicates that the article is available online at www.bitlessbridle.com

14. Cook, W.R., “Another view on bleeding; asphyxia is suggested as the cause of pulmonary edema” *Thoroughbred Times*, p52, November 9, 1996
19. Cook, W.R.: "Asphyxia as the cause of bleeding and the bit as the cause of soft palate displacement" Guest Commentary *Thoroughbred Times*, November 27, 1999, pp18-19*
20. Cook, W.R.: Why Do Horses’ Lungs Bleed? Published online 1999*
23. Cook, W.R.: Not Snoring but Suffocating. Published online 2002*

An Endoscopic Test For Bit-Induced Nasopharyngeal Asphyxia As A Cause Of Exercise-Induced Pulmonary Haemorrhage In The Horse
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In their article on sudden death in racehorses, Lyle et al (2012) expressed the hope that their study would stimulate hypothesis-led investigations into possible causes.

Currently, there is no consensus on the first cause of EIPH. Even the mechanisms are debated. But the two leading mechanistic hypotheses are only at odds over the flimsiest of barriers - the pulmonary air/blood barrier. The majority opinion is that ‘bleeding’ occurs because of abnormally high capillary pressure on the blood side of the barrier. Let’s call this the blood pressure hypothesis. The minority opinion is that it occurs because of abnormally low negative pressure on the air side of the barrier - the air pressure hypothesis.

The majority maintain that high pulmonary pressure is an inherent characteristic of the Thoroughbred. In other words, that the first cause of EIPH is the Thoroughbred itself. But EIPH is not confined to the racing Thoroughbred. It also occurs in the racing Standardbred, Arabian and Quarter Horse. It is not even confined to racing as it also occurs in the hyperflexed dressage horse, in the draft horse with a paralyzed larynx and in a horse of any breed that gets cast in its stall with its head twisted. Long before I contracted a conflict of interest, my colleagues and I at Tufts concluded that asphyxia was a possible cause of EIPH (Cook et al 1988).

The ‘blood-pressure-in-the-racing-Thoroughbred’ hypothesis does not lend itself to testing nor to a solution by removal of the supposed first cause. Regrettably, it gives credence to the sad idea that ‘bleeding’ is incurable or even physiological and needs to be ‘managed’ with medication. Because the blood pressure hypothesis is invulnerable to refutation, I conclude that it is not a scientific hypothesis.

In contrast, the air pressure hypothesis is highly vulnerable and eminently refutable. In 1988, we listed a number of ways in which asphyxia could occur, naming recurrent laryngeal neuropathy as the most likely candidate. Since then, I have realized that though this is prevalent enough to match the prevalence of EIPH, the severity of the neuropathy in many cases is insufficient to entirely explain the problem. In the last 15 years, I have come to recognize that the ubiquitous bit is a much stronger candidate for causing asphyxia and that it brings this about by triggering instability and dorsal displacement of the soft palate (Cook 1999, 2002, 2005, 2013, Cook and Strasser 2003. At liberty, the running horse has a closed mouth, sealed lips and an immobile tongue and jaw. I now have evidence that its oral cavity and oropharynx are under negative pressure (Cook 2012, unpublished material). By breaking the lip seal, I believe that the bit triggers a cascade of problems from lip to lung. In anatomical if not physiological order these are - loss of the oral vacuum - dynamic collapse of the nasopharynx (soft palate instability and dorsal displacement) – obstruction of the choanae – gaping of the pharyngeal orifices to the guttural pouch - dynamic collapse of the larynx – dynamic
collapse of the dorsal membrane of the trachea with, over time, permanent distortion of the tracheal cartilages - and EIPH. Shakespeare’s phrase is apt, “The lie in the throat as deep as to the lungs.” Allen and Franklin (2012) report endoscopic observations, during moments of soft palate instability, consistent with loss of the oral vacuum, i.e., a flattened epiglottis and convexity in the most caudal section of the soft palate.

Further light on the effect of the bit is shed by Hong Kong statistics for 2004/2005 (Watkins et al 2008). During the training and racing of 1,358 Thoroughbreds the prevalence of “blood at one or both nostrils” was 5.74%. During swimming, when 1,155 of these same horses - on 150,000 occasions - were (presumably) wearing nothing but a halter, there was no ‘epistaxis.’

Exercise-induced pulmonary haemorrhage is not exclusively dependent on exercise and neither is it a true haemorrhage. So-called ‘epistaxis’ is not blood but edema fluid coloured with red blood corpuscles. A more precise and scientifically useful name would be negative pressure pulmonary edema (NPPE). An internet search reveals that this relatively uncommon but life-threatening emergency in man is analogous to the bizarrely common and potentially fatal EIPH in the racehorse, a subset of NPPE in the horse.

The literature on NPPE in man provides an explanation for the abnormally high pulmonary pressure in the horse. In a review of NPPE, Deepika et al (1997) state that the primary mechanism is upper airway obstruction. This generates a markedly negative intrapleural pressure transmitted to the pulmonary interstitium, an increased venous return to the right side of the heart, and a rise of pulmonary capillary pressure. The NPPE evidence in man blends the two competing hypotheses for EIPH in the racehorse, citing air pressure as the causal factor and blood pressure as a secondary effect. I submit that the same blend applies to the horse.

The technology of over-ground nasopharyngoscopy provides a way of putting this to the test. The null hypothesis could be tested that if a horse was first ridden in a bitted bridle there would be no improvement in the patency of the choanae and nasopharynx as judged by endoscopy when the same horse was ridden again under similar conditions in a bitless bridle. If improvement occurred the null hypothesis would be refuted and the air pressure hypothesis supported.

An addition to the standard endoscopy protocol will be necessary in order to evaluate the patency of (at least) one choana and the rostral two thirds of the nasopharynx. To evaluate these critical regions of the airway, a step that is currently omitted, the distal tip of the endoscope must be placed at the caudal end of the nasal cavity. By positioning the endoscope in the caudal half of the nasopharynx only, information on dorsal displacement of the soft palate and laryngeal problems is gathered but some information on rostral palatal elevation will be overlooked and choanal stenosis, a potentially catastrophic ball-valve obstruction due to the Bernoulli effect, can never be documented.

Science advances either by refutation of hypotheses or by the failure of determined efforts to refute them. Science does not advance anything like as convincingly by simply adding evidence in support of a hypothesis. As the air pressure hypothesis has not been refuted in 25 years it seems to have survivor fitness but this could be it has never been tested. But if the air pressure hypothesis survived attempts to refute it with over-ground endoscopy evidence this would assert its claim to be acknowledged and point to the bit as the major cause of NPPE in the horse. Further research into the effect of the bit on the horse is needed to ratify or refute the bit as the cause of NPPE.
Authors’ declaration of interests
Chairman and majority owner of Bitless Bridle Inc (www.bitlessbridle.com).

References
B. The Individual Fatalities

The following information was obtained from medical records, interviews with owners, trainers, jockeys, practicing veterinarians, NYRA veterinarians, race charts, race videos and a review of data provided by epidemiologists. As previously noted, Raw Moon and Unruly Storm were anomalies to the rest in that they did not experience musculoskeletal failures. In addition, the Task Force did a cumulative risk assessment for each horse based upon Dr. Parkin’s presentation at the 2011 Jockey Club Roundtable. Dr. Parkin, has identified eight different events or circumstances that are associated with increased risk of fatal musculoskeletal injury in the Thoroughbred racehorse. They are:

- A horse that has not started in a race in the last 15 to 30 days
- A horse that has made its first start in the last nine months, (i.e., a horse still in its first racing season)
- Intact male horses
- A horse that is older (three years of age and up)
- A horse that made its first start as a three year old or older
- A horse with numerous starts in the period between one and six months prior to the current race
- A horse racing at a distance of < seven furlongs
- A horse with a claiming price < $25,000

The Task Force considered these as potentially useful factors in analyzing each of the fatally injured horses and included them as a part of each case review. The Task Force wishes to emphasize that although it performed a risk profile for each individual horse as a part of its investigation, performing a risk profile using the above--- described factors is not, and should not be, a “stand--alone” procedure in the determination of whether or not a horse should race. A more detailed discussion of the EID is discussed in Section VIII (C), Equine Injury Database and Other Risk Factors. The Conclusion reached by the Task Force on each individual horse does not take into consideration other factors that may have contributed to these fatalities. These other possible contributing factors are discussed in Section VI, Other Potential Contributing Factors.
Speight of Hand 12/14/11 3rd Race

i. This intact male horse sustained a fatal injury in his 19th career start.

ii. He made six race starts in the preceding 12 months, and made no starts in the 30 days prior to the race in which the fatal injury was sustained.

iii. He had a pre-existing medical condition at the site that was subsequently injured.

iv. The fetlock was radiographed and injected with an intra-articular (hereinafter “IA”) corticosteroid seven days prior to the race. (The Task Force believes it is reasonable to accept this would not have occurred in a horse competing at this level. The horse racing industry ultimately exists by agreement of the public. They have the right to say no. The public has previously shown its willingness and resolve to exercise this right by banning dog racing in several states when similar concerns went unaddressed by that sport’s regulators. Horse racing may be next on the list if meaningful reforms and the demand for improved safety are not realized soon.

v. The IA injection was not noted in the horse’s medical record.

vi. The trainer did not report the IA injection to the Stewards as required by NYSRWB Rule 4043.2 (i).

vii. The following risk factors were present:

1) Racing for a claiming price \( \leq \$25,000 \)

2) Racing at a distance \( \leq \) seven furlongs

3) Intact male

4) Older horse (\( \geq \) three yrs)

viii. Speight of Hand was claimed three times during his career, twice in the preceding six months, with the last claim being made one month prior to injury. The claiming process transfers the horse, but not its medical records. It is unlikely that the trainer had knowledge of any medical treatments performed prior to his claiming the horse. This may have compromised the trainer’s ability to make informed decisions regarding medication administrations.

ix. The purse of the race in which this horse was injured was twice his claiming price (purse-to-claim ratio: 2.0).

x. The intervals between the horse’s last five races were 21, 51, 35, and 35 days respectively.

xi. Blood was collected and analyzed. A urine sample was not collected. There were no reported overages of therapeutic medications and no prohibited substances were detected, based upon the limited screening the testing laboratory was able to perform on the blood sample.6

xii. The absence of a complete necropsy precludes an understanding of Speight of Hand’s musculoskeletal health

**Conclusion:** Given the diagnostic workup and an IA corticosteroid injection of the left front fetlock seven days prior to the race, the Task Force questions whether Speight of Hand should have started. Based upon the information provided, the Task Force believes that it is likely that an opportunity may have been missed to prevent this injury. Specifically, the interval from treatment to race was insufficient to assess the horse’s response to treatment. Also, the pre-race examination findings were likely confounded by this treatment.

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1 As discussed more fully in Section VI(G)(4), the reduced volume of plasma obtained immediately post exercise from the fatally-injured horses limited the ability of the testing laboratory to screen for drugs other than non-steroidal anti-inflammatory drugs, corticosteroids, anabolic steroids and local anesthetics.
Dreamin of Silver 12/30/11 9th Race

i. This gelding sustained a fatal injury in his 3rd career start.

ii. He made three race starts in the preceding 12 months, with one start in the 30 days leading up to his final race.

iii. He had a pre-existing medical condition in the joint that was subsequently injured. This horse had a chip fracture arthroscopically removed from the right carpus on 6/22/11. This is an ethical and appropriate procedure that generally results in a good prognosis for a return to racing. Post-surgery, the horse was out of training 107 days. The horse then breezed eight times in the 49 days prior to his first start back.

iv. IA corticosteroid treatment was administered six days prior to the race.

v. This treatment was not reported to the Stewards as required by NYSRWB Rule 4043.2 (i)

vi. Pre-race exam findings record a minor change in the horse’s clinical presentation for the race in which it was injured. While this finding alone would not necessarily warrant a scratch, it would justify additional scrutiny. The IA injection six days prior to the race may have compromised the ability of the NYRA veterinarian to properly assess this horse’s condition on race day.

vii. He was injured in his 2nd race after a reduction in class.

viii. The intervals between his races were 15 and 20 days respectively.

ix. He was trained his entire career by the same trainer.

x. The following risk factors were present:

1) No race starts in the preceding 15---30 days
2) First start in the preceding nine months
3) Racing at a distance of ≤7 furlongs
4) Racing for a claiming price ≤ $25,000
5) Older horse (≥ three yrs)
6) The purse of the race in which he was injured was 1.8 times his claiming price (purse to claim price ratio: 1.8).

xi. The trainer was based at another location. Management decisions were reliant upon information provided by the assistant trainer and the attending veterinarian. It is not possible to know if management decisions regarding this horse would have been different had the trainer been on site.

xii. Blood was collected and analyzed. No urine sample was collected. There were no reported overages of therapeutic medications and no prohibited substances were detected, based upon the limited screening the testing laboratory was able to perform on the blood sample.

xiii. The absence of a complete necropsy precludes an understanding of this horse’s musculoskeletal health.

**Conclusion:** Based upon the information available, it is the opinion of the Task Force that the physical condition of Dreamin of Silver’s carpal joint prior to entry, requiring diagnostic and therapeutic intervention, raises the question whether this horse should have raced six days post treatment. The Task Force believes that it is likely that an opportunity may have been missed to prevent this injury.
Specifically, the interval from treatment to race was insufficient to assess the horse’s response to treatment. Also, the pre-race examination findings were likely confounded by this treatment.

**Inishmore 1/8/12 2nd Race**

i. This filly sustained a fatal injury in her 5th career start.

ii. She made five starts in the preceding 12 months but no starts in the 30 days prior to the race in which she was injured.

iii. Veterinary records indicate that she was treated with non-steroidal anti-inflammatory drugs (hereinafter “NSAIDs”) prior to breezing, a practice that may have reduced the trainer’s ability to accurately assess her condition and response to high speed exercise.

iv. There was a noteworthy change in her clinical presentation in the pre-race examination for the race in which she was subsequently injured. While this finding alone would not warrant a recommendation to scratch from the race, it would justify additional pre-race scrutiny. From the examination records provided, it is not known if that occurred.

v. She appeared to be traveling poorly from the time the gates opened.

vi. She made all her starts in maiden special weight races.

vii. The purse for the race was 41% higher than that for the corresponding race in the 2010-2011 meet.

viii. The following risk factors were present:

   1) First start in the preceding nine months
   2) Older horse (≥ three yrs)

ix. The intervals between her last five races were 23, 16, 14, and 35 days respectively.

x. No blood or urine samples were collected from this horse.

xi. The absence of a complete necropsy precludes an understanding of Inishmore’s musculoskeletal health.

**Conclusion:** A review of the race video indicated that Inishmore appeared to be traveling poorly from the start of the race and pre-race examination findings indicated a noteworthy change in this horse’s clinical presentation for the race in which she was subsequently injured. A follow-up interview with the jockey indicated that he recognized that the filly was unsound in the post parade, but did not report it to a racing official to initiate a scratch for fear of economic reprisal (manifested as lost riding opportunities from trainers). Despite his reservations about Inishmore’s soundness, the jockey rode her competitively during the race. The Task Force is troubled that a jockey persevered on a horse he believed to be unsound, risking himself and others on the racetrack. Based upon the information provided, the Task Force believes that this represented a missed opportunity to prevent this injury.

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2 A filly is a female horse less than five years of age.
Mannington 1/12/12 6th Race

i. This gelding sustained a fatal injury in his 48th career start.
ii. He made nine starts in the preceding 12 months and one start in the 30 days prior to the race in which he was injured.
iii. He was claimed twice in his career, once in the six months leading up to the final race.
iv. The intervals between his last five races were 25, 65, 30, and 34 days respectively.
v. The purse of the race in which he was injured was 1.3 times the claiming price (purse-to-claim ratio of 1.3)
vi. There was a pre-existing medical condition of the front fetlocks.
vii. The following risk factors were present:
   1) Numerous starts in the 1---6 month interval prior to the race
   2) No race starts in the preceding 15---30 days
   3) Racing at a distance of ≤7 furlongs
   4) Older horse (≥ three yrs)
viii. Physical exam findings were within a range consistent with racing soundness.
ix. No blood or urine samples were collected from this horse.
x. The absence of a complete necropsy precludes an understanding of Mannington’s musculoskeletal health.

Conclusion: Although there was a pre-existing medical condition in this horse, the Task Force does not have enough information to comment on its significance or potential relevance to the horse’s injury. It is not clear from the information available that there was an opportunity to prevent this injury.

Scorper 1/14/12 4th Race

i. This gelding sustained a fatal injury in his 28th career start.
ii. He made nine starts in the preceding 12 months and one start in the 30 days prior to the race in which he was injured.
iii. He was claimed three times in his career, but raced for the same connections for more than six months prior to the race in which he was injured.
iv. The intervals between his last five races were 35, 30, 28, and 28 days respectively.
v. The purse of the race in which he was injured was 3.6 times the claiming price (purse-to-claim ratio of 3.6)
vi. The following risk factors were present:
1) Numerous starts in the 1---6 month interval prior to the race
2) Racing at a distance ≤7 furlongs
3) Racing for a claiming price ≤ $25,000
4) Older horse (≥ three yrs)
   vii. There was a pre---existing medical condition in the front fetlocks.
   viii. There was a noteworthy change in the horse’s pre---race examination for the race in which
   he was subsequently injured. While this change alone would not necessarily warrant a scratch, it
   would justify additional pre---race scrutiny. From the information provided, it is not known if this
   occurred.
ix. No blood or urine samples were collected from this horse.
x. The absence of a complete necropsy precludes an understanding of Scorper’s
   musculoskeletal health.

**Conclusion:** Although there was a pre---existing medical condition, the Task Force does not have
enough information to comment on its significance or potential relevance to the injury. It is not
clear from the information available that there was an opportunity to prevent this injury. In the
opinion of the Task Force, the disproportionate purse for this race may have influenced the
management of this horse.

**Afleet Sue 1/15/12 9th Race**

i. This filly sustained a fatal injury in her first career start.
ii. She did not race as a 2---year---old.
iii. The purse for the race was 31% higher than for the corresponding race in the
    2010---2011 meet.
iv. The following risk factors were present:
   1) First start in preceding nine months
   2) Racing for a claiming price ≤ $25,000
   3) Racing at a distance ≤ seven furlongs
   4) First start made at 3yrs of age or older
   5) Older horse (≥ three yrs)

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3 It is frequently asserted that not racing a horse as a 2---year---old is protective against injury by
allowing the horse to “mature” before it races. Numerous scientific studies unequivocally
demonstrate the opposite to be true. Horses that raced as 2---year---olds were more likely to
perform better than those that did not (More, 1999). Moderate exercise used in race preparation
resulted in increased cannon bone density in trained horses when compared to untrained horses
(Boyde, 2005). Finally, horses that raced as 2---year---olds: 1) had significantly more race starts
than those first raced as 3---year---olds or older; 2) had significantly more years of racing; 3) were
more likely to have won or been placed in a race; and, 4) had greater total earnings than those that
first raced at a later age (Tanner, 2012).
The horse’s 2---year---old year represents a critical “window” for bone remodeling to occur.
During this interval, the horse must be trained with exercise of sufficient intensity and duration to
stimulate healthy bone conditioning without causing a fracture. However, it is important to
remember that each horse is an individual and training programs must be customized to the athletic
potential and relative maturity of each horse.
v. She had a pre-existing medical condition in her front fetlocks.
vi. Joints were injected IA with corticosteroids two weeks before the race.
vii. The IA injections were not reported to the Stewards as required by NYSRWB Rule 4043.2 (i).

viii. While pre-race physical exam findings are within the range consistent with racing soundness, one would anticipate a first time starter to have 'cleaner' legs than described in this record.

ix. Blood was collected and analyzed. No urine sample was collected. There were no reported overages of therapeutic medications and no prohibited substances detected, based upon the limited screening the testing laboratory was able to perform on the blood sample.

x. The absence of a complete necropsy precludes an understanding of Afleet Sue’s musculoskeletal health.

**Conclusion:** Although there was a pre-existing medical condition in this horse that required medical intervention two weeks prior to her only start, the Task Force does not have enough information to comment on its significance or potential relevance to the horse’s injury. Based upon the information provided, The Task Force cannot conclude that an opportunity was missed to prevent this injury.

**Raw Moon 1/15/12 9th Race**

i. This filly was an anomaly. She did not sustain an orthopedic injury.

ii. She had one start in the preceding 12 months and this was within 30 days of the race following which she died.

iii. The interval between her races was 28 days.

iv. This filly was reported to be healthy with no history of medical or musculoskeletal problems.

v. Both starts were made in Maiden Special Weight races.

vi. The purse for the race was 31% higher than that for the corresponding race in the 2010--2011 meet.

vii. The following risk factors were present:
   1) First start in the preceding nine months
   2) Racing at a distance of ≤ seven furlongs
   3) Older horse (≥ three yrs.)

viii. Physical exam findings were within a range consistent with racing soundness.

   There was no substantive change in the horse’s clinical presentation from her previous start.

ix. No blood or urine samples were collected from this horse.

x. The absence of a complete necropsy precludes an understanding of the filly’s musculoskeletal health or her cause of death.

**Conclusion:** This case was an anomaly in that Raw Moon did not sustain a musculoskeletal injury. In the absence of a complete necropsy, it is not possible to determine the cause of death. It is not clear from the information available that there was an opportunity to prevent this fatality.
Fortydeuce 2/2/12 5th Race

i. This intact male horse sustained a fatal injury in his 5th career start.

ii. He made four starts in the preceding 12 months and one start within 30 days of the race in which he was injured.

iii. He was never claimed and was trained by the same trainer his whole career.

iv. The intervals between his last five races were 40, 83, 48, and 21 days respectively.

v. The purse of the race in which he was injured was 2.1 times the claiming price (purse-to-claim ratio of 2.1) and reflected a 63% increase compared to the corresponding race during the 2010-2011 meet.

vi. The following risk factors were present:

1) First start in the preceding nine months
2) Racing for a claiming price ≤ $25,000
3) Intact male horse
4) Older horse (≥ three yrs)

vii. According to the trainer, this horse had a pre-existing hind limb gait abnormality and back pain.

viii. This horse had evidence of inflammation in both carpi (one of which was associated with the fatal injury)

ix. Pre-race physical exam findings were within a range consistent with racing soundness. No substantive change was noted in the horse’s clinical presentation from previous starts.

x. He appeared to be traveling poorly from the time the gates opened.

xi. Blood was collected and analyzed. No urine sample was collected. There were no reported overages of therapeutic medications and no prohibited substances were detected, based upon the limited screening the testing laboratory was able to perform on the blood sample.

xii. The absence of a complete necropsy precludes an understanding of Fortydeuce’s musculoskeletal health.

Conclusion: In a review of the race video, it appeared that the horse was traveling poorly from the start. Although the rider did not acknowledge reservations about this horse’s soundness, he appeared to be riding very cautiously. After finishing a credible second in his last race for $20,000, he was dropped in class to $12,500. The trainer’s decision to enter the horse at this lower level suggested a lack of confidence in his horse’s durability and a disinclination to commit to this horse long-term. The Task Force that the connections’ intentions were to lose Forty Deuce in the claiming box sooner believes rather than later. Based upon the information provided, The Task Force believes that an opportunity may have been missed to prevent this injury. Specifically, this horse should not have raced.
Sheeds Paisley 2/3/12 9th Race

i. This filly sustained a fatal injury in her 4th career start.
ii. She made three starts in the preceding 12 months and two starts in the 30 days prior to the race in which she was injured.
iii. She made her first start late in her three year old year.
iv. She was never claimed and was trained by the same trainer for her entire career.
v. She routinely trained on medications in the month prior to her injury that may have reduced the ability of the trainer to accurately assess her condition and her response to high-speed exercise.
vi. The intervals between her last four races were 31, 9, and 12 days respectively.
vii. The purse value was twice the claiming price for which this horse was entered (purse-to-claim ratio: 2.0).
viii. The following risk factors were present:
1) First start in the preceding nine months
2) Racing for a claiming price ≤ $25,000
3) First start made at three years of age or older
4) Older horse (≥ three yrs)
ix. Physical exam findings were within a range consistent with racing soundness.
   There was no substantive change in the horse’s clinical presentation from previous starts.
x. No blood or urine samples were collected from this horse.
xi. The absence of a complete necropsy precludes an understanding of the horse’s musculoskeletal health.

Conclusion: This filly was trained by a well-intentioned trainer who was unaware of management practices identified as having a protective effect against orthopedic injury. Believing it in her best interest, he elected not to race this filly until she was almost four years old, which, in fact, likely increased her risk of catastrophic musculoskeletal injury. She trained on medication in the month prior to injury. This practice may have reduced the ability of the trainer to accurately assess the condition of his horse and her response to high-speed exercise. Based upon the information provided, The Task Force believes that these circumstances represented missed opportunities that could have possibly prevented this injury.

Skorton 2/5/12 4th Race

i. This gelding sustained a fatal injury in his 10th career start.
ii. He made nine starts in the preceding 12 months; and none of these starts occurred within 30 days of the race in which he was injured.
iii. He was never claimed and was trained by same trainer for his entire career.
iv. The intervals between his last five races were 14, 19, 15, and 35 days respectively.
v. The following risk factors were present:
1) Numerous starts in the 1-6 month interval prior to the race
2) First start in the preceding nine months
3) Racing at a distance ≤ seven furlongs
4) Racing for a claiming price ≤ $25,000
5) Older horse (≥ three yrs)

vi. The purse value was 1.8 times the claiming price for this horse (purse to claim price ratio of 1.8).
vii. He trained on medications that may have reduced the ability of the trainer to accurately assess the condition of the horse and his response to high-speed exercise.
viii. Pre-race exam findings record a change in the horse’s clinical presentation for the race in which he was injured. While this finding alone would not necessarily warrant a scratch, it would justify additional pre-race scrutiny.
ix. He appeared to travel poorly from the start of the race and sustained his injury after running less than ¼ mile.
x. No blood or urine samples were collected from this horse.
xii. The absence of a complete necropsy precludes an understanding of Skorton’s musculoskeletal health.

Conclusion: A review of the race video indicated that Skorton broke well from the gate, but appeared unsound early in the race, well before he was under racing pressure or urging. He trained on medication in the month prior to injury. This practice may have reduced the ability of the trainer to accurately assess the condition of his horse or the horse’s response to high-speed exercise. The Task Force believes, from the information available and its review of the race replay that the horse’s soundness was suspect because he appeared unsound early in the race and before he was placed under stress. Skorton should not have participated in the race.

Unruly Storm 2/17/12 5th Race

i. This mare was an anomaly. She experienced a racing accident, not a musculoskeletal failure.
ii. She sustained a laceration to her left front leg that subsequently became infected.
   She was euthanized at a referral hospital as a result of this infection.
iii. This horse’s injury was sustained in her 31st career start and in her first race since being claimed by the current trainer. She was claimed three times during her racing career.
iv. This horse made 17 starts in the preceding 12 months, none of which were within 30 days of the race in which she was injured.
v. The intervals between her last five races were 11, 13, 11, and 32 days respectively.

4 A mare is a female horse five years of age or older.
vi. The purse value was three times her claiming price (purse to claim price ratio of 3.0).
vii. The following risk factors were present:
1) Racing at a distance ≤ seven furlongs
2) Racing for a claiming price ≤ $25,000
3) Older horse (≥ three yrs)
viii. Physical exam findings were within a range consistent with racing soundness. 
     There was no substantive change in her clinical presentation from previous
     starts.
ix. No blood or urine samples were collected from this horse.

x. The absence of a complete necropsy precludes an understanding of Unruly
    Storm’s musculoskeletal health.

Conclusion: Unruly Storm experienced a racing accident and not a musculoskeletal failure.
Based on the information available, the Task Force does not believe that there was an
opportunity to prevent this injury.

Coronado Heights 2/25/12 10th Race

i. This gelding sustained a fatal injury in his 3rd career start.
ii. He made two starts in the preceding 12 months, one of which was made within 30 days of the
    race in which he was injured.
iii. The intervals between his three lifetime races were 21 and 23 days respectively.
iv. He was never claimed and raced for the same trainer throughout his brief racing career.
v. He made his first start in January of his four-year-old year.
vi. There were substantial gaps in his published preparatory works in the fall of 2011.
vii. The trainer reported that the pre---race medication program for this horse was standard
    practice for all of the horses in his stable.
viii. The purse value was 3.3 times his claiming price (purse to claim price ratio of 3.3).
ix. The following risk factors were present:
    1) No starts in preceding 15---30 days
    2) First start in the previous nine months
    3) Racing at a distance ≤ seven furlongs
    4) Racing for a claiming price ≤ $25,000
    5) First start made at three years of age or older
    6) Older horse (≥ three years)
x. His stifles received an IA injection of hyaluronic acid and Depo---Medrol®, five days before
    the race.
xii. This treatment was not reported to the Stewards as required by NYSRWB Rule 4043.2(i).
This horse was routinely treated pre-race with two NSAIDs, as well as Legend® and Adequan®\textsuperscript{10}. These latter two therapeutic medications are commonly used to protect the joints of horses. The concurrent administration of NSAIDs is controversial because of potentially harmful side effects.

Pre-race examination findings indicated a subtle gait abnormality noted consistently over time.

He sustained his injury early in the race, after running approximately one furlong.

No blood or urine samples were collected from this horse.

The absence of a complete necropsy precludes an understanding of Coronado Heights’ musculoskeletal health.

**Conclusion:** The trainer reported that there were a number of minor problems that kept this horse from racing until his 4-year-old year. However, because he sustained his injury early in the race, the Task Force believes this horse’s musculoskeletal system was suspect prior to the race. The aggressive pre-race medication protocol in the days leading up to his final race may have masked clinical signs of lameness and confounded the pre-race examination. Based upon the information provided, The Task Force believes this medication practice may have represented a missed opportunity to prevent this injury.

**Tiz a Lil Meatball 2/26/12 3rd Race**

i. This gelding sustained a fatal injury in his 8th career start.

ii. He made seven starts in his racing career, but none within 30 days of the race in which he was injured.

iii. He was claimed twice in his racing career, once at 30 days prior to the race in which he was injured.

iv. He sustained his fatal injury in his first start after being claimed. It is unlikely that the trainer was aware of any medical treatments that may have occurred prior to his claiming the horse. This may have compromised the trainer’s ability to make informed decisions with regard to medication administrations.

v. His stifles and hocks were treated with IA corticosteroid injections after the claim.

vi. This treatment was not reported to the Stewards as required by NYSRWB Rule 4043.2 (i)

vii. The intervals between his last five races were 27, 27, 28, and 30 days respectively.

viii. The purse value was 1.9 times his claiming price (purse-to-claim ratio of 1.9).

ix. The following risk factors were present:

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\textsuperscript{10}Legend® is a form of hyaluronic acid, a normal constituent of joint fluid. Adequan® is a form of glycosaminoglycan, a normal constituent of articular cartilage. Both of these medications promote joint health and were administered in accordance with NYSRWB Rule 4038.5, which prohibits administration of these medications within 48 hours of racing.
1) Numerous starts in the one---six month interval prior to the race
2) No starts in the last 15---30 days
3) First start in the preceding nine months
4) Racing at a distance of ≤ seven furlongs
5) Racing for a claiming price ≤ $25,000
6) Presence of a suspensory ligament abnormality
7) Older horse (≥ three yrs)

x. Physical exam findings, including a gait abnormality, were consistent over time.

xi. Blood was collected and analyzed. No urine sample was collected. There were no reported overages of therapeutic medications and no prohibited substances detected, based upon the limited screening the testing laboratory was able to perform on the blood sample.

xii. The absence of a complete necropsy precludes an understanding of Tiz a Lil Meatball’s musculoskeletal health.

Conclusion: Even in the presence of multiple risk factors, the pre---race examination was unremarkable and a reasonable determination of racing soundness was made. Tiz a Lil Meatball was claimed one month prior to his last race. The claiming trainer had no knowledge of the horse’s medical treatment history, if any, prior to the claim. Based on the information available, the Task Force is not able to speculate on the cause of Tiz a Lil Meatball’s injury nor is it clear that there was an opportunity to prevent this fatality.

Bernie’s Love 3/2/12 2nd Race

i. This colt sustained a fatal injury in his 4th career start.
ii. He made three starts in the preceding 12 months, one of which occurred within 30 days of the race in which he was injured.
iii. He made his first start in December of his three---year---old year.
iv. He made all of his starts in maiden special weight races, and never started for a claiming price.
v. The purse for the race was 43% higher than that for the corresponding race in the 2010---2011 meet.
vi. The intervals between his races were 22, 29 and 27 days respectively.
vii. The following risk factors were present:
1) No starts in the previous 15---30 days
2) First start in the preceding nine months
3) Racing at a distance ≤ seven furlongs
4) Racing for a claiming price ≤ $25,000
5) Intact male horse
6) First start made at three years of age or older
7) Older horse (≥ three yrs)
viii. Pre-race exam findings record a minor change in the horse’s clinical presentation for the race in which he was injured. While this finding alone would not necessarily warrant a scratch, it would justify additional pre-race scrutiny.

ix. No blood or urine samples were collected from this horse.
x. The absence of a complete necropsy precludes an understanding of Bernie’s Love’s musculoskeletal health.

**Conclusion:** The Task Force does not have enough information to speculate on the cause of Bernie’s Love’s fatal injury, nor could it conclude that an opportunity may have been missed to prevent this injury. Although there was a minor change in this horse’s condition noted during the pre-race examination, the Task Force does not have enough information to comment on its significance or potential relevance to the injury. It is not clear from the information available that there was an opportunity to prevent this injury.

**Wes Vegas 3/3/12 2nd Race**

i. This gelding sustained an injury in his 1st career start.

ii. Treatment records indicate he received NSAIDs prior to and after high-speed exercise. This practice may have reduced the trainer’s ability to accurately assess this horse’s condition and response to high-speed exercise.

iii. The purse value was 2.6 times his claiming price (purse-to-claim ratio of 2.6).

iv. The following risk factors were present:
1) First start in the preceding nine months
2) No starts in the previous 15 to 30 days
3) Racing at a distance ≤ seven furlongs
4) Racing for a claiming price ≤ $25,000
5) First start made at three years of age or older
6) Older horse (≥ three yrs)

v. While physical exam findings were within the range consistent with racing soundness, one would anticipate a first-time starter to have ‘cleaner’ legs than described in this record.

vi. Blood was collected and analyzed. No urine sample was collected. There were no reported overages of therapeutic medications and no prohibited substances were detected, based upon the limited screening the testing laboratory was able to perform on the blood sample.

vii. The absence of a complete necropsy precludes an understanding of Wes Vegas’ musculoskeletal health.

**Conclusion:** A review of the race video indicated that the jockey was riding confidently and Wes Vegas appeared to be travelling normally until the moment he sustained his injury. This horse was treated with NSAIDs both before and after breezing. This medication protocol caused the Task Force to question the horse’s soundness leading up to the race. Further, this aggressive medication protocol in
the month prior to injury may have reduced the ability of the trainer to accurately assess the condition of this horse. Based upon the information provided, The Task Force believes this medication practice may have represented a missed opportunity to prevent this injury.

**Almighty Silver 3/4/12 3rd Race**

i. This gelding sustained a fatal injury in his 44th career start.

ii. He made 12 starts in the preceding 12 months, but did not start within 30 days of the race in which he was injured.

iii. He was claimed five times during his racing career. He was claimed three times in the three months leading up to his final race.

iv. The intervals between his last five races were 13, 35, 8, and 45 days respectively. The trainer reported the horse was in poor condition when claimed and it took seven weeks to return him to good health.

v. The purse value was 5.3 times his claiming price (purse-to-claim ratio of 5.3).

vi. Four IA injections were performed five days prior to the race. This treatment was not reported to the Stewards as required by NYSRWB Rule 4043.2 (i).

vii. The following risk factors were present:

1) Numerous starts in the one & six month interval

2) No starts in the previous 15-30 days

3) Racing for a claiming price ≤ $25,000

4) Older horse (≥ three yrs)

viii. Pre-race physical exam findings were within a range consistent with racing soundness. There was no substantive change in the horse's clinical presentation from previous starts.

ix. Blood was collected and analyzed. No urine sample was collected. There were no reported overages of therapeutic medications and no prohibited substances were detected, based upon the limited screening the testing laboratory was able to perform on the blood sample.

x. The absence of a complete necropsy precludes an understanding of Almighty Silver's musculoskeletal health.

**Conclusion:** Almighty Silver was claimed three times in the three months leading up to his final race. The lack of transfer of medical history became increasingly problematic with each claim. While in some cases the Task Force noted that the augmented purse might have incentivized poor decisions, in this case the availability of augmented purses may have created a situation in which the trainer could afford to invest seven weeks of care and training in a lower level claiming horse before running him back. The use of NSAID’s in training may have confounded an accurate assessment of his soundness. Based upon the information provided, it is the opinion of the Task Force that the physical condition of Almighty Silver, requiring therapeutic intervention five days prior to racing, raises the question whether this horse should have raced. It is likely that an opportunity may have been missed to prevent this injury. Specifically,
interval from treatment to race was insufficient to assess the horse’s response to treatment. Also, this treatment may have confounded the pre---race examination.

**Big Polka Dot 3/2/12 3rd Race**

i. This gelding sustained a fatal injury in his 23rd career start.
ii. He made 13 starts in the preceding 12 months, one of which was in the 30 days prior to the race in which he was injured.
iii. He was claimed three times during his racing career, but not within six months of his last race.
iv. The intervals between his last five races were 22, 63, 7, and 30 days respectively.
v. The purse value was 5.3 times his claiming price (purse---to---claim ratio of 5.3).
vi. The following risk factors were present:
   1) Numerous starts in the 1---6 month interval prior to the race
   2) Racing for a claiming price ≤ $25,000
   3) Older horse (≥ three yrs)
   vii. He received two DepoMedrol® (methylprednisolone acetate) IA injections in the 30 days prior to the race in which he was injured. These treatments were not reported to the Stewards as required by NYSRWB Rule 4043.2(i). Additionally, he received orally administered corticosteroids for four days preceding the race.
   viii. Physical exam findings were within a range consistent with racing soundness.

   *There was no substantive change in his clinical presentation from previous starts. However, the administration of corticosteroids may have compromised the NYRA veterinarian’s ability to accurately assess his soundness.*
ix. No blood or urine samples were collected from this horse.
x. The absence of a complete necropsy precludes an understanding of Big Polka Dot’s musculoskeletal health.

**Conclusion:** The Task Force questions the musculoskeletal soundness of Big Polka Dot prior to entry in his last race. The repeated administrations of long---acting corticosteroids likely confounded the pre---race examination. The protocol of multiple intra---articular injections in a brief time frame is understood to be a technique used to keep an unsound horse functional rather than a judicious therapeutic administration to facilitate recovery from injury. Based upon the information provided, The Task Force believes that this medication protocol represented an opportunity that was missed to prevent this injury.

**Hubbard 3/8/12 8th Race**

i. This gelding sustained a fatal injury in his 41st career start.
ii. He made 10 starts in the preceding 12 months; one start occurred within 30 days of the race in which he was injured.
iii. At the instruction of the owner, he received no pre---race medication.
iv. The trainer reported that this horse had a ‘funny way of going’, but gave no indication the horse had undergone a soundness evaluation by a veterinarian.
v. He was not claimed during his racing career.
vi. The intervals between his last five races were 14, 11, 26, and 19 days respectively.
vii. The purse value was 2.3 times his claiming price (purse-to-claim ratio of 2.3).
viii. The following risk factors were present:
1) Numerous starts in the 1-6 month interval prior to the race
2) No starts in the preceding 15-30 days
3) Racing for a claiming price ≤ $25,000
4) Older horse (≥ three yrs.)
ix. Pre-race exam findings recorded a minor change in his clinical presentation for the race in which he was injured. While this finding alone would not necessarily warrant a scratch, it would justify additional scrutiny.
x. No blood or urine samples were collected from this horse.
xi. The absence of a complete necropsy precludes an understanding of Hubbard’s musculoskeletal health.

Conclusion: While Hubbard was participating in claiming races, it did not appear to the Task Force that there was any desire on the part of the horse’s connections for this horse to be claimed. With the limited information available, including a lack of a complete necropsy, the Task Force has no opinion as to the cause of Hubbard’s injury nor could the Task Force conclude that an opportunity may have been missed to prevent this injury.

Hillsboro Bay 3/14/12 9th Race

i. This filly sustained a fatal injury in her 9th career start.
ii. She made seven starts in the preceding 12 months, but did not start within 30 days of the race in which she was injured.
iii. She was claimed twice in her racing career, once in the six months leading up to her final race.
iv. The intervals between her last five races were 17, 23, 30, and 42 days respectively.
v. The purse value was 2.2 times her claiming price (purse-to-claim ratio of 2.2).
vi. The following risk factors were present:
1) Numerous starts in the 1-6 month interval prior to the race
2) Racing at a distance ≤ seven furlongs
3) Racing for a claiming price ≤ $25,000
4) Older horse (≥ three yrs.)
vii. The medications Banamine® (flunixin) and compounded naquasone (trichlormethiazide and dexamethasone) were dispensed to the trainer two weeks prior to the last race. Medical records did not provide justification or identify the condition requiring such treatment.
viii. Physical exam findings were within a range consistent with racing soundness. No substantive change in this filly’s clinical presentation from previous starts was noted.

ix. Blood was collected and analyzed. No urine sample was collected. There were no reported overages of therapeutic medications and no prohibited substances were detected, based upon the limited screening the testing laboratory was able to perform on the blood sample.

x. The absence of a complete necropsy precludes an understanding of Hillsboro Bay’s musculoskeletal health.

**Conclusion:** Based on the information available, the Task Force has no opinion as to the cause of Hillsboro’s Bay’s injury, nor could the Task Force conclude that there was a missed opportunity to prevent this injury.

**Deferred Risk 3/17/12 7th Race**

i. This filly sustained a fatal injury in her 1st career start.

ii. The purse value was 1.4 times her claiming price (purse-to-claim ratio of 1.4).

iii. The following risk factors were present:

1) No starts in the preceding 15-30 days
2) First start in nine months
3) Racing at a distance ≤ seven furlongs
4) Racing for a claiming price ≤ $25,000
5) Older horse (≥ three yrs.)

iv. Pre-race physical exam findings were within a range consistent with racing soundness.

v. The jockey felt that this filly was unsound during the warm-up.

vi. No blood and urine samples were collected from this filly.

vii. The absence of a complete necropsy precludes an understanding of this Deferred Risk’s musculoskeletal health.

**Conclusion:** Despite the fact that the jockey indicated Deferred Risk was unsound during warm-up, he did not approach a racing official to initiate a scratch for fear of economic reprisal (manifested as lost riding opportunities from trainers). Nonetheless, the jockey rode her competitively during the race. The Task Force is troubled that a jockey persevered on a horse he believed to be unsound, risking himself and others on the racetrack. Based upon the information provided, the Task Force believes that an opportunity to prevent this injury may have been missed.

**Smartie Bobbi 3/18/12 7th Race**

i. This filly was fatally injured in her 19th career start.
ii. She raced 10 times in the past 12 months, one of which was within 30 days of her final race.

iii. She was claimed twice during her racing career, but not in the six months leading up to her final race.

iv. The intervals between her last five races were 16, 168, 14, and 21 days respectively.

v. The purse value was 3.6 times her claiming price (purse-to-claim price ratio of 3.6).

vi. The following risk factors were present:
1) Racing at a distance ≤ seven furlongs
2) Racing in claiming races ≤$25,000
3) Older horse (≥ three yrs)

vii. Pre-race exam findings record a change in this filly’s clinical presentation for the race in which she was injured. While this finding alone would not necessarily warrant a scratch, it would justify additional scrutiny.

viii. Blood was collected and analyzed. No urine sample was collected. There were no reported overages of therapeutic medications and no prohibited substances were detected, based upon the limited screening the testing laboratory was able to perform on the blood sample.

ix. The absence of a complete necropsy precludes an understanding of Smartie Bobbi’s musculoskeletal health.

**Conclusion:** With the limited information available, including the lack of a complete necropsy, the Task Force has no opinion as to the cause of Smartie Bobbi’s injury. However, the change in clinical presentation noted during the pre-race examination is suggestive of a pre-existing condition.