The golden hours in equine emergency rescue.

[1]Tomas Gimenez, Dr. *med vet*

Introduction

For a number of different reasons, it is not uncommon for large animals involved in entrapment incidents to be rescued several hours after their occurrence. The rescue may take place efficiently and safely, and the victim may not appear to have any life threatening issues. However, several hours or within a few days after the rescue the victim's condition may deteriorate, and in some cases die.

A common denominator in horses that are recumbent during entrapment, is the abnormal compression of muscle tissue for prolonged periods of time resulting in decreased or lack of blood supply (ischemia). Damage to tissues devoid of blood supply due to pressure/compression was attributed to the lack of blood supply itself. Beginning in the early 1980's, however, it was demonstrated that injuries occurring in the ischemic tissue and distant organs were caused by tissue breakdown products *after* the reestablishment of blood flow known as reperfusion injury (Bulkley 1987)

The purpose of the present review is to provide evidence that the combination of abnormal pressure and a prolonged (>4-6 hours) period, can be the cause of localized and distant organ injury or death after rescue in the horse.

The information presented includes studies performed in the horse, human, dog, pig, and rabbit.

Normal recumbency in the horse

Depending on a number of variables (light, noise, smell, surface, etc), horse's sleep can be classified as half-sleep, light, medium or deep. During deep sleep the horse will assume sternal or lateral, left or right, recumbency. Average periods of lateral recumbency are 23 minutes, with the longest being 1 hour (Steinhart 1937, Littlejohn and Munro 1972,). In a study utilizing 600 horses, Steinhart (1937) observed that when assuming recumbency 85% of the horses adopted lateral recumbency of which 65% was right lateral recumbency. Except when rolling, horses do not assume dorsal (lying on the back) recumbency.

[1] Technical Large Animal Emergency Rescue, TLAER™
www.tlaer.org, tlaer@bellsouth.net

The reperfusion syndrome

The combination of gravity in the form of pressure/compression, and prolonged time, has a negative effect on blood supply on body tissues. From the tissues present in a leg, muscle is the most susceptible to ischemia (Blaisdell 2002)

Reestablishment of blood flow to previously ischemic organs or tissues initiates a damaging cascade of events in both the reperfused tissue and distant organs known as the reperfusion syndrome as illustrated in figure1.

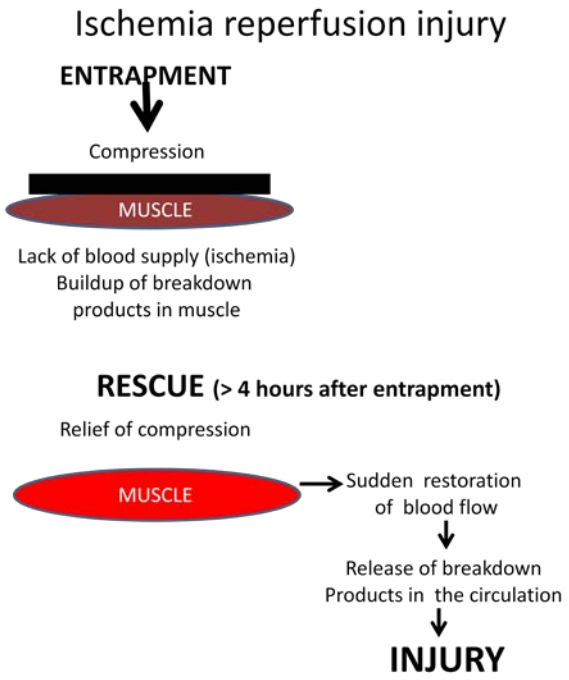
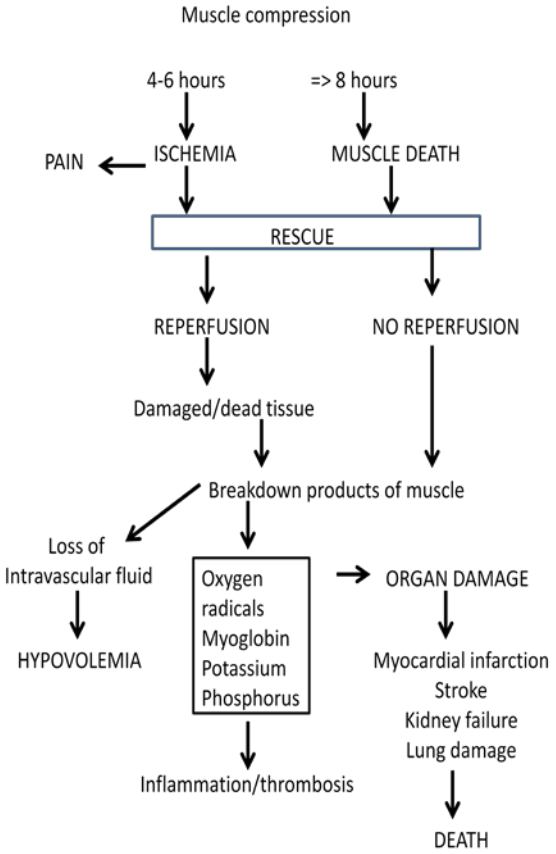


Figure 1. Compression of muscle tissue for a prolonged period of time results in the accumulation of breakdown products which in turn cause damage to the muscle itself and distant organs.

In humans, muscle is tolerant to ischemia up to 4 hours, nerve changes are reversible up to 8 hours, fat up to 13 hours, and skin up to 24 hours (Steinau 1988). In horses, decreased circulation and muscle inflammation (myositis) has been detected after 2 hours of recumbency. Once muscle compression is released, it is followed by massive hyperemia (Sertyen et al 1988). Most of the damage to muscle occurs after oxygenation is restored rather than during the period of ischemia (Odeh, 1991). The injuries caused by ischemic reperfusion can continue for several days after reperfusion and convert injured tissue into dead tissue. Death of muscle tissue will occur after 6 or 7 hours of ischemia (Walker, 1991). Figure 2 shows the sequence of events leading to tissue/organ injury after rescue/muscle decompression.

Figure 2. Sequence of events leading to tissue/organ injury after rescue/muscle decompression.



Systemic effects of prolonged recumbency

Figure 3 shows some of the effects of prolonged dorsal, lateral and posterior recumbency on respiration and nerve function.

Lungs

Tidal volume is reduced during normal recumbency but compensated by increased breathing rate, tending to maintain a normal minute volume (Hall 1984). However, ventilation and gas exchange are negatively affected by lateral and dorsal prolonged recumbency, mainly through the pressure exerted by the abdominal contents and the diaphragm on the thoracic cavity (Hornof et al. 1986, Moens et al. 1995). The resulting atelectasis and pulmonary congestion (Nyman et al. 1990) is more pronounced in the caudal pulmonary lobes (Stegmann and Littlejohn 1987).

Size matters during prolonged recumbency. Larger horses will have more vascular congestion than smaller ones (Nyman et al 1990), and smaller horses will be able to oxygenate better, showing higher arterial oxygen values than larger horses (Mansel and Clutton 2008)

Ischemic reperfusion is also known to stimulate the activation of a number of immune factors (complement) and white blood cells (neutrophils). These white blood cells can cause damage to the lung when trapped in lung tissue (Goldman et al 1990)

Circulation

Decreased cardiac output. Arterial CO2 was higher and O2 lower during prolonged dorsal recumbency (Stegmann and Littlejohn 1987). One consequence of ischemic reperfusion is the increase of potassium concentrations in the circulation which can cause cardiac disfunction or arrest (Walker 1991)

Kidneys

The release into the circulation of myoglobin from injured muscle tissue is highly toxic to renal cells (Lewis et al 1984). Further injury is caused by tubular obstruction by myoglobin (Odeh 1991)

Prolonged dorsal/posterior recumbency and spinal cord injury

Several studies exist on the effect of dorsal recumbency on muscle and nerve function in the horse, which is the most common posture during abdominal surgery. In addition to skeletal muscle compression, dorsal recumbency can cause myelopathy of the lower spinal cord resulting in hind limb paralysis. The mechanisms leading to myelopathy after prolonged dorsal recumbency are not known (Ragle et al. 2011).

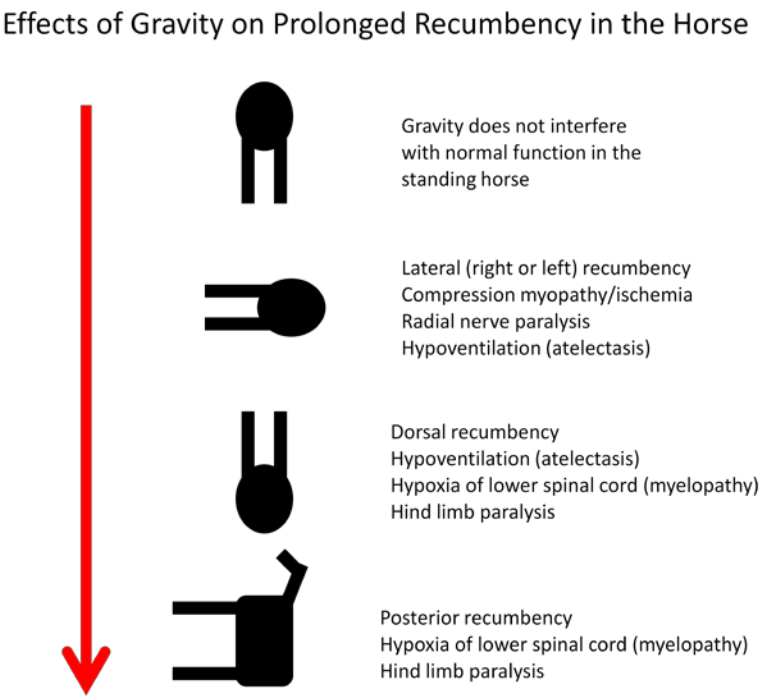


Figure 3. Effects of gravity/compression on prolonged recumbency on ventilation and nerve function in the horse. No information exists on prolonged posterior recumbency, but the consequences are probably similar or more severe than those under dorsal recumbency. Red arrow=gravity

Management

Depending of the severity of the case, the attending veterinarian will select from the following treatments/procedures to alleviate and help prevent organ damage. Veterinary care should be initiated as soon as possible after the rescue.

Repositioning. Turning the recumbent horse to the opposite side will improve gas exchange (Hornof et al 1986)

Disperse bodyweight over a large surface area to reduce compressive forces. Protective padding. Extension of the lower limb (Lindsay et al. 1985)

Intranasal oxygen

iv fluids

Analgesics

Anticoagulants

Anti-inflammatories

Antioxidants, vitamin C

Monitor muscle damage with creatine phosphokinase measurements

Conclusions

Rescue within four hours (“golden hours”) after an incident will improve the chances of uneventful recovery and survival.

Recumbency for more than four hours can have irreversible damaging consequences on a large animal victim.

The attending veterinarian should initiate post-incident care as soon as possible.

Glossary

Atelectasis Incomplete expansion or collapse of lung tissue.

Compartment syndrome A condition that involves increased pressure in a muscle compartment. It can lead to muscle and nerve damage and problems with blood flow. Muscles are contained in "compartments" by a non-elastic tissue called fascia. Fascia does not expand.

Creatinine phosphokinase (CK, CPK) is an enzyme present in heart and skeletal muscle. It is involved in the production of rapid energy (ATP), and its concentration increases during muscle cell damage, making it a marker for muscle cell injury.

Critical ischemic time – The maximum period of time a tissue can tolerate lack of blood supply and still remain viable.

Crush syndrome (traumatic rhabdomyolisis) Is the systemic manifestation of muscle cell damage. After compression is relieved and reperfusion is established, the breakdown products of muscle tissue enter the circulation and affect distant organs.

Edema presence of abnormally large amounts of fluid in intercellular spaces.

Hypovolemia Abnormally decreased volume of circulating blood in the body.

Ischemia reperfusion injury – The consequence of reperfusion following a period of ischemia.

Ischemic reperfusion Restoration of blood flow to an area that had previously experienced deficient blood flow. Oxidative stresses associated with this situation may cause damage to the affected tissues or organs

Minute volume is the amount of air inhaled in a minute. The minute ventilation is calculated by the multiplication of the tidal volume and the respiratory rate.

Myelopathy a term denoting functional and/or pathological changes in the spinal cord.

Myositis Inflammation of voluntary (skeletal) muscle.

Necrosis Tissue death

Recumbency The act of lying down.

Rhabdomyolisis (*rhabdo*, rod shaped, *myo*, muscle, *lysis*, dissolution) A syndrome in which injury to skeletal muscle results in the leakage of contents of the muscle cells into the blood.

Reperfusion Restoration of blood flow to a tissue.

Revascularization Resumption of blood flow to a tissue.

Tidal volume is the lung volume representing the normal volume of air displaced between normal inspiration and expiration when extra effort is not applied.

Vasodilation Expansion of blood vessels leading to increased blood flow.

References

Blaisdell F W 2002 The pathophysiology of skeletal muscle ischemia and the reperfusion syndrome: a review. Cardiovascular Surgery 10(6): 620-630

Bulkley G B 1987 Free-radical mediated reperfusion injury: a selective review. Br J Cancer Suppl 8:66-73

Gillani S, Cao J, Suzuki T and Hak D J 2011 The effect of ischemia reperfusion injury on skeletal muscle . Injury In press, Epub ahead of print PMID 21481870

Hall L W 1984 Cardiovascular and pulmonary effects of recumbency on two conscious ponies. Equine vet J 16(2):89-92

Hornof W J, Dunlop C I and Amia T C 1986 Am J Vet Res 47(2): 278-302

Lindsay W A, Pascoe P J,McDonel W N and Burgess M L F 1985 Effect of protective padding on forelimb intracompartmental muscle pressures in anesthetized horses. Am J Vet Res 46(3): 688-691

Littlejohn A and Munro R 1972 Equine recumbency Vet Rec 90: 83-85

Mansel J C and Clutton R E 2008 The influence of body mass and thoracic dimensions on arterial oxygenation in anaesthetized horses and ponies. Veterinary Anaesthesia and Analgesia 35: 392-399

Moens Y, Lagerweij E Gootjes P and Poortman J 1995 Distribution of inspired gas to each lung in the anesthetized horse and influence of body shape. Equine Vet J 27(2): 110-116

Nyman g Funkquist B, Kvart, Frostell C, Tokcs L, Strandber A, Lundquist H, Lundh B, Brismar B and Hedenstierna G 1990 Atelectasis causes gas exchange impairment in the anesthetized horse. Equine Vet J 22(5):317-324

Odeh M 1991 The role of reperfusion-induced injury in the pathogenesis of the crush syndrome. New Eng J Med 324(20):1417-1422

Ragle c, Baetge C, Yiannikouris S, Dunigan C, Schneider R and Keegan R 2011 Development of equine post anaesthetic myelopathy: Thirty cases (1979-2010). Equine Veterinary Education 23(12): 630-635

Sertyen D, Lavergne L, Coppens P, Mottart E, Philippart C, Micheels M and Lamy M 1986 Equine post anaesthetic myositis: Muscular post ischaemic hyperaemia measured by laser Doppler flowmetry. Vet Rec 123:126-128

Stegmann G F and Littlejohn A 1987 The effect of lateral and dorsal recumbency on cardiopulmonary function in the anesthetized horse. Journal of the South African Veterinary Association 58(1):21-27

Steinhart P 1937 Der Schlaf des Pferdes, seine Dauer, Tiefe, Bedingungen (The horse's sleep, duration, depth, conditions)

Zeitschrift für Veterinärkunde 49(6) :193-232

Walker P M 1991 Ischemia/reperfusion injury in skeletal muscle. Annals of Vascular Surgery 5(4):399-402