

Extracorporeal Shock Wave Therapy: What is It? What Does it Do to Equine Bone?

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When extracorporeal shock wave therapy (ESWT) was administered to the equine metacarpus/metatarsus, there was subperiosteal and subendosteal hemorrhage, but no microfractures were detected. No damage to the surrounding soft tissues was detected. The principle of ESWT and the acute effects of ESWT on equine bone and soft tissue are explained. Authors' Addresses: Department of Veterinary Clinical Sciences (McClure), Department of Basic Medical Sciences (VanSickle), and Department of Pathobiology (White), Purdue University, West Lafayette, IN 47907-1248. © 2000 AAEP.

ESWT—What is It?

Extracorporeal shock waves are pressure waves generated outside the body that can be focused at a specific site within the body. Shock waves are characterized by high positive pressures, up to 80 MPa, and negative pressures of 5–10 MPa.¹ They have a rapid rise time of 30–120 nsec and a short, 5 μ sec, pulse duration.¹ They are differentiated from ultrasound by lower frequency, minimal tissue absorption, and no thermal effects. The pressure waves travel through fluid and soft tissue and their effects occur at sites where there is a change in impedance, such as the bone-soft tissue interface. The common use for shock waves is to break renal and ureteral uroliths into small fragments (lithotripsy) that can then be passed from the body by the lower urinary tract.^{2,3}

There are 3 mechanisms to generate a shock wave: 1) piezoelectric; 2) electromagnetic; and 3) electrohydraulic.¹ The piezoelectric system utilizes a crystalline material, that when stimulated with

high voltage electricity, can expand or contract to initiate a pressure wave in the surrounding fluid. The electromagnetic mechanism has coils that create opposite magnetic fields when an electric current is applied to them causing a submerged membrane to move, starting a pressure wave within the fluid. The electrohydraulic method uses a high voltage spark gap. The spark generates a plasma bubble that compresses the liquid, initiating the pressure wave. Each mechanism creates a characteristic wave form and energy density.

When the shock wave meets an interface of different impedance, compression and shear loads develop.¹ Cavitation occurs with the development of gas bubbles as a result of the rapid interaction between compression and shear.¹ The collapse of the gas bubbles leads to the development of fast flows or jet streams that are postulated to contribute to the effect on the hard tissue. Mechanisms of stone destruction are thought to be the result of compression and tension generated by the shock

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wave. The most common use of shock waves is for lithotripsy. Uroliths are a brittle material easily fractured by these physical forces and scanning electron microscopy of urolith fragments provides tangible evidence that stone fracture is the result of these forces.

Extracorporeal shock wave therapy (ESWT) has nonurologic uses, including treatment of sialoliths and gall bladder stones. At high energies, ESWT can be cytotoxic and has been investigated as a noninvasive mechanism to destroy neoplastic cells and to increase cell wall permeability, thereby increasing cytotoxicity of chemotherapeutics. It was also noted early in the development of lithotripsy that shock waves have an osteostimulatory effect.

Shock waves are now routinely used in Europe to treat common orthopedic conditions in humans including plantar calcaneal spurs⁴ (heel spurs), epicondylar pathic humeri radialis^{4,5} (tennis elbow), and nonunions,^{4,6} and are currently being evaluated by the Food and Drug Administration (FDA) for similar use in the United States. In Europe and the United States, shock wave technology is being used for the treatment of equine musculoskeletal diseases.

ESWT—What Does it Do to Equine Bone?

In the 1990s ESWT was found to have a stimulatory effect on bone formation. Subsequently, it has become an accepted therapeutic modality to treat nonunions and enthesopathies in humans. The mechanisms by which shock waves affect bone are minimally understood. It is unknown whether the results of shock waves on bone are the result of direct pressure effects or by cavitation. Furthermore, the actual mechanism by which bone formation is stimulated is unknown. Initial *in vitro* experiments identified that formalinized rabbit bone can be fractured with ESWT and that the effects are dose-dependent.⁷ The energy release focused on the formalinized bone has resulted in microfracturing of cortical trabeculae and medullary hemorrhage in long bones of rabbits.⁸ Subsequent *in vivo* studies have shown that shock waves can stimulate osteogenesis in the long bones of rabbits and dogs when the correct number of shock waves are delivered at the appropriate energy level.^{8–10} Several weeks following treatment, responses noted in the bones included thickening of cortical bone, increase in the numbers of trabeculae, and increase in the number of osteoblasts. *In vivo* studies did not support microfractures as the mechanism of osteogenesis; rather it was suggested that bone marrow hypoxia and subperiosteal hemorrhages stimulated the osteogenesis.⁸

Histologic studies of the effect of ESWT on bone have been limited to smaller laboratory species, primarily sheep, dogs, and rodents. To determine if shock waves have an acute effect on the dense thick equine cortical bone, we evaluated the effect of extracorporeal shock waves on the equine metacarpus/metatarsus (MC/MT). The objective of this study

was to identify the acute morphological changes that occur in equine bone and associated soft tissue structures with ESWT.

Materials and Methods

Four sound geldings (1 Arabian, 1 American Saddlebred, 2 Quarter Horses) with a mean age of 4.5 years (range 3–7) with radiographically normal MC/MT were used for this investigation. One metacarpus was treated with 1000 pulses from an electrohydraulic generator at an energy 0.89 mJ/mm² to the dorsal aspect of the middle of the bone and the opposite limb served as the nontreated control. One metatarsus was treated the same as the metacarpus and the opposite at the maximum energy of 1.80 mJ/mm² for 1000 pulses. The treated leg (left or right) and the leg position (up or down) were randomly assigned. The horses were euthanized without recovery from anesthesia.

At necropsy, soft tissues of treated limbs and untreated (control) limbs were harvested, identified, and placed in 10% neutral buffered formalin solution. Representative sections of skin, subcutis, extensor and flexor tendons, suspensory ligament, and associated neurovascular structures were obtained at the focal point of treatment and at 5 mm increments for a distance of 25 mm proximal and distal to the focal point. Following fixation, soft tissues were trimmed, processed for routine histology, and resulting slides stained with hematoxylin and eosin stains.

Bones, including periosteum were embedded in polymethylmethacrylate, sectioned, and ground using the Exakt^a cutting/grinding system resulting in 75–100 μ m thick sections. Then 5–10 μ m of the surface was etched with dilute formic acid and the sections were stained Wright's-Giemsa/toluidine blue technique.

Results

There were no significant lesions identified grossly or histologically in the skin, tendons, suspensory ligament, or associated neurovascular structures in the treated or control specimens. Subperiosteal and endosteal hemorrhage was seen grossly at the focal point in the treated limbs. The area of subperiosteal hemorrhage was greater in the bones treated at the higher energy level. There were no microstructural changes observed in the bone.

Discussion

Included in the issue of how shock waves affect living tissue are the variables of energy levels, number of shocks applied, and the shock wave generation mechanism. One consistent finding is that the energy level selected is important. Studies in pig skin defects found that low energy shock waves stimulate skin healing whereas high energy shock waves slowed healing.¹¹ In a rabbit Achilles tendon dose effect study, tendon necrosis occurred at energy densities over 0.28 mJ/mm². In studies in-

volving the application of shock waves on bones, it was determined that relatively low energy levels do not stimulate bone formation whereas those that use high energy levels result in bone formation.¹²

In this acute study of the effect of extracorporeal shock waves on the equine MC/MT at energy densities up to 1.8 mJ/mm², no damage to the associated soft tissue structures was found and the treatment did not induce microfractures of the cortical bone. This study supports the findings of a previous study that the primary mechanism for stimulation of osteogenesis is not the induction of microfractures.⁸ Other potential mechanisms for the stimulation of osteogenesis by extracorporeal shock wave therapy are bone marrow hypoxia, subperiosteal hemorrhage, increased regional blood flow, and activation of osteogenic factors such as bone morphogenic protein, direct cellular effects, and mechanical effects as a result of strain gradients. Further investigations are needed to evaluate the factors that stimulate osteogenesis following shock wave therapy. Shock wave therapy can be utilized in the horse without acutely damaging the surrounding soft tissues.

References and Footnotes

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