The objective of this seminar will be to review some physical methods of stimulation of bone healing. Most cells in the body experience a range of physical forces, which when isolated individually are capable of modulating cellular activity. Gravitational, acoustic, thermal, shear, compressive/tensile, electrical and hydrostatic forces can all result in a cellular response. We will cover 3 modalities, electrical/magnetic, ultrasound, and shockwaves that create physical forces within the body and have been used for a physical method of stimulating bone healing.

**Electrical Forces**

The recognition of piezoelectric properties in bone resulted in speculation and research into electrical fields to stimulate bone formation. Early work utilized direct current through the bone using implanted electrodes. This resulted in bone formation near the cathode and potentially, tissue damage at the anode. Pulsed electromagnetic fields utilize parallel coils which create a magnetic field and electric currents between them without electrodes. The pulses can be varied to change the electrical fields, potentially creating an environment ideal to stimulate different tissue types. Utilization of pulsed electromagnetic fields has been shown effective in decreasing bone loss in laboratory animals. There is limited information available from use of PEMF in the horse, most from the late 1980’s. Two separate scintigraphic studies showed either no effect or a positive effect, both done in normal metacarpi. Two trials utilizing osteotomy models in horses failed to show any benefit of PEMF.

**Ultrasound**

Ultrasound has been shown to stimulate the healing of fresh fractures in double-blind controlled clinical trials in humans. A meta-analysis of low-intensity pulsed ultrasound therapy in humans concluded that it was an effective therapy with an improvement in healing time of 64 days compared to similar untreated fractures. Animal studies indicate that treatment with low-intensity ultrasound results in a stronger and stiffer callus formation with acceleration in endochondral ossification. In the horse 2 studies have been completed. A densitometric evaluation of dorsal cortical drill holes showed a varied response but overall was considered to have enhanced repair. A randomized, blinded, controlled trial was conducted in 8 healthy adult horses with a 1-cm osteotomy of each 4th metacarpal bone. There were no significant differences between treated and control bones for any of the radiographic, pQCT or histological parameters evaluated.

**Shockwave Therapy**

Early studies of SWs on bones yielded variable results and were confusing. Very high energy treatment of rodent and rabbit bones resulted in physical disruption of the bone and periosteum resulting in the thought that bone remodeling was the result of this physical damage. Studies in more appropriate tissue and species have resulted in a stimulation of bone formation without physical disruption. The ability to stimulate bone healing resulted in the interest in utilizing SWs for the treatment of nonunion fractures. Nonunion fractures were created in 10 beagles with segmental radial osteotomies. Five of the dogs with nonunions were treated with 4000 SWs at 0.54 mJ/mm² and 5 served as untreated controls. All of the treated dogs had
osseous union by 12 weeks after treatment compared to only 1 dog in the control group. Clinical trials in humans have demonstrated bone healing following SWT in delayed and nonunions. Wang reported 80% success 12 months after treatment in 72 nonunions of long bones, Schaden had a 75% success rate in delayed unions and nonunions in 115 patients and Rompe reported on 43 patients with femoral and tibial nonunions with 72% healed in 4 months.

The mechanisms of SW induced osteogenic stimulation are being investigated. There are 3 potential mechanisms: 1) Stimulation of growth factor expression, 2) Recruitment and differentiation of mesenchymal cells, and 3) Response to periosteal damage and microfractures. There is an increase in osteoprogenitor colony forming units from the marrow of rat femora 24 hours after treatment with 500 SWs at 0.16 mJ/mm². A dose response was seen with the highest TGF-B1 concentrations in the supernatant after 500 SWs with lower numbers of pulses ineffective and higher numbers inhibitory. The same treatment protocol was administered to segmental defects of rats. This resulted in an increased expression of TGF-β1 and VEGF-A mRNA and an increase in osteoprogenitor cells in shock wave treated femora. While it is possible to disrupt the periosteum and create microfractures with shock waves, similar osteogenic responses are seen without utilizing these energy levels, decreasing the significance of that proposed mechanism.

To date there are no controlled studies of osteotomy or fracture gap healing with shockwave therapy in the horse. In an in vivo pilot study in horses that used 2000 SWs at 0.89 mJ/mm², 2 treated MCIII had 30% more activated osteons than the opposite untreated control. Scintigraphic and histological examination of the fourth metatarsal bone after 2000 pulses at 0.15 mJ/mm² resulted in an increased number of osteoblasts and correlated with an increased radiopharmaceutical uptake in the treatment area.

References