REFERENCES:


CRANIAL BONE HEALING was evaluated using a rat model at UCLA.¹

Seventeen 3 mm calvarial defects were created in rat calvaria. The amount of new bone formation at 3 weeks was calculated using micro-CT data. The amount of new bone formation filling the defects is shown above. The Ostene filled defects had significantly more bone formation than those filled with bone wax (p ≤ .001).

STERNAL BONE HEALING was evaluated in a rabbit sternotomy model.²

Median sternotomies were performed on 20 rabbits, and hemostasis was achieved using either bone wax or Ostene.

After 6 weeks the Ostene treated sternums were significantly stronger (p ≤ .001) with twice the strength of bone wax treated ones. The bone wax treated sternums had fibrous tissue and residual wax interfering with healing. The Ostene group healed normally.

TIBIAL BONE HEALING was evaluated in a rabbit model. Twenty-four 3 mm circular cortical defects were created in proximal tibias of rabbits. Immediate hemostasis was achieved with either Ostene or a microfibrillar collagen hemostat. After 17 days, bone healing was measured quantitatively using micro-CT. Near complete bone healing occurred in the Ostene treated group, with little healing in the microfibrillar collagen group. Compared to Ostene, osteogenesis was significantly impaired by microfibrillar collagen (p < .0001).

Inflammation and swelling are particularly problematic in closed spaces such as those encountered in the cranium and spine.

Ostene is non-inflammatory. Studies have shown that bone wax, gelatin sponges, collagen and oxidized cellulose may lead to chronic inflammatory reactions and/or interfere with bone healing.³ Inflammation and swelling are particularly problematic in closed spaces such as those encountered in the cranium and spine.

SUSCEPTIBILITY TO BONE INFECTION was evaluated in a rabbit tibial model. Cortical bone defects were created in the proximal tibia in 24 rabbits. The defects were either treated with bone wax, Ostene, or left without a hemostatic agent, and all were inoculated with Staph aureus.

After 4 weeks all defects containing bone wax became infected and developed osteomyelitis, with destruction of the bone marrow, abscess formation, periostal reaction, and bone lysis. There was no bone healing in any of the defects treated with bone wax.

In the Ostene and control groups, 2 defects in each group (25%) developed osteomyelitis. The remaining 6 defects in each group (75%) showed no evidence of osteomyelitis, and all exhibited normal bone healing.

The application of bone wax to the cortical defect significantly increased the osteomyelitis rate (p ≤ .004). There was no difference between the Ostene group and the control group in the rates of osteomyelitis, positive cultures, or bone healing.