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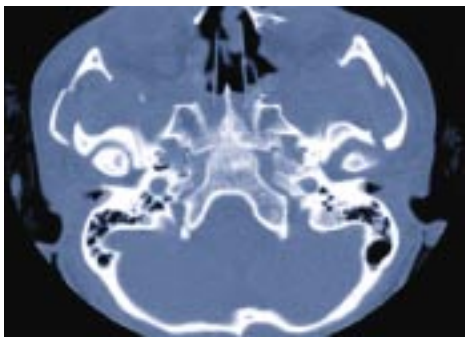
Epidemiology of facial trauma and associated morbidity of mandibular fracture treatment

by **Vincent B. Ziccardi**

The causes of facial trauma are multifaceted with a variety of etiologies ranging from falls and assaults to motor vehicle and sporting accidents. The frequency of any specific etiology varies within geographic regions and is dependent upon life style factors, population density and socioeconomic status. Urban trauma centers such as UMDNJ-University Hospital in Newark evaluate and treat many facial trauma patients on a daily basis. The Department of Oral and Maxillofacial Surgery and the divisions of Plastic Surgery and Otolaryngology are consulted by the Emergency Department and trauma team to assist with the management of many facial injuries at this center. Our research has focused on data collection regarding facial trauma and studies on the outcome and morbidity associated with the treatment of mandibular fractures.



Axial CT scan of a patient with a LeFort III facial fracture. This section depicts the area of the zygomatic arches.

We evaluated the patients presenting to the oral and maxillofacial surgery service for consultation regarding facial trauma. Patients were asked to consent to participate in our study and underwent a standardized interview to collect data regarding age, race, gender, social habits, mechanism of injury and incidence of previous facial trauma. Mandibular fractures accounted for the majority of injuries encountered at our institution, followed by lacerations and then by other miscellaneous facial injuries. Assault was the primary cause of injury and motor vehicle accidents were the next most frequent. In those patients who had experienced previous injuries, the majority had been victims of an assault. Patients who were previously assaulted were 1.5 times more likely to have experienced an assault as the cause for the current injury. The study demonstrated that the most common facial injuries treated at this trauma center resulted from interpersonal violence and that half those patients presenting have a history of previous facial traumatic injuries.

Other research has focused on the outcome and morbidity associated with the treatment of mandibular fractures. Some controversy still remains in the literature regarding the optimal treatment modalities for mandibular fracture management. Since the development of rigid fixation plating systems, options for mandibular fracture treatment now also include open techniques in addition to traditional techniques utilizing closed reductions. Open reduction with rigid fixation techniques involve the reduction of fractures through surgical incisions with stabilization using plate and screw fixation systems. This contrasts to closed techniques, where patients' jaws are wired together for immobilization of the fractures using their natural dentition, surgical stents or a combination of both. In a patient population sometimes demonstrating poor compliance with follow-up, inadequate nutrition, high incidence of substance abuse and unwillingness to perform appropriate homecare, open techniques offer an advantage over closed techniques, which enable accurate approximation of

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segments with earlier restoration of function and concomitant decreased need for maxillo-mandibular fixation or the wiring of jaws.

The most common location for mandibular fractures was found to be in the third molar or angle region of the mandible. Our overall complication rate was consistent with other large retrospective published studies. Infection accounted for the highest incidence of complications followed by non-union of the fractures. Closed reduction techniques yielded a lower level of complications when compared to open techniques; however, the data were not standardized as to severity of presenting fractures. Most complications encountered in the study population were relatively minor and resulted in eventual favorable outcomes.

Newer techniques of rigid fixation are constantly being developed to optimize treatment outcomes for facial fracture management. We have been involved with laboratory testing of different plating systems including resorbable plates, lag screw systems and most recently mandibular locking screw plates. Recent biomechanical studies involving resorbable plating systems yielded some interesting findings. It is necessary to heat plates in order to adapt the plates to the facial skeleton. Our study involved repetitive heating cycles with adaptation of the plate to the orbital-zygomatic region and stress testing with molecular weight analysis. Molecular weight was found to decrease with repetitive heating and bending by up to 18%. Results of the study indicate that repetitive bending and heating of the resorbable plating systems may affect the mechanical and molecular properties, although not to an extent that is clinically significant. It would be prudent for the surgeon to limit the number of heating cycles when possible.

Another study assessing failure strengths of locking screw plates versus conventional mandibular plates was undertaken utilizing bovine ribs as a model for the human mandible. The premise behind the locking screw plate is to distribute forces between the threaded portion of the plate and screw rather than generating compressive forces between the plate and the lateral cortical plate of the mandible. This is postulated to limit stress shielding and allow for more stable fixation over time, hence preventing failure of rigid internal fixation. From a mechanical perspective, it was determined that there was no statistical difference between both systems. It was concluded that success of the plates may be more related to variables in operator application and bone quality rather than to differences in the hardware. Clinical prospective data are needed to investigate this hypothesis further. It is hoped that studies such as those described above will aid in the development of improved systems and techniques for the surgical treatment and rehabilitation of our facial trauma patients.

Vincent B. Ziccardi, DDS, MD, is an associate professor and chair of the Department of Oral and Maxillofacial Surgery at UMDNJ-New Jersey Dental School. Dr. Ziccardi joined the faculty of NJDS in 1997. He received his BA in chemistry from New York University, DDS from Columbia University School of Dental and Oral Surgery and MD from the University of Pittsburgh School of Medicine. He completed his training in oral and maxillofacial surgery, including a general surgery internship, at The University of Pittsburgh Medical Center. A large part of Dr. Ziccardi's clinical practice involves the management of facial trauma, including secondary post-traumatic reconstructive surgery which may encompass soft tissue surgery, bone grafting and possibly placement of dental implants to restore avulsed dental and alveolar structures. Dental reconstruction is coordinated with his colleagues at New Jersey Dental School. Many of the principles of facial trauma secondary reconstruction also apply to his efforts in restoring form and function for patients having undergone ablative surgery for pathological conditions.

Impaired bone healing in patients with diabetes mellitus

by **Sheldon S. Lin**

Fracture healing is a process of restoring the structural and biological properties of injured bone. It has been well documented that diabetes mellitus (DM), a systemic disease affecting 17 million Americans, causes increased healing time with a concomitant increase in delayed unions and non-unions. Unfortunately, the specific mechanism for the delayed fracture healing in patients with diabetes has yet to be elucidated.

The purpose of our research is to evaluate the role of insulin and glucose control on the fracture healing process using our recently established femur fracture model in diabetic BB Wistar rats. We theorize that insulin plays a critical role in the fracture callus, especially on the early inflammatory phase and the expression of local growth factors, and its relative absence leads to impaired fracture healing in diabetics.

Annually, 6 million fractures are treated in the U.S., with 5-10% exhibiting complications such as delayed union or non-union. Etiologies of impaired fracture healing include smoking, open fractures, presence of underlying infection, certain medications (i.e. steroids) as well as systemic disease such as diabetes mellitus (DM). It is estimated that more than 30 million Americans will be diagnosed with diabetes within the next decade.

The effects of diabetes upon fracture healing have been well documented with increased healing time (2-3 times the normal rate) and concomitant increased complications (delayed union, non-union, etc). In order to study these problems, our lab has established a diabetic femur fracture model for analyzing the effect of tight blood glucose and the role of early critical growth factors upon impaired diabetic fracture healing.

Diabetic Femur Fracture Model

Previous diabetic models were induced with cytotoxic agents (alloxan, streptozotocin) that preferentially destroy pancreatic beta cells. These cytotoxins result in a clinical condition of insulin-dependent DM, Type I diabetes. The key criticism of this method was its inability to determine whether the deficient diabetic fracture healing process is due to the systemic effect of the cytotoxins, malnutrition and/or the diabetic condition itself.

Our recently published diabetic femur fracture model utilizes BB Wistar rats, which spontaneously develop diabetes through the autoimmune destruction of pancreatic β cells. The spontaneous onset of diabetes in the BB Wistar rat confers advantages over the viral, chemical and immunological induction of DM. Within seven days after glycosuria, the beta cells were completely destroyed and if untreated, marked wasting of the body tissue, including fat, muscles, protein, dehydration and ketosis supervene. Death usually resulted within five to 10 days after onset. These conditions were resolved with insulin treatment. The BB Wistar rat currently represents a close homology of human Type I diabetes in a laboratory animal.

Diabetic Femur Fracture Model: Effect of Blood Glucose Control

Our published studies were able to demonstrate that femur fracture healing in poorly controlled diabetic rats (blood glucose > 300 mg/dl) is reproducibly delayed compared to non-diabetic control animals. Tight glucose control, through increased insulin treatment resulting in blood glucose val-