Enclosed in this issue: Trauma Reports

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INSIDE

MRI Finds Hip or Pelvic Fractures After Initial Negative Plain X-rays

ABSTRACT & COMMENTARY

Source: Dominguez S, et al. Prevalence of traumatic hip and pelvic fractures in patients with suspected hip fracture and negative initial standard radiographs — a study of emergency department patients. *Acad Emerg Med* 2005;12:366-370.

THE PURPOSE OF THIS STUDY WAS TO EVALUATE THE INCIDENCE of hip fractures presenting to the emergency department (ED) with negative initial radiographs. This was a retrospective cohort study performed in an academic community ED. The cohort included all adult patients presenting to the ED during a one-year period with hip pain who had received standard plain radiographs. Eligible patients were collected by a query of an electronic radiology log and medical records.

All plain radiographs and magnetic resonance imaging (MRI) studies were ordered solely at the discretion of the treating emergency physician. Initial plain films were read in real-time by a board-certified radiologist; all MRI studies were read by a fellowship-trained musculoskeletal radiologist, and re-read by a similarly trained radiologist to assess interobserver agreement. A fracture was defined as any fracture seen on plain film or MRI, or subsequent diagnosis of hip fracture as determined by follow-up phone call or review of subsequent medical records.

A total of 895 patients had hip films performed during the study period, with 764 (85%) completing follow-up. The mean age of the 764 patients included was 66.7 years, and 62% were female. Of the patients enrolled, 95% had a history of blunt trauma, most of whom (85%) were injured in a fall. Two hundred-nineteen patients (29%) had fractures identified on plain films; the most common fractures involved the femoral neck (36%) and intertrochanteric region (29%).

Of the 545 patients with negative plain films, 62 (11%) had MRI studies ordered by the treating physician. MRI identified 29 fractures in 24 patients (10% of all fractures identified in the study) with negative initial plain films. Overall, 4.4% of all patients with negative initial plain films had a fracture identified by MRI. Of the

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patients with negative plain films and positive MRI study, 92% were 65 years of age or older. Characteristics of the fractures identified by MRI were as follows: 35% involved the pubic rami, 28% involved the sacrum, 14% involved the femoral neck, 7% involved the intertrochanteric region, 3% involved the femoral head, 3% involved the acetabulum, and 10% involved other areas. The interobserver agreement for the presence of fracture on MRI was very good.

Among patients with initial negative plain films who did not receive MRI in the ED, no patient was determined to have a fracture on follow-up review of records or one-month post-visit phone call. The authors suggest that there is considerable potential for the development of a clinical decision rule to identify patients presenting to the ED with hip pain and negative plain films who remain at risk for fracture. ❖

COMMENTARY BY JACOB UFBERG, MD

This study noted that approximately 10% of fractures in the cohort were not recognized by initial plain radiography, and that 4.4% of patients with hip pain and negative initial plain films had fractures identified by MRI. At first glance, these numbers suggest that MRI (or perhaps computerized tomography?) should be used more liberally by emergency physicians in an attempt to reduce the number of missed fractures.

However, it is important to note several aspects of this study. It appears that by using only physician discretion, all fractures were identified on the initial visit. No patients were diagnosed with hip fracture subsequent to the initial visit, and the fracture rate among the patients who had MRI studies was 39%. It appears that the clinical judgment of emergency physicians (at least the ones in this particular ED) is pretty good, and may not need the help of a clinical decision rule.

Additionally, we should note that the majority of fractures identified by MRI were types of fractures that are managed conservatively. However, was every fracture truly identified? I find it hard to believe that while 10% of fractures were missed by initial radiograph in this study, there were truly no fractures among the nearly 500 patients with negative initial radiographs who did not undergo MRI. Most likely, some number of patients went home with clinically insignificant or conservative management-type fractures and recovered uneventfully.

What we really need is a clinical decision rule that targets the use of advanced imaging toward identifying fractures that are truly significant: ones that require operative management or carry a high risk of complication. My guess is that the medical/legal risks of not diagnosing ANY fracture will keep us from reaching this goal.

GIK Infusion Ineffective in Acute MI

ABSTRACT & COMMENTARY

Source: Mehta SR, et al. Effect of glucose-insulin-potassium infusion on mortality in patients with acute ST-segment elevation myocardial infarction. The CREATE-ECLA randomized controlled trial. *JAMA* 2005;293:437-446.

GLUCOSE-INSULIN-POTASSIUM (GIK) INFUSION LONG has been touted as a simple, inexpensive, and effective treatment for patients presenting with acute myocar-

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dial infarction (AMI).[1] However, the evidence in support of GIK infusion largely has been based on small, non randomized clinical reports. In this study, investigators conducted a large, international, randomized controlled clinical trial (as a part of the CREATE trial) to determine the effect of GIK infusion on mortality in patients presenting with acute ST-segment elevation MI (STEMI).

More than 20,000 patients presenting to 470 centers worldwide within 12 hours of symptom onset with STEMI were randomized to receive either GIK infusion in addition to usual care or usual care alone. Contraindications included diabetes mellitus type 1, renal impairment, or known hyperkalemia. Patients were randomized from the original CREATE study in India and China (see original article abstract), as well as from a separate GIK infusion trial ongoing in Latin America (ECLA). Patients assigned to the GIK infusion group received a 24-hour infusion at a rate of 1.5 mL/kg/hour for 24 hours that included a 25% glucose solution with 50 u/L of regular insulin and 80 mEq/L of potassium.

Overall, 10,091 patients were included in the GIK infusion group and 10,110 in the control group, indicating that this study had 95% power to detect a 15% relative risk reduction with the GIK infusion group. There was no difference between the two groups in the administration of aspirin (97.4 vs 97.1%), fibrinolytic therapy (73% vs 74.2%), or direct percutaneous coronary intervention (9.2% vs 9.0%) for GIK infusion vs control, respectively. At 30 days, there was no difference in the primary outcome measure of death (10.0% vs 9.7%, respectively, $P = 0.45$). Similarly, there were no significant differences in the composite endpoints of death and nonfatal cardiac arrest, death and cardiogenic shock, or death and reinfarction. There was also no difference in the rates of ventricular tachycardia and fibrillation (21.0% vs 21.4%), second- or third-degree atrioventricular block (19.9% vs 19.8%), or pulseless electrical activity (0.4% vs 0.5%), respectively.

Furthermore, there was no difference in onset of new congestive heart failure episodes at 30 days (17.4 vs 17.4%). However, there was an increase in both hypoglycemic (0.4% vs 0.1%) and hyperkalemic (>5.5 mEq/L) (4.3% vs 1.6%) episodes in the GIK infusion group compared with the control group. In addition, on subgroup analysis, the investigators could not detect any benefit of GIK infusion over usual care alone for those who were treated early (< 4 hours), nor was there any difference with GIK infusion in patients who received fibrinolysis or direct percutaneous coronary intervention (PCI) reperfusion therapies.

Based upon their results, the CREATE-ECLA group concluded that high-dose GIK infusion for patients with acute STEMI has a neutral effect on mortality, cardiac arrest, and cardiogenic shock. ❖

COMMENTARY BY THEODORE CHAN, MD, FACEP

The idea of GIK infusion for AMI patients has been reported in the medical literature since the 1960s. The theory of metabolic modulation to reduce AMI mortality rests on several different mechanisms. Exogenous insulin may suppress the circulation and myocardial uptake of toxic free fatty acids; high-dose glucose may provide an important energy substrate for ischemic myocardium; and potassium infusion would raise the dysrhythmia threshold in ischemic mycytes. However, investigations into the benefits of GIK infusion are limited to small clinical studies and reports that likely were skewed toward a positive outcome as a result of publication bias.

With this large, randomized, international clinical trial, the idea of GIK infusion for AMI largely has been laid to rest.

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Prehospital Intubation with Neuromuscular Blockade: The Pendulum Swings Again?

ABSTRACT & COMMENTARY

Source: Bulger EM. The use of neuromuscular blocking agents to facilitate prehospital intubation does not impair outcome after traumatic brain injury. *J Trauma* 2005; 58:718-724.

This is a retrospective study of consecutive head injury patients admitted to a single Level 1 trauma center. The authors studied the effect of the use of prehospital neuromuscular blocking agents (NMBAs) on outcome. Intubations were performed by ground paramedics and flight personnel with training and experience in rapid sequence intubation (RSI). Patients were stratified by Glasgow Coma Scale (GCS) score into *mild* (GCS score 14-15), *moderate* (GCS score 9-13) and *severe* (GCS score <9) groups.

Of 2,012 patients with complete records, 920 were classified as mild (intubation rate 17.4%), 293 as moderate (intubation rate 57.7%), and 799 as severe (intubation rate 95%). Overall, 72% of patients received NMBAs. The two groups (those receiving NMBAs and those not) had similar demographics and GCS scores, although hypotension was more common in patients intubated without paralysis. Patients receiving neuromuscular blockade were significantly more likely to survive (odds ratio 0.63; 95% CI, 0.4-0.97) and have a good neurologic outcome (odds ratio 1.7; 95% CI, 1.2-2.6). The authors concluded that, while the fund amental "to intubate-or-not-to-intubate" question requires additional scrutiny, the use of prehospital RSI provided a survival advantage when compared with prehospital intubation without NMBAs. ❖

COMMENTARY BY MICHAEL GIBBS, MD, FACEP

During the past two decades, the prehospital airway management pendulum has been swinging violently from side to side. Early studies demonstrating an outcome benefit of prehospital RSI prompted widespread adoption of the technique by air-medicine and a growing appetite for it among ground-based programs.[1,2] The scene changed completely in 2003, when Davis reported significantly worse outcomes in head injury patients intubated in the field with RSI.[3] A follow-up study of the same patient cohort demonstrated a frightening incidence of hypoxemia.[4] Finally, a large review of the Pennsylvania trauma database suggested that prehospital intubation was associated with a higher risk of death.[5]

Despite some design flaws, Bulger's study is important, and it pushes the pendulum back a bit toward the "RSI-yes" camp. That being said, the only way we will ever answer this vexing question is by conducting a larger prospective randomized trial.

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The Clostridial Connection

ABSTRACT & COMMENTARY

Source: Passaro DJ, et al. Wound botulism associated with black tar heroin among injection drug users. *JAMA* 1998; 279:859-863.

A 46-YEAR-OLD MALE PRESENTED TO A SAN JOSE, California, hospital ED with bilateral diplopia, dysphagia, dysphonia, and weakness of his proximal arms. He had been seen in several other medical care facilities during the previous days without diagnosis. The patient regularly self-injected with black tar heroin and had been doing so for 8 years.

On examination, he had ophthalmoplegia, mild ptosis, and hypophonia with a nasal quality, as well as mild proximal weakness in both arms. Subcutaneous abscesses were present.

Because of a presumptive diagnosis of wound botulism, the California Department of Health Services was contacted immediately, and the Centers for Disease Control and Prevention (CDC) promptly provided bivalent A/B antitoxin. The antitoxin was administered within 12 hours of presentation to the ED. The patient was monitored closely in the intensive care unit, where he was given intravenous penicillin G. However, he became progressively weaker and required mechanical ventilation. His subsequent course was complicated by the development of pneumonia due to Enterobacter organisms. He was extubated after 3 weeks, but required an additional 6 weeks of physical rehabilitation because of his dysphagia and proximal upper arm weakness. He finally was discharged 64 days after admission. ❖

COMMENTARY BY ALEX STUDEMEISTER, MD

Since the 1990s, black tar heroin use among injection drug users (IDUs) has increased, especially in California. Public health officials from the California Department of Health Services have recognized epidemics of three types of Clostridium-associated diseases: wound botulism, necrotizing soft-tissue infections, and tetanus. These emerging infections and intoxications have been associated with the use of contaminated black tar heroin, an association known as "the clostridial connection".

Wound Botulism

Wound botulism, caused by *Clostridium botulinum*, causes acute flaccid paralysis with cranial nerve dysfunction. Botulinum toxin blocks the release of acetylcholine by the presynaptic nerve endings of the peripheral nervous system and cranial nerves leading to muscle

weakness. Patients develop descending flaccid paralysis with cranial nerve palsies, but without sensory deficits and with maintenance of a clear sensorium. Untreated, botulism can lead to respiratory paralysis and death.

The number of cases of wound botulism has risen sharply since 1994. Health officials have documented 163 cases since 1988, 156 (96%) of which occurred among IDUs. Of the 163, 142 (88%) injected heroin, and 106 (65%) of these specified the use of black tar heroin. The botulinum toxin was type A in most cases.[1] A case control study identified subcutaneous infection of black tar heroin as a significant factor for wound botulism among IDUs.[2]

A clinician who suspects botulism should immediately call the emergency 24-hour telephone number at the Department of Public Health in his state. The state health department will contact the CDC to arrange for a clinical phone consultation and, if indicated, release of botulinum antitoxin. The CDC can be reached directly at 404-639-2206 for questions about the use of botulinum antitoxin.

Necrotizing Soft-Tissue Infections

Beginning in the mid-1990s, several California hospitals reported clusters of IDUs presenting with necrotizing soft-tissue infections. Subcutaneous injection of heroin, including black tar heroin, was common among reported cases. Wound cultures grew polymicrobial flora with *C perfringens* and *C sordellii* as the most commonly isolated clostridial species.[2]

Clostridial species produce cytotoxins that may cause tissue necrosis and shock. In a five-month period between 1999 and 2000, nine cases of necrotizing fasciitis caused by *C sordellii* were identified in Ventura County, CA. Of the eight hospitalized patients, three died with toxic shock syndrome. Older age, marked leukocytosis, and hemoconcentration significantly were associated with death.[3]

Tetanus

Since 1994, California has seen an increase in cases of tetanus among IDUs. As in wound botulism and necrotizing soft-tissue infections, these cases had the following factors in common: older age, subcutaneous injection of heroin or black tar heroin, and polymicrobial wound culture results. The distinguishing factors were the predominance of Hispanic patients and their lack of immunity to tetanus.[1] Like botulinum, tetanospasmin also blocks the release of acetylcholine at the neuromuscular synapse, but it specifically affects inhibitory motor neurons of the spinal cord, resulting in spastic paralysis and rigidity.

Early management of tetanus includes the use of benzodiazepine to control muscle spasm, and the administration of tetanus immune globulin (TIG) and toxoid. A recent, randomized trial found evidence that intrathecal injection of TIG together with intramuscular delivery, was superior to intramuscular administration alone.[4]

Efforts should be made to ensure that IDUs are up-to-date for tetanus vaccination. Clinicians taking care of IDUs should keep in mind the clostridial connection, and educate IDUs about the potentially severe and often fatal consequences of skin popping of black tar heroin.

Dr. Studemeister is an Infectious Disease Specialist with the San Jose Medical Group in San Jose, California.

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Special Feature

Neuroleptic Malignant Syndrome

By William J. Brady, MD, FACEP, FAAEM

Introduction

Neuroleptic malignant syndrome (NMS) is a disease process usually occurring in patients who use neuroleptic agents; classically, it is characterized by altered mental status, muscular rigidity, fever, and autonomic instability. The incidence of NMS ranges from 0.5% to 1% of all patients exposed to neuroleptic agents.[1,2] Men are affected twice as often as women; mean age is 40 years at syndrome onset; however, NMS can affect people of any age. NMS generally is associated with exposure to dopamine antagonist agents in the management of psychiatric illness, most commonly

antipsychotic medications (e.g., butyrophenones, phenothiazines, and thioxanthenes);[1,2,3] however, these agents may be used in different clinical settings for other indications, such as the management of emesis and vascular headache. Additionally, patients with idiopathic parkinsonism who undergo either a rapid reduction in, or cessation of, dopaminergic therapy may develop NMS.[1,4]

Etiology

While the exact pathogenesis of NMS remains unclear, reduced dopaminergic activity in the central nervous system likely plays a major role as suggested by the development of NMS with withdrawal of dopamine agonist therapy and successful treatment of NMS with L-dopa replacement therapy.[1,3] Dopamine is involved in the control of body temperature centrally in the CNS as well as peripherally at the muscular level. Neuroleptic agents block dopaminergic receptors in the hypothalamus; such receptor antagonism may produce elevations in body temperature and, if unchecked, result in systemic hyperthermia. Acute dopamine depletion also may lead to development of extrapyramidal symptoms, including muscular rigidity, which can contribute to increased heat production and further heat stress.

Neither the dosage of neuroleptic medication used nor the duration of therapy appears to be important. Particular agents, however, do have a higher associated risk. For example, high-potency neuroleptic drugs more often are encountered in the NMS patient and, therefore, are believed to have a higher risk of inducing the illness. Additionally, depot forms of these drugs seem to impart a higher risk of developing NMS than the oral or shorter-duration parenteral forms. Other risk issues for NMS include dehydration, malnutrition, physical exhaustion, intense psychomotor agitation, lithium co-therapy, and organic brain disease.[5]

Clinical Presentation

NMS typically develops rapidly over 24 to 72 hours.[2,4] The progression of symptoms most commonly starts with mental status changes followed sequentially by muscular rigidity, hyperthermia, and autonomic dysfunction.[6] The mental status almost always is impaired, ranging from agitation and rage to stupor and coma. Muscular rigidity is described as lead-pipe rigidity, similar to that seen in the patient with severe Parkinsons disease. Other motor abnormalities include akinesia, cogwheeling, fluctuating tremors, and involuntary movements.[7] Fever as high as 41°C generally follows. Autonomic changes are manifested by alterations in blood pressure and heart rate. Dehydration, most likely secondary to the patient's increased metabolic demand coupled with a reduction in oral intake, often is present and clinically significant.

Although most laboratory studies are either normal or nonspecific, several investigations may assist in establishing the diagnosis. Serum levels of muscle enzymes, especially creatine phosphokinase (CPK), often are elevated and most likely result from myonecrosis developing due to sustained muscle contractions.[2] However, at least one study has suggested that CPK elevation in in febrile, neuroleptic-treated patients is a nonspecific finding; its presence in this setting as a diagnostic criterion could lead to an overdiagnosis of NMS.[8] Elevation of liver transaminases, alkaline phosphatase, and lactate dehydrogenase levels also are found often. Electrolyte abnormalities generally will reflect underlying complications of the syndrome, including rhabdomyolysis, dehydration, and acute renal failure. Analyses of the cerebrospinal fluid as well as the results of computed tomography of the head and electroencephalography studies are often normal or nonspecifically abnormal; these studies serve to rule out life-threatening syndromes such as meningitis, encephalitis, intracranial hemorrhage, and status epilepticus.

Several different sets of diagnostic criteria for NMS have been proposed. Levenson published the diagnostic criteria that are best known and employed most widely, including major and minor descriptors. Major criteria include fever, muscular rigidity, and elevated CPK levels. Minor criteria include tachycardia, abnormal blood pressure, tachypnea, altered consciousness, diaphoresis, and leukocytosis. The diagnosis strongly is suggested by the presence of all three major criteria; alternatively, two major criteria and four minor criteria also support the diagnosis.[9]

Management

Treatment of NMS centers primarily on supportive care, coupled with removal of the inciting agent and exclusion of other diseases (e.g., meningitis and intracranial hemorrhage). Rhabdomyolysis is treated with the usual measures. Dantrolene, which has been used very successfully in treating malignant hyperthermia, has been effective in shortening the duration of illness.[10] Dosages range from 0.8-3.0 mg/kg intravenously (IV) every 6 hours—to 10 mg/kg/day; certain authorities recommend initial bolus doses of 2 mg/kg IV, repeated once as needed. It is important to note that dantrolene is hepatotoxic at levels above 10 mg/kg/day — patients should be started at lower drug dosages, and then gradually advanced as needed. Dantrolene appears to offer the most benefit in patients with pronounced muscular rigidity. The second most widely used agent, bromocriptine mesylate, is a dopamine agonist that also

has been administered alone, or in combination with dantrolene, to treat NMS. Dosage ranges from 2.5 to 7.5 mg orally (or via nasogastric tube) every eight hours. Other dopamine agonists also have been used to treat patients with NMS, including amantadine, levodopa, and carbidopa-levodopa. ❖

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CME Objectives

To help physicians:

- Summarize the most recent significant emergency medicine-related studies;
- Discuss up-to-date information on all aspects of emergency medicine, including new drugs, techniques, equipment, trials, studies, books, teaching aids, and other information pertinent to emergency department care; and
- Evaluate the credibility of published data and recommendations.

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge.

To clarify confusion surrounding any questions answered incorrectly, please consult the source material. After completing this activity, *you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

Physician CME Questions

1. In the study of GIK infusion in STEMI, the investigators found that:
   a. potassium, but not insulin or glucose, was effective in reducing mortality rates.
   b. GIK infusion reduced the rates of cardiogenic shock and nonfatal cardiac arrest.
   c. GIK infusion had no effect on 30-day mortality rates.
   d. insulin infusion decreased hypoglycemic episodes.

2. Neuroleptic malignant syndrome usually features:
   a. fever, muscular rigidity, and altered mental status.
   b. fever, altered mental status, and muscular flaccidity.
   c. hypothermia, altered mental status, and muscular rigidity.
   d. fever, cataplexy, and microhemorrhage of the pons.

3. Bulger and colleagues' study on the use of prehospital neuromuscular blockade while intubating patients suggested that these agents conferred a survival benefit and improved neurological outcome in:
   a. the mild head injury group only.
   b. victims of penetrating trauma with hypotension.
   c. victims of penetrating head trauma.
   d. all head injury victims.

4. Wound infection with *Clostridium botulinum* classically presents with:
   a. ascending flaccid paralysis with altered mentation.
   b. descending spastic paralysis with hydrophobia.
   c. cranial nerve palsies and descending flaccid paralysis.
   d. altered mental status and cranial nerve palsies.

5. In patients presenting with hip pain after blunt trauma, approximately _____ were found to have fractures on MRI after initially negative plain x-rays.
   a. 4%
   b. 16%
   c. 28%
   d. 51%

Answers:
1. c
2. a
3. d
4. c
5. a

What a Difference a Lead Makes

By Ken Grauer, MD

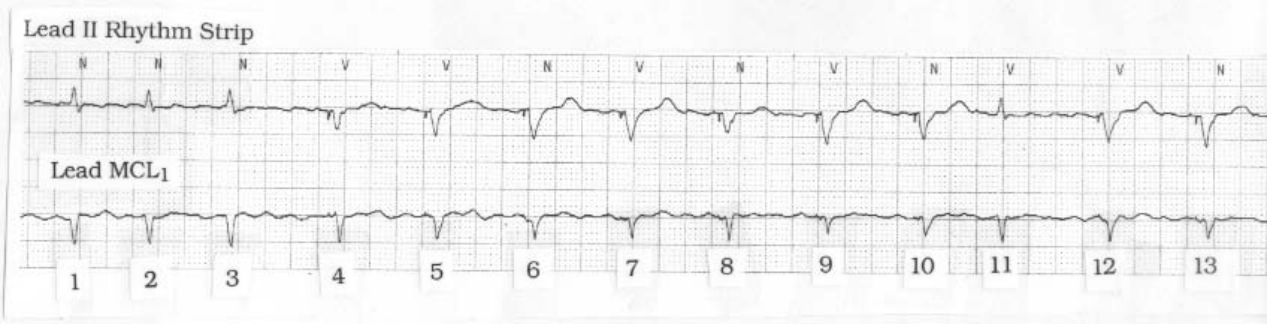

Figure: Telemetry rhythm strip obtained from a 67-year-old woman with heart failure.

Clinical Scenario: The telemetry rhythm strip shown in the Figure was obtained from a 67-year-old woman who presented with heart failure. A permanent pacemaker had been implanted a number of years earlier. Interpret the tracing initially by looking *only* at lead MCL_1. How does the addition of a second simultaneously recorded lead (lead II) help in your interpretation? How many findings can you identify on this two-lead telemetry tracing? (Hint: Some of these findings are very subtle.)

Interpretation/Answer: Accurate interpretation of this tracing would be virtually impossible if one only had access to a single MCL_1 monitoring lead. This is because the QRS complex looks similar for virtually all beats in MCL_1, and pacer spikes are practically nondetectable in this lead. This highlights the importance of viewing arrhythmias from more than the limited perspective of a single monitoring lead. Addition of lead II to our database allows identification of regular ventricular pacing spikes at a rate of 80/minute for much of the tracing (beats #4 through 10 and 12-13 are paced). Each pacer spike is followed by a QRS complex and T wave, indicating appropriate capture. Several spontaneous beats are seen on the tracing (beats #1, 2, 3, and 11). From the complete absence of P waves, the presence of fine undulations in the ECG baseline, and apparent irregularity of spontaneous beats #1, 2, and 3, the underlying rhythm appears to be atrial fibrillation. Appropriate sensing of the pacemaker is suggested by the absence of pacer spikes during the spontaneous rhythm, with appropriate return of pacer spikes following the two brief pauses that occur after beat #3 and beat #11. Note that the R-R interval preceding the pacer spikes occurring after these two pauses (i.e., the R-R interval between beats #3-4 and 11-12) is virtually the same as the R-R interval during the seven-beat sequence of consecutively paced beats (that occurs between beats # 4-10). This confirms that the pacemaker is sensing appropriately, as well as capturing the ventricles.

The final finding of interest relates to the presence of fusion beats. The importance of recognizing this finding on a pacemaker tracing is primarily so that one does not misinterpret the changes in QRS morphology that may result as indicative of ventricular ectopy or pacer malfunction. Fusion beats commonly are seen in patients with pacemakers (especially when the underlying spontaneous rhythm is atrial fibrillation) because the presence of the pacemaker itself predisposes to a situation in which some spontaneous beats are likely to occur (by chance alone) in close temporal proximity to paced impulses. The result of near simultaneous occurrence of a spontaneously occurring supraventricular impulse (from the patient's atrial fibrillation) with an impulse originating for the ventricles (from a paced beat) is a fusion complex that manifests characteristics of *both* the spontaneous beat and the paced QRS complex. Thus, the paced QRS complexes of both #4 and 5 clearly are not as wide in lead II as the other paced beats—a result of *fusing* QRS morphology of spontaneous beats with completely paced complexes. Note that the T wave in lead II of these fusion beats (i.e., the T wave of beats #4 and 5) is not as prominent as the T wave of fully paced beats #6, 7, 9, 10, 11, and 12. In addition to a fusion effect on QRS morphology, near simultaneous occurrence of a supraventricular beat and a paced ventricular complex also may produce a fusion effect on T-wave appearance. Awareness of this last point supports our suspicion that the slight alteration in QRS and T-wave morphology of paced beat #8 also reflects some degree of fusion between this paced beat and a spontaneously occurring impulse from this patient's underlying atrial fibrillation. ❖

In Future Issues:

Injuries from less-lethal weapons: Are you ready?

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Patients with maxillofacial injuries represent the very core of the specialty of emergency medicine. This group encompasses all age ranges—from the very young child to the elderly— and a spectrum in the severity of injury. The patient also may have associated injuries and medical conditions; the emergency practitioner must be aware of these injury patterns and know how to manage them.

Emergency physicians (EPs) may evaluate, manage, and discharge the patient; however, other specialists are likely to be involved in the care of the patient, and therefore, the EP also must be aware of the appropriate referral patterns.

To adequately address the complexity and breadth of maxillofacial trauma, the current issue reviews the anatomy, recognition of common injury patterns, and initial stabilization. A second issue will address specific injuries in detail, diagnostic imaging, definitive management, and appropriate consultation and disposition strategies.

— The Editor

Maxillofacial Injuries: Clinical Characteristics and Initial Management

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Introduction

Five to ten percent of all patients presenting to the emergency department (ED) do so because of a maxillofacial injury, therefore, maxillofacial trauma is an area of great importance to the practicing EP and emergency nurse.[1,2] Although physicians from other specialties (e.g., plastic surgery, otolaryngology, and oral and maxillofacial surgery) may become involved in the care of the patient with a maxillofacial injury, it is the EP who performs the initial assessment, coordinates the patient's care, and manages other injuries or medical conditions that may be present.[3]

Patients classified as having maxillofacial trauma can present with a variety of injuries, ranging from minor to life-threatening. Injuries classified as maxillofacial include 1) fractures to any of the facial bones; 2) soft tissue injuries to the face and intraoral structures, including lacerations, abrasions, and contusions; 3) dental injuries, including impaction, subluxation, avulsion, and fractures of the teeth or alveolar bone; and 4) temporomandibular joint dislocation.[4] Injuries generally not classified as maxillofacial trauma include 1) injuries to the orbits themselves; 2) intracranial conditions such as closed-head injury, subdural and epidural hematomas, and traumatic subarachnoid hemorrhage; 3) skull fractures; and 4) cervical spine injuries. These injuries certainly may occur in the patient with maxillofacial trauma, however, and

the ED practitioner also must be skilled in their management. These associated injuries in the head and neck area, as well as injuries to the other systems, such as the pulmonary system, can add greatly to the complexity and difficulty of patient management.[5]

Management of patients with maxillofacial trauma may be challenging to the ED for a variety of other reasons. This type of trauma may be associated with social or public-health issues such as child and elder abuse, interpersonal violence, alcohol-related violence, domestic violence, incorrect seatbelt and car seat use, and a lack of helmet use by bicycle and motorcycle riders.[6–18] The urgency of some of these situations demands that they be addressed at the time of ED presentation. The EP and emergency nurse also need to be cognizant of the psychological issues that may result from maxillofacial trauma (e.g., posttraumatic stress disorder and the effects of altered facial appearance).[19–21] Certainly, management of medical and surgical issues is the priority at the time of the ED visit; however, awareness of these psychological issues may be helpful in the overall management of the patient.

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Etiology

Much of the published literature regarding assessment and management of maxillofacial injuries has come from military wartime experiences. During World War I, trench warfare dominated, therefore, the majority of serious wounds sustained by infantrymen were to the head and neck.[22] These devastating facial injuries required dentists and general surgeons to review the anatomy of the face and develop innovative surgical reconstructive techniques. In 1917, the Surgeon General's Office (SGO) conducted 3- to 6-week courses to train general surgeons and dentists to treat maxillofacial wounds.[22] As a result of these wartime experiences, the SGO published abstracts in the literature, and the specialty of maxillofacial surgery began.

Although EPs seldom are called upon to care for wounded soldiers, maxillofacial injury has become a common complaint in EDs. Maxillofacial trauma most commonly is associated with motor vehicle crashes (MVCs) and assault; however, significant epidemiologic differences exist, depending upon the location of the treating facility and the mean age of the population in the catchment area.

There are almost 30 million more vehicles than licensed drivers in the United States; it is not surprising that MVCs accounted for 37,795 fatalities and more than 2 million injuries in 2001.[23] Some reports suggested that nearly 75% of these injuries involved the craniofacial structures or the cervical spine.[24] The enforcement of mandatory seatbelt laws and, more recently, the widespread incorporation of airbag restraint systems into automobiles have led to a significant reduction in MVC-related mortality; however, in persons age 35 years or younger, MVCs remain the most common cause of death or trauma.[17]

Assault is a common reason for ED visits. Domestic violence, child abuse, and altercations routinely result in facial injuries. The midface is the most common target of assailants, with injury typically resulting from a blow by a fist. The majority of injuries are isolated to the soft tissue; however, fractures of the nasal bones and mandible are quite common. Additionally, left hemifacial injuries are more common, owing to the greater prevalence of right-hand-dominant individuals.

Firearm-related injuries are the second leading cause of injury-related deaths in the United States.[25] Approximately 115,000 firearm-related injuries occur annually; roughly 10% of these injuries occur to the face.[25,26] This percentage increases to more than 50% if the injury was intentionally self-inflicted.[25] The severity of injury depends upon the caliber of the weapon, the velocity of the projectile, and the range at which the patient was shot.[27,28] Sixty-eight percent of firearm-related injuries in persons 15 to 24 years old result from interpersonal violence, while 78% of firearm injuries in older persons result from suicide attempts.[26] With the help of firearm awareness programs, firearm-related injuries and mortality have decreased significantly. Nevertheless, firearm injuries remain a devastating problem in the United States.

Approximately 22 million children are injured in the United States annually. The number of injuries surpasses all other major diseases of children.[29] Children are uniquely susceptible to maxillofacial injury because of their disproportionate cranial-body

Figure 1. Skeletal Anatomy of the Facial Area

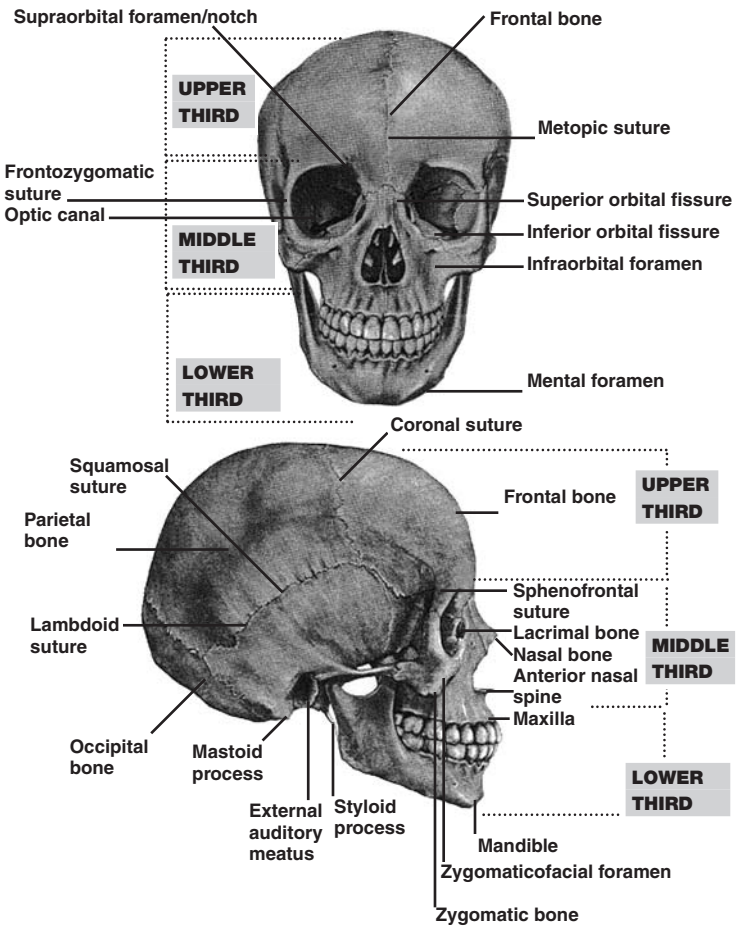

Figure 1. Frontal (A) and lateral (B) view of the skull, showing the division of the facial skeleton into thirds.

Reprinted with permission. Fonesca RJ, Walker RV, eds. *Oral and maxillofacial Trauma*. Philadelphia:WB Saunders;1991:226.

mass ratio.[29] In young children, falls are the most common cause of facial injury.[30] In older children, bicycle crashes and sports-related injuries dominate, with assault becoming more common with increasing age.[30,31]

Patients older than 65 years account for approximately 1% of maxillofacial trauma, and falls are the most common cause in this age group.[8] Each year, nearly a third of individuals older than 65 years suffer a fall.[8] In one retrospective review of 42 patients presenting to an ED with maxillofacial injury, 36 (85.7%) gave a history of falling. MVCs and elder abuse also are important causes to consider in elderly patients with facial injuries.

Anatomy

The anatomy of the maxillofacial area is quite complex, involving many organ systems. Differences exist between pediatric and adult patients, especially in dental anatomy. The ability to properly assess and manage the patient with maxillofacial trauma starts with a good understanding of the pertinent anatomy, which is best understood by division into the categories of skeletal, soft tissue, vascular, neurologic, and intraoral structures.

Skeletal. The facial area and its bones have typically been divided into upper, middle, and lower thirds (*See Figure 1.*).[32] The upper portion of the face is that area overlying the frontal bone. The frontal bone forms portions of the roof and lateral wall of the orbit. Its thickened anterior portion forms the supraorbital ridges. The supraorbital foramen is located in this area and serves as a passageway for the supraorbital nerve. The frontal sinus is contained within this bone.

The middle third of the face is the most complex and is formed by the maxilla and the zygomatic, nasal, lacrimal, palatine, vomer, and inferior nasal concha bones.[32] The maxilla is the largest bone in this portion of the face. The inferior portion of the maxilla (the alveolar process) serves as the attachment for the upper teeth. The maxillary sinus is located within the body of the maxilla. The maxilla is closely involved with the orbit, forming portions of the medial and inferior orbital rims, medial orbital wall, and orbital floor. The infraorbital foramen, containing the infraorbital nerve and artery, is located in the maxilla. The zygomatic bone forms the zygomatic arch and gives structure and shape to the cheek.[32] It articulates with the frontal, temporal, maxilla, and sphenoid bones.The nasal bones give shape to the superior portion of the nose. They articulate with the frontal and maxilla bones.[32] The lacrimal, palatine, vomer, and inferior nasal concha bones are smaller, minor bones that may be involved in maxillofacial trauma but rarely require specific management or attention when injured.

The lower portion of the face is dominated by the largest and strongest facial bone, the mandible.[32] The mandible is made up of the body and two rami. The junction between body and ramus is referred to as the angle.[33] The most anterior portion of the body is the symphysis. The body serves as the attachment for all of the lower teeth. The mental foramen, containing the mental nerve and artery, is located in the anterior portion of the body.

The superior portion of the ramus contains the condylar process and the coronoid process, which are separated by the mandibular notch. The condylar process articulates with the glenoid fossa of the temporal bone to form the temporomandibular joint. This joint contains a fibrocartilaginous disc within it and is surrounded by a fibrous capsule.[32]

Soft Tissue. The soft tissue of the maxillofacial region consists largely of skin, cartilage, and muscle. The skin of the face is attached loosely to the underlying structures, except in the area of the lower lateral cartilages in the tip of the nose.[32] The skin of the eyelids is unique because it is especially thin and contains lacrimal, sweat, and sebaceous glands.[32]

Cartilage is found in the maxillofacial region in the ear and nose. In the ear, the auricle cartilage is located just beneath the skin and gives structure to the majority of the ear, except in the inferior portion, the lobule. The shape of the nose is formed largely by the cartilage in the lower half.[32] The cartilages involved are the septal cartilage, the lateral nasal cartilage, and the greater and lesser alar cartilages.

The numerous muscles in the maxillofacial area are superfi-

cial and thus likely to be injured during trauma. The various muscles can be considered as several large groups.[32]

The muscles of facial expression constitute one group.[32] These superficial muscles move the skin when they contract. Various muscles in this group are found in the neck, scalp, nose and mouth, and surrounding the eyes. The large, strong muscles of mastication move the mandible.[32] Included in this group are the medial and lateral pterygoid, the masseter, and the temporalis muscles. This group can pull and manipulate fractures, and therefore, is important to the treating physician. The suprahyoid muscles are a group of small muscles attached to the hyoid bone.[32] They move the hyoid bone, mandible, and tongue. Individual muscles in this group are the mylohyoid, digastric, geniohyoid, and stylohyoid muscles. The last group of muscles to be considered is that of the soft palate:[32] the levator vel palatini, the tensor veli palatini, and the muscular uvulae. They act to manipulate the soft palate and uvula.

Vascular. The vascular supply to the maxillofacial region is rich and complex.[2,32,34] The arterial blood supply originates with the bilateral common carotid arteries, which, in the neck, bifurcate into the internal and external carotid arteries.

The internal carotid artery continues through the neck with no branches. It enters the cranial fossa and serves largely to provide the brain with blood. Of importance is that the internal carotid artery will supply blood to the eye and eyelids, the upper face, and the nasal cavity through several branches. With the exception noted above, the majority of blood supplied to the maxillofacial region arrives via the external carotid artery.[32] The external carotid artery gives off branches of the superior thyroid, lingual, facial, occipital, posterior auricular, and ascending pharyngeal arteries before terminally branching into the superficial temporal and the maxillary arteries.[32]

All venous blood drainage from the maxillofacial region occurs ultimately via the internal jugular vein, before it joins the subclavian vein.[32] Superficial areas of the face are drained by the external and anterior jugular veins. The internal jugular vein drains the deeper structures.

Neurologic. The cranial nerves are numerous and complex; however, it is possible to concentrate on two in the patient with maxillofacial trauma—cranial nerves V and VII.[32]

Cranial nerve V (the trigeminal nerve) has both sensory and motor function in the maxillofacial region. The sensory function is provided by three divisions: the ophthalmic nerve, the maxillary nerve, and the mandibular nerve, which are responsible for sensation in the upper, middle, and lower portions of the face, respectively. One of the more notable branches of the maxillary nerve is the infraorbital nerve, which passes through the infraorbital foramen to provide sensation to the mid-portion of the face. An important branch of the mandibular nerve is the inferior alveolar nerve, which provides sensation to the mandibular teeth. The motor function of the trigeminal nerve is provided by branches of the mandibular nerve, which innervate the mastication muscles.

Cranial nerve VII, the facial nerve, has a largely motor function as it innervates the muscles of facial expression.[32] This nerve has five branches—temporal, zygomatic, buccal, mandibular, and cervical—that travel to the various muscles of the face and neck.

Clinical Features and Evaluation

Prehospital Care. Emergency medical service (EMS) personnel routinely are called upon to care for patients with maxillofacial injury. As the point of first care, paramedics and emergency medical technicians perform critical interventions that can greatly influence patient morbidity and mortality. A careful, stepwise approach, including assessment of airway integrity, cervical spine immobilization, and control of ongoing bleeding, is important for patient survival.

Airway compromise is common with maxillofacial trauma and should be the primary concern of prehospital personnel. Initial assessment of injury severity can be misleading, necessitating vigilance, as airway compromise can occur rapidly due to aspiration of blood and gastric contents or from the progression of previously unrecognized injuries. Supplemental oxygen administered by facemask may be sufficient for patients with minor injuries. Adequate ventilation using bag-valve-mask (BVM) may be difficult to achieve, owing to disruption of anatomy and loss of structural support. Airway obstruction from posterior displacement of the tongue resulting from loss of anterior support is common with bilateral mandibular fractures and may require placement of an oral airway in the patient with an absent gag reflex.[35–37] In patients with maxillofacial trauma, the oral airway should be inserted with the concavity toward the tongue after displacement of the tongue with a tongue depressor instead of the alternative method of insertion followed by 180º rotation. In general nasal airways should by avoided in patients with midfacial injury; it is not possible to visualize the entire path that the nasal airway will traverse.

The gold standard for definitive airway management by EPs remains orotracheal intubation. The safety of out-of-hospital rapid sequence intubation (RSI) performed by paramedics was questioned by researchers in response to the high incidence of transient hypoxia and bradycardia experienced by patients with severe closed head injury (Glasgow Coma Scale [GCS] score < 8) during intubation.[38] This concern was based upon the finding that 84% of patients who developed hypoxia during RSI performed by prehospital care providers had initial oxygen saturation measurements in the normal range. However, because of anatomic problems associated with facial fractures, successful airway management with BVM ventilation may not be possible.[5,36,37,39,40] Therefore, if airway protection is indicated in the field, out-of-hospital RSI should be attempted. Additionally, a recent study found that Combitube insertion as an airway salvage maneuver was highly successful after multiple failed out-of-hospital orotracheal intubation attempts.[41] In general, blind nasotracheal intubation or nasogastric tube insertion should be avoided because of the possibility of intracranial tube placement associated with fractures of the cribiform plate or nasoethmoid complex.[24,37,42,43]

Cervical spine injury associated with maxillofacial trauma is

well documented in the literature. An estimated 1–7% of patients with facial injuries have concomitant cervical spine injury.[1,32,36,39,40] In a five-year review of 151 multitrauma patients with severe maxillofacial injuries (Injury Severity Score ≥ 12; mean, 21.4) presenting to an urban Level I trauma center, the incidence of cervical spine injury was 7.3%.[5] Therefore, before moving a patient with any likelihood of cervical spine injury, prehospital care providers should immobilize the patient's neck with a semi-rigid cervical collar and a backboard with lateral head restraints. Care also should be taken to secure the patient's body to the backboard in several places during transport to minimize cervical motion.

Bleeding from facial injuries can be brisk because of the extensive vascularity of the face and scalp and may cause life-threatening airway obstruction or hypovolemia. The reported incidence of severe bleeding varies greatly—from 1.25% to as high as 24.4%.[44] Although bleeding from isolated facial injury rarely is significant enough to produce hemorrhagic shock in adults, children may experience massive blood loss and shock secondary to their smaller total blood volume. Initial control of bleeding from soft-tissue sources is best obtained by direct pressure when possible. Bandages with large quantities of gauze do little to slow bleeding and may mask the amount of blood loss. Persistent oral or nasal bleeding is common in patients with midfacial fractures. Ongoing nasopharyngeal hemorrhage may require placement of anterior nasal packing with petroleum gauze and unilateral or bilateral posterior nasal pressure balloons with a Foley catheter.[44] Management of ongoing soft-tissue hemorrhage may require placement of interlocking sutures for temporary control.

Prehospital management also includes establishment of intravenous access and initiation of isotonic fluid resuscitation. Early volume resuscitation, commonly with normal saline or lactated Ringer's solution, is vital for circulatory support and may prevent transient hypotension upon induction of anesthesia for RSI.

ED Care. After life-threatening conditions have been stabilized, a thorough history should be obtained from the patient, available witnesses of the incident, and EMS personnel. Obtaining an accurate history may be difficult, but details of the patient's medical and surgical history, current medications, and allergies should be elicited if possible. A detailed description of the mechanism of injury will guide further evaluation. High-speed incidents, such as a MVCs, are likely to produce associated head and cervical spine injuries.[33] Closed head injury should be suspected whenever a history of decreased level of consciousness or alcohol involvement is obtained.[33] A detailed description of vehicle damage may increase suspicion for concomitant injuries. The use of seat belt restraints or the deployment of airbags is important, because the presence of these devices alone or in combination is associated with less severe maxillofacial injuries.[17,18] It is essential to question women regarding the possibility of domestic-violence–related injury and potential pregnancy. Though less critical for initial management, the patient's tetanus immunization status should be determined.[33]

Table 1. The LEMON Law

L: LOOK EXTERNALLY

- Short, muscular neck
- Protruding upper incisors
- Receding mandible
- Full dentition
- High-arched palate
- Severe facial trauma

E: EVALUATE THE 3-3-2 RULE

Describes the ideal external dimensions of the airway:

3 — The opening of the jaw should be large enough to accommodate three fingers (3-4 cm).

3 — The distance from the mentum to the hyoid bone should be at least three fingerbreadths.

2 — The distance from the floor of the mouth to the thyroid cartilage should be at least two fingerbreadths.

M: MALLAMPATI CLASSIFICATION

- Mallampati Class I: no difficulty; soft palate, uvula, fauces, pillars visible
- Mallampati Class II: no difficulty; soft palate, uvula, fauces visible
- Mallampati Class III: moderate difficulty; soft palate, base of uvula visible
- Mallampati Class IV: major difficulty; hard palate only visible

O: OBSTRUCTION

- Blood in the upper airway
- Expanding hematoma
- Swelling of intraoral structures
- Foreign body
- Abscess
- Laryngeal edema

N: NECK MOBILITY

- Inability to flex or extend the neck (due to cervical collar, arthritis, etc.)
- Cervical spine injury

Adapted from Kaide CG, Hollingsworth JC. Current strategies for airway management in the trauma patient. Trauma Rep *2004; 2:3-4.*

Initial evaluation in the ED should proceed according to standard trauma evaluation protocol (Table 1). If the cervical spine is not immobilized already, an assistant should maintain in-line manual stabilization while the physician applies a semi-rigid collar.

The primary survey is performed to ensure integrity of the airway, breathing, and circulation (ABCs). Careful assessment of airway integrity begins by asking the patient his or her name. Inspect the mouth and oral pharynx to assess intraoral bleeding, which can lead to aspiration of blood and airway compromise. Direct laryngoscopy to confirm correct endotracheal tube placement is necessary if the patient was intubated in the field. Inspection of the patient's chest wall should include evaluation for symmetric chest rise. Auscultation of the lungs assesses air movement for bilateral equality. Tracheal position should be noted and tracheal auscultation performed. The presence of ecchymosis or stridor over the laryngeal cartilages may indicate significant upper airway injury, necessitating further management.

Tachypnea should be considered a sign of distress and requires careful monitoring. Tachycardia may be present as a physiologic attempt to maintain cardiac output in the setting of ongoing hemorrhage. Hypotension is a late finding indicating failed compensatory mechanisms. The presence of any gross neurologic deficits should be noted in addition to the calculation of the GCS score. Finally, the patient should be exposed completely, removing all clothing and accessories.

Direct laryngoscopy is the mainstay of emergency airway management. EPs routinely perform endotracheal intubation with great success. Recent literature suggests that EPs and anesthesiologists are equally successful in managing trauma patients' airways.[45] Failed intubation rates typically are less than 1% under ideal conditions.[46,47]

Although airway management in patients with facial trauma can be challenging, securing a definitive airway is crucial for patient survival. Aspirated blood, foreign bodies, displaced fractures, or direct trauma can compromise the airway. Conditions such as cervical spine injury, alcohol intoxication, or a full stomach may further complicate airway management.

Most emergency airways in the ED are managed by RSI. In the cooperative patient, the oral pharynx and facial anatomy should be examined to identify clues that may signify a difficult intubation using direct laryngoscopy, such as active intraoral bleeding or dental injuries. If obtained, a history of difficult intubations may be one of the strongest predictors of airway difficulty.[48] Assessment of the distance between the upper and lower incisors as well as estimation of the distance from the inferior tip of the chin to the top of the hyoid bone may alert the laryngoscopist to a difficult airway. (*See Table 1.*)

RSI proceeds with selection of an induction agent and paralytic agent. A paralytic agent may be unnecessary in a patient with a depressed level of consciousness. An adequate bedside rigid suction catheter is essential for successful intubation in patients with maxillofacial trauma. Direct laryngoscopy followed by endotracheal tube placement with primary and secondary confirmation of correct position completes RSI. Methods of secondary confirmation include use of an end-tidal carbon dioxide detector or esophageal detector. External laryngeal manipulation of the thyroid cartilage with the laryngoscopist's right hand (bimanual laryngoscopy) may improve visualization of the glottic opening if difficulty is encountered.[46]

Appropriate preparation ensures that a backup plan is available should RSI fail. Commonly used airway salvage devices include the 1) intubating laryngeal mask airway (LMA), 2) esophageal-tracheal Combitube, 3) gum elastic bougie, and 4) fiberoptic bronchoscope. Each device has advantages and disadvantages, but all have been used successfully in patients with maxillofacial injury.[42,47,49,50] One or all of these devices should be within reach at all times when performing RSI.

In certain situations, the severity of the injury may necessitate establishing a surgical airway. In a review of 92 patients with maxillofacial gunshot injuries, 20 patients had a threatened airway on presentation to the ED. Of these 20 patients, 12 (60%) were intubated successfully, while eight (40%) required immediate surgical airways.[51] Of the 72 patients initially evaluated as having a normal airway, 11% later required some form of emergency airway management, highlighting the importance of early airway protection.[51]

After completion of the primary survey and, if necessary, definitive airway management, the practitioner's focus should turn to the secondary survey, with particular attention to the facial complex.

Soft Tissue Injuries. Evaluation and management of soft tissue injuries of the face deserve special attention. The importance of the delicate structures of the maxillofacial complex is not limited to physiologic function. The face is one of the most aesthetically appealing structures of the human body. The negative effect of facial deformities on social functionality is well documented.[21] A thorough examination of all soft tissue injuries will minimize factors that may contribute to a poor aesthetic outcome. Soft tissue injuries of the face may result in a sensory or motor deficit if the trigeminal or facial nerve is involved (*See Anatomy section.*). The location of the injury in relation to the origin of the facial nerve will dictate the feasibility of microscopic nerve repair.

The extent of wound contamination should be recorded; the presence of a foreign body greatly increases infection risk. Inspect all wounds cautiously; sharp foreign bodies pose a threat not only to the patient but to the health care worker as well. Extensive wound contamination ("road rash") may result in traumatic tattooing if the wound is not scrubbed completely clean of all foreign particles. Failure to remove foreign bodies may result in discomfort and disfiguring scars. Carefully inspect the scalp. Matted hair with dried blood can make identification of underlying lacerations or abrasions difficult. Descriptive features of lacerations to the scalp include location, length, depth, and the presence or absence of ongoing bleeding. The presence of a bony step-off indicates a skull fracture.

Laceration to the orbital ridge or eyelid may result from blunt trauma. Inspect the wound to rule out the presence of periorbital fat. If present, the laceration should be considered to involve the orbital space, therefore, ophthalmologic consultation is needed. Other eyelid injuries that warrant consultation include 1) lacerations extending through the tarsal plate, 2) lacerations that prevent the patient from opening the eye, signifying involvement of the levator palpebrae superioris muscle or tendon, and 3) any potential injury to the lacrimal system.[51]

Inspect lacerations to the cheek for parotid duct or facial nerve injury. Stenson's duct may be considered intact if parotid gland massage produces clear fluid from the parotid papilla located on the buccal mucosa opposite the upper second molar. Failure to express clear glandular fluid or the expression of blood with massage is indicative of duct injury. Surgical consultation is needed for injuries to the parotid duct or facial nerve injury resulting from lacerations located lateral to a vertical line running through the lateral canthus of the eye.[51]

Lacerations of the nose or ear can be particularly difficult to manage. There is little laxity in the skin of these cartilage-bearing structures. Inspect the nasal cavity for the presence of an intranasal laceration or septal hematoma. Lacerations extending

Figure 2. Classification of Tooth Fractures

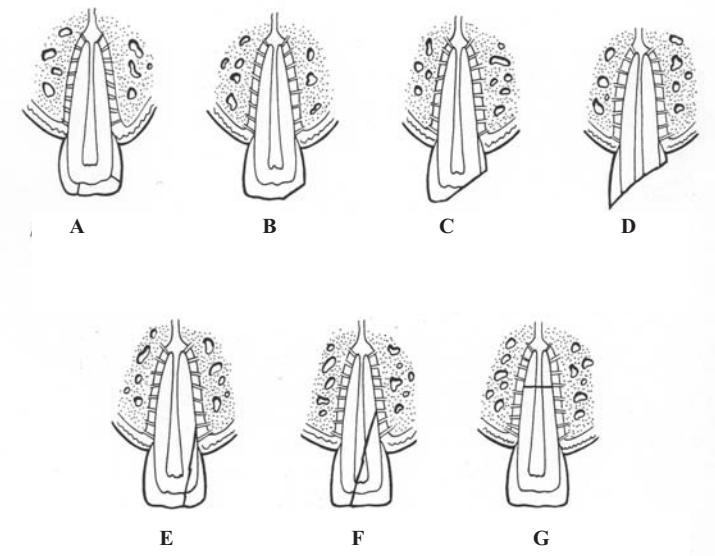

Figure 2. Dental injuries. A: crown infraction. B, uncomplicated crown fracture (Ellis I). C: uncomplicated crown fracture (Ellis II). D: complicated crown fracture (Ellis III). E: uncomplicated crown-root fracture. F: complicated crown-root fracture.

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through the auricular cartilage require special repair techniques to avoid a poor result. Auricular hematomas must be noted; surgical drainage is required to prevent cauliflower deformity and infection.

Intraoral soft tissue injuries may occur with maxillofacial injuries. Small lacerations of the tongue generally heal well without intervention. Large lacerations or those involving the tongue margins require a layered repair to provide hemostasis and preserve function. Lacerations of the lip extending through the vermillion border or involving the philtrum may warrant plastic surgery consultation for repair.

Dental Injuries. Injuries to the maxilla, mandible, and adjacent soft tissues commonly are associated with dentoalveolar injuries. Careful inspection of the dentition and gingival mucosa utilizing a gloved finger, a pair of tongue depressors, and a bright light source will identify most dental injuries.[52] Account for all of the patient's teeth; avulsed teeth may be displaced into adjacent soft tissues or become aspirated, swallowed, or simply lost at the scene.[36] Chest and abdominal radiographs may be necessary to locate missing teeth.

Examination of the oral cavity begins with inspection. Remove all blood and debris from the oral cavity prior to examination. Record the location and size of soft-tissue injuries. The presence of an empty socket may indicate complete avulsion of the tooth. Gingival bleeding may occur as a result of laceration from an underlying fracture.

Ask the patient to close his teeth together to evaluate occlusion. Abnormalities of occlusion indicate an underlying traumatic injury. Each tooth is then palpated to evaluate for mobility. Abnormal mobility may indicate a subluxation (nondisplaced) injury or luxation (displaced) injury of the involved tooth.

Perform percussion of each tooth. Sensitivity elicited by percussion suggests a concussion injury in which the tooth generally is not mobile or displaced, but has sustained an injury.[53]

Fractures of teeth can be classified as *crown fractures* (involving the portion of the tooth above the gingival surface) or *root fractures* (involving the portion of the tooth below the gingival mucosa) (See Figure 2.). The Ellis classification system commonly is used to describe crown fractures. Ellis I fractures involve the enamel only; the tooth appears normal in color but has an obvious irregularity in the enamel surface. Ellis II fractures involve the enamel and dentin; the fractured surface may reveal a yellow center from exposed dentin surrounded by enamel. Also, the patient may complain of pain or sensitivity of the involved tooth. An Ellis III fracture involves the enamel, dentin, and pulp; bleeding from the pulp may be observed. Bacterial contamination of the pulp can result in necrosis of the pulp, necessitating endodontic therapy. Root fractures represent 0.5–7% of dentoaveolar trauma in permanent teeth. Suspect injury to the root when bleeding from the gingival sulcus is observed; however, these injuries are best detected with radiographs.[53]

Mandibular Fractures. The mandible is one of the strongest of the facial bones; but, because of its prominent position on the face, it is one of the most frequently fractured.[4,32,37,54] When taking the history from a patient with a suspected mandibular fracture, pay particular attention to the mechanism of injury; various types of mechanisms can cause specific injuries. During an MVC, the force generally is directed to the patient's chin, which may cause fractures of the condylar and symphysis areas.[36] Injuries from interpersonal violence are most likely to be in the left angle because most assailants are right-handed. MVC victims tend to have multiple or comminuted fractures because of the large forces involved, compared with assault victims who tend to have single, non-displaced fractures.[32]

The three questions presented below should be asked of every patient with a suspected mandible fracture; answers will guide the clinician in further evaluation.[36]

"Does your bite feel normal?" Any change in the patient's occlusion makes a mandibular fracture very likely.[32] It is the patient's impression of a change that matters. Patients are very sensitive to very small changes from their normal bite.

"Is your lip or chin numb?" Decreased or absent sensation in these areas is likely the result of involvement of the inferior alveolar nerve, which runs through the ramus, angle, and body of the mandible before exiting through the mental foramen. Numbness on one side localizes the fracture to one of the segments of the mandible, on the same side.[36]

"Does your jaw hurt when you open and close your mouth?" Mandible fractures are especially painful while opening and closing the mouth, because of distraction of the fracture segments.[36] In addition, a patient with a mandible fracture may be unable to open or close his or her mouth fully.

Figure 3. Le Fort I Fracture

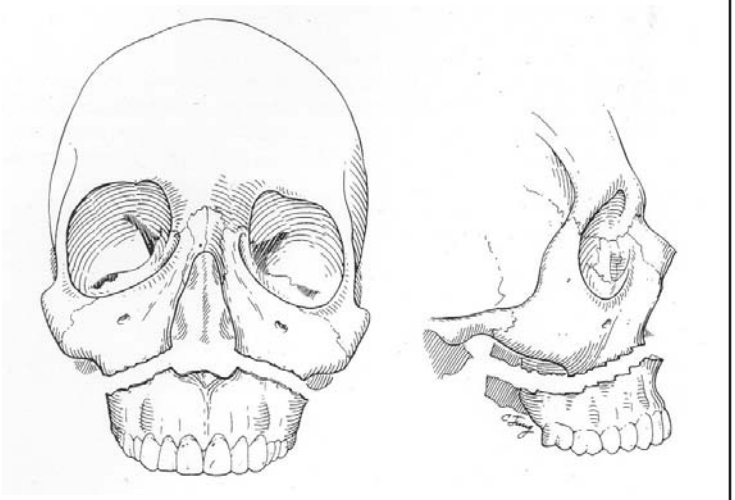

Figure 3. Le Fort I fracture involves separation of the maxillary alveolus from the upper midface at the level of the piriform aperture.

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Figure 4. Le Fort II Fracture

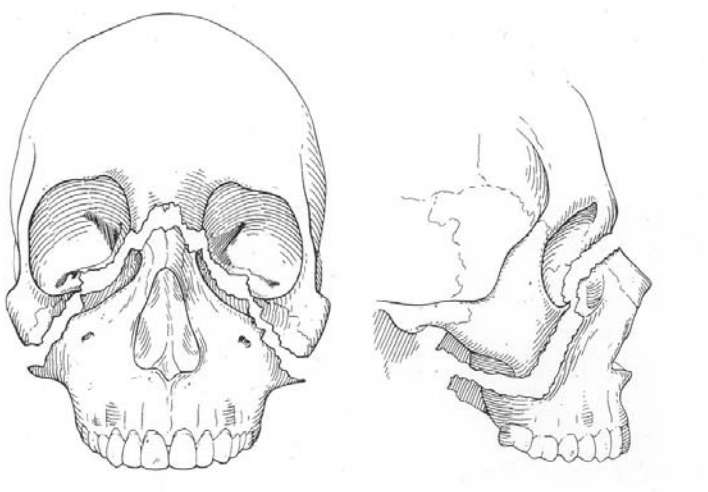

Figure 4. Le Fort II fracture occurs when a central nasomaxillary segment is separated through the inferior orbital rims and nasofontal junction.

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Physical examination of the patient starts with inspection of the external portion. Asymmetry of facial appearance can be a clue to the presence of a fracture. The mandible is a relatively superficial bone, and its borders generally can be palpated quite well for areas of tenderness, swelling, or step-offs. An important part of the examination is inspection of the external auditory meatus. At times the condyle can be displaced posteriorly and may disrupt the skin of the external meatus.[36] After the external meatus is inspected, palpation may be performed by inserting a fingertip and pressing lightly in an anterior direction.[36] This action can detect injury to the temporomandibular joint.

Next examine the intraoral area. Carefully inspect the lower teeth for any gap or step-offs between them; if noted, a mandible fracture is present.[36] Hematomas on the jaw or floor of the mouth commonly are associated with mandible fractures. Careful palpation of the mandible may reveal swelling, tenderness, or sharp step-offs of the normally smooth mandible. The physician can use both hands to grasp the mandible and check for areas of mobility. Ask the patient to open and close his or her mouth and examine the bite for malocclusion.

Mandible fractures in the condylar and subcondylar area can be difficult to diagnose. Patients with this type of injury can present with malocclusion but no other evidence of a fracture. However, on opening the mouth, deviation of the jaw to the side of the fracture is evident.[36]

Maxillary Fractures. Maxillary fractures occur less commonly than mandibular fractures. They generally are caused by mechanisms of injury involving greater amounts of force, involve more facial edema, and likely are associated with other midface fractures.[36,37]

The Le Fort classification classically has been used to describe maxillary fractures. The Le Fort I fracture is the least severe and involves separation of the maxillary alveolus from the upper midface at the level of the piriform aperture (*See Figure 3.*). In the Le Fort II fracture, a central nasomaxillary segment is separated through the inferior orbital rims and nasofrontal junction (*See Figure 4.*). The Le Fort III fracture has a fracture line at the upper portion of the zygomaticofrontal junction, orbital floors, and nasoethmoid area (*See Figure 5.*). These fracture patterns often may be mixed; for example, a Le Fort III can be combined with a Le Fort I. Another variation is an asymmetric pattern with, for instance, a Le Fort II fracture on one side and a Le Fort III on the other.

The history and physical examination follow the same basic approach as in the patient with a suspected mandible fracture. The patient with these fractures often reports malocclusion. Facial numbness can be a clue; however, in this case the numbness involves the upper lip, side of the nose, or upper gingiva because of the involvement of the infraorbital and superior alveolar nerves.[36]

The physical examination consists of inspection for swelling, abrasions, lacerations, echymoses, and asymmetry. The maxillary bone is palpated, especially along the orbital rims and the bridge of the nose.[36] Instability is assessed by grasping and attempting to manipulate the anterior maxillary teeth. Any movement of the maxilla indicates a fracture. The level of the movement can give a preliminary indication of the classification of the fracture. With a Le Fort I fracture, only the central portion of the maxilla should be mobile. In a Le Fort II, the bridge of the nose will move along with the maxilla, and in a Le Fort III, the bridge of the nose, infraorbital rims, and zygomas will show movement.[36]

Figure 5. LeFort III Fracture

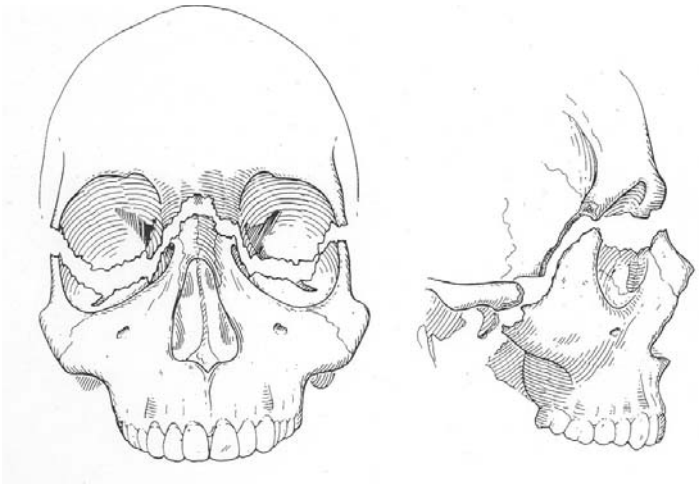

Figure 5. Le Fort III fracture consists of a fracture through the upper portion of the zygomaticofrontal junction, orbital floors, and nasoethmoid area. On physical exam, the bridge of the nose, infra-orbital rims, and zygoma may show movement.

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Figure 6. Orbital Blow-out Fracture

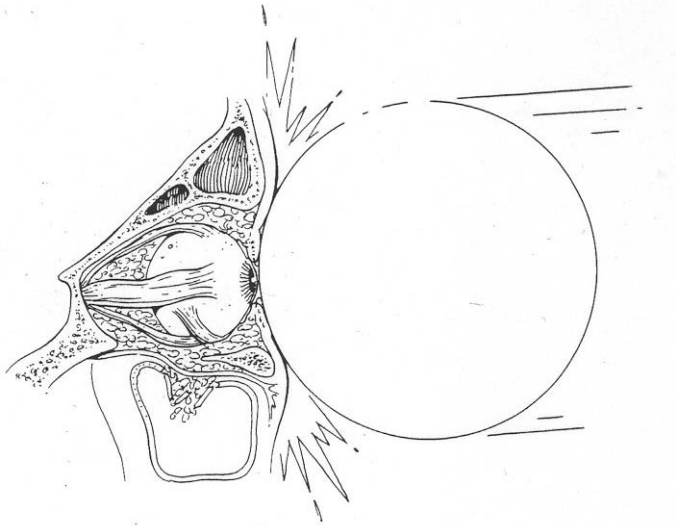

Figure 6. Orbital blow-out fracture. A direct blow to the orbit may result in fractures to the bones surrounding the orbit.

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Zygoma Fractures. The zygoma is one of the most frequently fractured facial bones, because of its prominent position in the facial skeleton.[36,37] There are several classification schemes for zygoma fractures; many are complex and of little use to the EP. One general scheme of classification that is useful is the broad division into zygomatic and zygomatic complex fractures.[32]

A zygomatic fracture involves just the zygoma bone itself, such as an isolated zygomatic arch fracture. A zygomatic complex fracture involves the zygoma itself as well as parts of the surrounding four bones with which the zygoma articulates—the temporal, orbital, maxillary, and frontal.[32,36] The zygoma forms a significant portion of the lateral and interior walls of the orbit as well as portions of the maxillary sinus,[36] thus, fractures of the zygoma can disrupt these surrounding structures.

During the history, direct attention to several areas. Numbness of the lower eyelid or upper mouth is again important, as the infraorbital nerve may be involved in a zygomatic complex fracture.[36] Painful opening of the mouth may be present because of the attachment of the masseter muscle to the zygomatic body and arch.[36] Finally, a report of binocular diplopia is important, because it commonly accompanies zygomatic fractures.[32]

Physical examination often can be less revealing with this type of fracture because of large amounts of swelling and edema that may accompany it. However, there are several important findings on physical examination. Because of the prominent position of the zygoma in the face and the large role it plays in giving shape to the midface, a depressed fracture of the zygoma often causes a flattening of the malar area, which is evident despite the edema. Bleeding from the tissue disruption in a zygomatic fracture may cause preorbital or subconjunctival ecchymosis or epistaxis.[32] When the patient complains of diplopia, examination may reveal limitations of extraocular movements, which are caused by edema or muscle entrapment.

Orbital Fractures. Orbital fractures can be caused in two ways. An orbital blowout fracture results from a direct blow to the orbit; the resulting hydraulic pressure of the compressed orbital tissues fractures the bone surrounding the orbit (*See Figure 6.*). This is likely to occur in the medial aspect of the orbital floor, which is the weakest area of orbital bone. As a result, orbital contents can herniate into the maxillary sinus, resulting in enophthalmos or limitation of eye movements and diplopia with upward gaze. The other type of orbital fracture is caused by direct trauma to the orbital rim, resulting in a fracture of the involved bone.

During the history, any complaints related to abnormal vision should be sought, including diplopia, blurry vision, loss of vision, or change in field of vision. Again, numbness of the lower eyelid or upper lip is important because the infraorbital nerve may be involved.

Physical examination should focus on the orbit and the surrounding orbital rim. A complete ocular and funduscopic exam should be performed. It is important to detect any ocular injury such as hyphema, corneal or globe lacerations, impaired visual acuity or extraocular movements, and proptosis. One third of patients with a comminuted zygomatic complex fracture have a severe ocular injury.[36] Careful palpation of the orbital rim will reveal any step-offs or localized tenderness.

Nasal and Nasal-Orbital-Ethmoid Fractures. Nasal and nasal-orbital-ethmoid fractures are considered together here, but they should be differentiated clearly from each other, because they differ in severity.

Nasal bones are fractured frequently because of their prominent position and the minor force needed to fracture them. However, they are generally minor injuries, therefore, some patients may not seek evaluation or treatment. Nasal injuries can involve the nasal bones, cartilage, or both.

A nasal-orbital-ethmoid fracture is more severe and involves not only the nasal bones but also portions of the frontal, maxillary, nasal, and ethmoid bones.[36,37] Fractures can involve the posterior wall of the frontal sinus and result in dural tears with cerebrospinal fluid rhinorrhea or brain tissue injury.

The physical examination starts with inspection of the nose and surrounding area. Observe the nose for symmetry and swelling. One important portion of the examination is assessment of the intercanthal distance (i.e., the distance between the medial commissures of the eyelids). It can be increased in nasal-orbital-ethmoid fractures due to the positional disruption of the medial canthal tendon; this distance normally measures 30–34 mm.[36]

Inspect the nose internally with a nasal speculum to look for bleeding, lacerations, or septal hematomas. Palpation of the nose may reveal tenderness, step-offs, mobility, or crepitus.

Temporomandibular Joint Dislocation. Dislocation of the temporomandibular joint (TMJ) may be caused by trauma, generally by a blow to the chin while the mouth is open. It also may occur from a non-traumatic mechanism such as yawning, laughing, laryngoscopy, or prolonged dental work.[36,37]

The TMJ is a hinge and sliding joint. During normal mouth opening, the condyle rotates and translates forward to the area of the temporal eminence. In a dislocation, the condylar head slides farther forward and becomes locked anterior to the temporal eminence. Trismus and spasm of the masticatory muscles prevent the condyle from sliding back. A TMJ dislocation may occur in a unilateral or bilateral pattern.

Obtaining a history from a patient with a TMJ dislocation can be difficult because he may have difficulty speaking; however, any history of trauma or an inciting event should be sought. The possibility of a dystonic reaction should be considered. TMJ dislocation may be a chronic problem in some patients, so it is important to ask about any prior occurrences.

A TMJ dislocation usually is evident on physical examination. The patient will present with difficulty talking and inability to close the anterior teeth. The patient with bilateral TMJ dislocations will present with a symmetric open anterior bite; in a unilateral dislocation, the jaw will be deviated to the unaffected side.

Palpation of the TMJ area may reveal the condylar head in an abnormal position below the zygomatic arch. If the dislocation resulted from trauma, the remainder of the mandible, intraoral, and facial areas should be inspected carefully and palpated to assess for an accompanying fracture.

Conclusion

ED physicians commonly are confronted with patients who have sustained maxillofacial trauma through a variety of mechanisms. A comprehensive understanding of anatomy and special considerations for the initial stabilization of these patients, such as airway management and cervical spine stabilization, allows the ED physician to confidently manage even the most critically injured patient. The next issue will focus on imaging considerations and techniques, definitive management, consultation and disposition decisions, and special circumstances for patients who have sustained maxillofacial trauma.

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CE/CME Objectives

Upon completing this program, the participants will be able to:

a.) recognize or increase suspicion for traumatic injuries that present to the emergency department;

b) describe the various modalities used to identify different traumatic conditions covered in the newsletter;

c) describe how to correctly and quickly stabilize, and then to manage patients with the particular condition covered in the newsletter; and

d) identify both likely and rare complications that may occur with traumatic injuries.

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

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CE/CME Questions

1. Which anatomic portion of the face is the most complex?
   A. Intraoral
   B. Upper third
   C. Middle third
   D. Lower third

2. A patient with a possible maxillary fracture is being examined. When the anterior maxillary teeth are grasped and a distracting force is applied, movement of the central portion of the maxilla and the bridge of the nose is noted. The infraorbital rims and zygomas do not move. What type of fracture is likely?
   A. Zygomatic complex
   B. Le Fort I
   C. Le Fort II
   D. Le Fort III

3. Blind nasotracheal intubation should be avoided in patients with:
   A. a soft-tissue auricular injury.
   B. a dental fractures.
   C. a mandibular fracture.
   D. a nasoethmoid complex fracture.

4. Examination of an injured tooth shows a fracture line with a small amount of bleeding. The dental injury is most likely an:
   A. Ellis class I fracture.
   B. Ellis class II fracture.
   C. Ellis class III fracture.
   D. subluxation injury.

5. A patient presents after facial trauma with difficulty talking and inability to close the anterior teeth. The jaw is deviated to the patient's right. Which of the following is most likely present?
   A. TMJ syndrome
   B. Bilateral TMJ dislocation
   C. Left TMJ dislocation
   D. Right TMJ dislocation

6. A 25-year-old male presents following an altercation. The patient has a very swollen orbital area, enopthalmos, limitation of eye movement, and diploplia with upward gaze. Which of the following types of fractures is this patient most likely to have?
   A. Zygoma fracture
   B. Orbital blow-out fracture
   C LeFort I fracture
   D. LeFort II fracture

7. Which of the following arteries provides blood flow to the eyes, eyelids, upper face, and the nasal cavity?
   A. Internal carotid artery
   B. External carotid artery
   C. Superior thyroid artery
   D. Ascending pharyngeal artery

8. Which of the following nerve and function pairs is matched incorrectly?
   A. Infraorbital nerve - sensation to the midportion of the face
   B. Inferior alveolar nerve - sensation to the mandibular teeth
   C. Maxillary nerve - mastication muscles
   D. Cranial nerve VII - muscles of facial expression

9. Which of the following is *not* true regarding maxillofacial injuries?
   A. Airway obstruction from posterior displacement of the tongue resulting from loss of anterior support is common with bilateral mandibular fractures and may require placement of an oral airway in a patient without a gag reflex.
   B. In patients with mandibular fractures, the oral airway should be inserted with the concavity toward the tongue after displacement of the tongue with a tongue depressor.
   C. An estimated 1-7% of patients with facial injuries also have cervical spine injuries.
   D. ED physicians have failed intubations rates of about 20%.

10. A 36-year-old male presents with a facial injury. The patient has swelling of the face, a flattening of the malar area and subconjunctival ecchymosis. Which of the following injuries is most likely?
    A. LeFort I fracture
    B. Orbital rim fracture
    C. Depressed fracture of the zygoma
    D. LeFort III fracture

Answers:
1. C; 2. C; 3. D; 4. C; 5. C; 6. B; 7. A; 8. C; 9.D 10. C

In Future Issues: **Maxillofacial injuries: imaging, management, and referrals**