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УО «Витебский государственный медицинский университет»

ЧЕЛЮСТНО-ЛИЦЕВАЯ ТРАВМА

MAXILLOFACIAL TRAUMA

учебно-методическое пособие

Рекомендовано учебно-методическим объединением по высшему медицинскому, фармацевтическому образованию в качестве учебно-методического пособия для студентов учреждений высшего образования, обучающихся по специальности 1-79 01 07 «Стоматология»

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Учебно-методическое пособие «Челюстно-лицевая травма = Maxillofacial trauma» содержит информацию о травматических повреждениях зубов, мягких тканей, костей челюстно-лицевой области и их осложнениях на английском языке. Предназначено для студентов стоматологического факультета, магистрантов, аспирантов, клинических ординаторов, слушателей факультета повышения квалификации и переподготовки кадров.

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TOPIC: FACIAL SOFT TISSUE TRAUMA

Soft Tissue Injuries

Soft tissue injuries are one of the most common problems treated in the emergency rooms and the facial region is an esthetically sensitive area with a complex variety of tissues. Around one-third of patients presenting with oral injuries have sustained soft tissue injuries.

Soft tissue in the oral and maxillofacial region absorbs a lot of energy when the region is subjected to impact from trauma. This may result in various injuries such as contusions, abrasions, lacerations and tissue avulsion. Foreign bodies are often found in the tissue. Teeth often penetrate the soft tissue and parts of the teeth may be found as foreign bodies in lacerations. Correct emergency treatment is a prerequisite for uneventful healing and the oral and maxillofacial region is an esthetically sensitive area. Failure to remove foreign bodies may result in permanent tattoos and anesthetic scarring and delayed healing and infection.

Types of soft tissue trauma

Soft tissue injuries can be divided into the following types of injuries:

- Contusion
- Abrasion
- Laceration
- Tissue avulsion.

Contusion

A contusion is a bruise without a break in the skin or mucosa. Subcutaneous or submucosal hemorrhage in the tissue results in hematoma and swelling of tissue is seen. A contusion may be isolated to the soft tissue but often indicates a deeper injury, such as an underlying bone fracture.

No treatment is necessary for contusions when the injury is limited to soft tissue injury. Blunt trauma and contusion to the soft tissues of the head and neck result in hematoma formation, or a self-limiting bleeding within the subcutaneous tissue which resorbs spontaneously. Make sure that there is no ongoing deep bleeding if a swelling is located in the floor of the mouth or tongue with risk for blocking of airway. Such patients should be observed. Contusions often indicate underlying bone fractures therefore it is important to always carry out radiographic examination to detect bone fractures, which may require separate treatment.

Abrasion

An abrasion is a superficial wound in the skin or mucosa produced by rubbing and scraping of the skin or mucosa leaving a raw, bleeding, and often very painful surface.

Abrasions require thorough lavage and irrigation along with careful inspection and removal of any solid remnants and necrotic epithelium. Remaining irritants may be the source of prolonged inflammation, infection, and discoloration following healing, if they are not removed. Abrasions are generally more painful than lacerations or puncture wounds and more aggressive forms of pain management should be considered. Remove all dirt, gravel, asphalt and other foreign bodies. The only chance for a complete removal is in the emergency situation and these injuries should be treated as soon as possible after the injury. Failure to do so may result in future permanent tattoo and scarring in the skin. Apply local anesthesia as topical and/or terminal injection. Rinse the abrasion with saline. Use a scrub brush, gauze swabs or a soft tooth brush. With severe contamination a mild soapy solution may be used. Remove all foreign bodies with a small excavator or a surgical blade. Irrigation with saline should be performed. Then the wound should be dressed with antibiotic ointment and covered with sterile gauze if necessary to maintain wound moisture until epithelialization is complete. Intra oral abrasions do not have to be treated other than removal of any foreign bodies.

Laceration

Laceration is a wound in the skin or mucosa penetrating into the soft tissue.

A laceration may disrupt blood vessels, nerves, muscles, and involve salivary glands. The most frequently occurring lacerations in the maxillofacial region are seen in lips, oral mucosa, and gingiva. More seldom the tongue is involved.

Administer local anesthesia. Inspect the laceration wound for foreign bodies or tooth fragments, which is the most common reason for lip lacerations. With deep wounds, supplement with radiographic examination. Place a dental film between the lip and the alveolar process with lowest exposure time. Remove all foreign bodies in the emergency phase to prevent infection and disfiguring scarring or tattooing in the skin. Use a syringe with saline underhigh pressure, a scrub brush, and gauze swabs soaked in saline, a surgical scalpel blade or a small spoon excavator.

Avulsion

Avulsion (loss of tissue) injuries are rare but seen with bite injuries or as a result of a very deep and extended abrasion.

Emergency management of soft tissue injuries

Besides medical history and history of medications it is important to determine the mechanisms of injury and time of occurrence to provide information of any potential foreign bodies that may affect healing.

Moreover, it is important to know the tetanus immunization status of the patient.

Wound classification:

- acute – an acute wound is an injury to the skin that occurs suddenly rather than over time. It heals at a predictable and expected rate according to the normal wound healing process.
- chronic – a chronic wound develops when any acute wound fails to heal in the expected time frame for that type of wound, which might be a couple of weeks or up e.g. ulcer, decubitus, burn wound.

Wound types are simple (includes skin, mucous membrane, subcutaneous tissue, superficial fascia, partially the muscle) and compound (any other tissues).

Classification of the accidental wounds:

1. Based on the origine

I. Mechanical:

- Abraded wound (vulnus abrasum)
- Puncured wound (v. punctum)
- Incised wound (v. scissum)
- Cut wound (v. caesum)
- Crush wound (v. contusum)
- Torn wound (v. lacerum)
- Bite wound (v. morsum)
- Shot wound (v. sclopetarium)

II. Chemical:

- Acid
- Base

III. Wounds caused by radiation

IV. Wounds caused by thermal forces:

- Burning
- Freezing

V. Special

2. According to the bacterial contamination:

I. Clean wound (A) – in operation, no inflammation;

II. Clean-contaminated wound – infected clean wound, respiratory, GI, urogenital system is opened under aseptic condition antibiotic profilaxis in high risk patients;

III. Contaminated wound (B) – septic operation the microorganisms involved in the infection was in the operation site before the operation, acute accidental wounds; perforation, fistula, abscess Betadin or physiological salt solution lavage, antibiotic profilaxis;

IV. Heavily contaminated wound (C) – sever septic operation long time between the contamination and the wound care war wounds, gangrene, abcess, ileus, tissue necrosis, organ necrosis.

The mnemonic ABCDE is used to remember the order of assessment with the purpose to treat first that kills first.

A: Airway and C-spine stabilization

B: Breathing

C: Circulation

D: Disability

E: Environment and Exposure.

TOPIC: SURGICAL TREATMENT OF SOFT TISSUES INJURES. PREVENTION OF TETANUS, CLOSTRIDIAL INFECTION AND RABIES

Treatment of injures of soft tissues

Factors that affect wound healing and the potential for infection

- Patient:
  - Age
  - Underlying illnesses or disease: consider anemia, diabetes or immunocompromised
  - Effect of the injury on healing (e.g. devascularization)
- Wound:
  - Organ or tissue injured
  - Extent of injury
  - Nature of injury (for example, a laceration will be a less complicated wound than a crush injury)
  - Contamination or infection
  - Time between injury and treatment (sooner is better)
- Local factors:
  - Haemostasis and debridement
  - Timing of closure Wound: Primary repair
- Primary closure requires that clean tissue is approximated without tension.
- Injudicious closure of a contaminated wound will promote infection and delay healing.

Essential suturing techniques include:

- Interrupted simple
- Continuous simple
- Vertical mattress
- Horizontal mattress
- Intradermal.
- Staples are an expensive, but rapid, alternative to sutures for skin closure.

The aim with all techniques is to approximate the wound edges without gaps or tension. The size of the suture "bite" and the interval between bites should be equal in length and proportional to the thickness of tissue being approximated. As suture is a foreign body, use the minimal size and amount of suture material required to close the wound. Leave skin sutures in place for 5 days; leave the sutures in longer if healing is expected to be slow due to the blood supply of a particular location or the patient's condition. If appearance is important and suture marks unacceptable, as in the face, remove sutures as early as 3 days. In this case, re-enforce the wound with skin tapes. Close deep wounds in layers, using absorbable sutures for the deep layers. Place a latex drain in deep oozing wounds to prevent haematoma formation. Wound: Delayed primary closure. Irrigate clean contaminated wounds; then pack them open with damp saline gauze. Close the wounds with sutures at 2 days. These sutures can be placed at the time of wound irrigation or at the time of wound closure.

Wound: Secondary healing

To promote healing by secondary intention, perform wound toilet and surgical debridement.

1. Surgical wound toilet involves:

– Cleaning the skin with antiseptics

– Irrigation of wounds with saline

– Surgical debridement of all dead tissue and foreign matter. Dead tissue does not bleed when cut.

2. Wound debridement involves:

– Gentle handling of tissues minimizes bleeding.

– Control residual bleeding with compression, ligation or cautery.

– Dead or devitalized muscle is dark in color, soft, easily damaged and does not contract when pinched.

– During debridement, excise only a very thin margin of skin from the wound edge.

Stages of wound treatment:

1. Systematically perform wound toilet and surgical debridement, initially to the superficial layers of tissues and subsequently to the deeper layers.
2. After scrubbing the skin with soap and irrigating the wound with saline, prep the skin with antiseptic.
3. Do not use antiseptics within the wound.
4. Debride the wound meticulously to remove any loose foreign material such as dirt, grass, wood, glass or clothing.
5. With a scalpel or dissecting scissors, remove all adherent foreign material along with a thin margin of underlying tissue and then irrigate the wound again.
6. Continue the cycle of surgical debridement and saline irrigation until the wound is completely clean.
7. Leave the wound open after debridement to allow healing by secondary intention.

8. Pack it lightly with damp saline gauze and cover the packed wound with a dry dressing.
9. Change the packing and dressing daily or more often if the outer dressing becomes damp with blood or other body fluids.
10. Large defects will require closure with flaps or skin grafts but may be initially managed with saline packing).

Although many different ways of closing wounds have been presented, such as suturing, gluing, taping and using staples, in the oral and maxillofacial region suturing is still the most frequently used method of wound closing. Suturing is carried out with simple interrupted sutures in the oral mucosa and gingiva. Use absorbable synthetic material suture material in sizes 4-0 to 5-0 for intra oral sutures. For skin sutures a thinner suture, 5-0, 6-0, is preferable in esthetically sensitive areas. Absorbable or non-absorbable sutures may be used in the skin. With deep lacerations suture in layers. It is important that the layers of the laceration be reapproximated to their appropriate counterpart.

Intracutaneous (intradermal, subcuticular) suturing technique may be preferred for cutaneous closure in esthetically sensitive areas. Scars contract with time, so a slight wound-edge eversion is an important principle. A technique that slightly raises the wound edges above the skin plane will ultimately result in a cosmetically acceptable closure.

Adhesive tape/strips may be used to relieve tension

Penetrating lacerations engaging both the skin and the oral cavity are complex lacerations necessitating layered closure. After thorough debridement, cleaning and irrigation, repair begins with alignment of the vermillion (the red part of the lip) border and white roll (the prominent ridge just above where the lip meets the facial skin), if involved. Special attention must be paid to carefully approximate the transition of skin to mucosa (vermillion border) as any inaccuracy in wound closure will be very apparent and compromise esthetics. Next, the muscle is closed with absorbable sutures followed by mucosal closure and, finally, skin closure. Ideally, lacerations in the maxillofacial region are repaired in order for healing to occur by primary intention. This type of healing takes place quickly and with minimal scar formation. The consensus is that uncomplicated clean lacerations of the face can be closed primarily up to 24 hours after injury. Delayed primary closure is a method of management of dirty or infected traumatic wounds or wounds that have gone unrepaired for a considerable amount of time. Severely dirty wounds are converted to fresh wounds through debridement and removal of tissue edges and can later be closed if no infection is evident. When a wound is left open, a more prolonged healing process, healing by secondary intention, occurs. This will result in scar contraction and can be sometimes be accepted in the oral mucosa but should be avoided in the skin because of compromised esthetical result.

Avulsions

Administer local anesthesia. Clean the wound. Small defects can be left for spontaneous healing especially in young individuals who have a higher regeneration capacity than older individuals. With large tissue loss, excision and primary closure with flaps or skin grafts may be necessary for coverage. With animal bites, usually dogs, antibiotics should always be administered regardless of duration. Rabies vaccine should be considered, depending on the status of the dog.

Antibiotics to prevent wound infection

Bacterial infection is the most common complication and is highly related to time between trauma and wound closure. There is no convincing evidence that antibiotics are useful for preventing infections in simple lacerations. However, there are situations when antibiotics should be given:

- heavily contaminated wound;
- compromised wound cleansing;
- delayed treatment >24 hours;
- injuries penetrating through the whole lip or cheek;
- human or animal bite wounds;
- simultaneous extensive surgery such as open reduction of fracture;
- when general defense system of the patient is compromised e.g. diabetes and immunocompromised patients.

Tetanus

In case of contaminated wounds, especially wounds occurring outdoors, tetanus prophylaxis should be considered. Tetanus has declined considerably since the use of widespread vaccination. However, tetanus has a high morbidity so it is important to always check if the patient is immunized. If there is more than 10 years since last immunization, a booster dose should be given.

Clostridia are strictly anaerobic to aerotolerant sporeforming bacilli found in soil as well as in normal intestinal flora of man and animals. There are both gram-positive and gram-negative species, although the majority of isolates are gram-positive. Exotoxin(s) play an important role in disease pathogenesis.

The clostridia that cause gas gangrene are anaerobic, spore-forming bacilli, but some species may not readily sporulate, e.g., *C perfringens*.

Wounds are contaminated by clostridia from the environment or the host's normal flora. The anaerobic tissue environment facilitates replication of clostridia and secretion of toxins.

Patients may present with a wound infection. Severity varies from invasion of live tissue with systemic toxemia to relatively benign superficial contamination of already necrotic tissue.

Wound management - anamnesis

- When and where did the injury happen?
- Alcohol and drug consumption
- What did cause the wound?
- The circumstances of the injury
- Other diseases eg. diabetes mellitus, tumour, atherosclesosis, allergy
- The state of patient's vaccination against Tetanus
- Prevention of rabies
- The applied first-aid

Diagnosis

These infections are diagnosed by recognition of a characteristic lesion coupled with tissue Gram stains and bacterial culture.

Control

Wound infections are controlled by administration of antimicrobial agents (e.g., penicillin, chloramphenicol) coupled with tissue debridement (for more severe forms of clostridial wound infections).

Tetanus and cloctrudium tetani

Clinical Manifestations

Tetanus is a severe disease caused by the toxin of *C. tetani*. This organism grows in a wound and secretes a toxin that invades systemically and causes muscle spasms. The initial symptom is cramping and twitching of muscles around a wound. The patient usually has no fever but sweats profusely and begins to experience pain, especially in the area of the wound and around the neck and jaw muscles (trismus). Portions of the body may become extremely rigid, and opisthotonos (a spasm in which the head and heels are bent backward and the body bowed forward) is common. Complications include fractures, bowel impaction, intramuscular hematoma, muscle ruptures, and pulmonary, renal, and cardiac problems.

Structure and Classification

C. tetani is an anaerobic gram-positive rod that forms terminal spores, giving it a characteristic tennis racquet appearance. Some strains do not sporulate readily, and spores may not appear until the third or fourth day of culture. Most strains are motile with peritrichous flagella; colonies often swarm on agar plates, but some strains are nonflagellated and nonmotile. The presence of *C. tetani* should be suspected on isolation of a swarming rod that produces indole and has terminal spherical spores, but does not produce acid from glucose. Toxigenic *C. tetani* contains a plasmid that produces a toxin called tetanospasmin, but nontoxigenic strains also exist. Tetanospasmin is responsible for the infamous toxemia called tetanus. The two animal species most susceptible to this toxemia are horses and humans.

Diagnosis

Diagnosis of tetanus is obvious in advanced cases; however, successful treatment depends on early diagnosis before a lethal amount of toxin becomes fixed to neural tissue. The patient should be treated on a clinical basis without waiting for laboratory data. *C. tetani* can be recovered from the wound in only about one-third of the cases, and a wound is not even evident in 10–20% of cases. It is important for the clinician to be aware that toxigenic strains of *C. tetani* can grow actively in the wound of an immunized person, but the presence of antitoxin antibodies prevents initiation of tetanus. Also, because tetanus is commonly found in the soil, the mere presence of tetanus in a wound does not imply that the organism is actively replicating and secreting toxin.

Numerous syndromes, including rabies and meningitis, have symptoms similar to those of tetanus and must be considered in the differential diagnosis. Ingestion of strychnine (found in rat poison) can cause symptoms that closely resemble those of generalized tetanus. Trismus can occur in encephalitis, phenothiazine reactions, and diseases involving the jaw.

Control

Injections of tetanus toxoid are prophylactic. Currently, booster doses are recommended only every 10 years by the CDC. More frequent boosters are unnecessary and may cause local reactions resembling the Arthus phenomenon or a delayed hypersensitivity reaction. It has been noted that, because of their immunodeficiency state, AIDS patients may not respond to prophylactic injections of tetanus toxoid. An antibody titer above 0.01 international units (IU) per ml is usually considered protective.

Human tetanus immunoglobulin (HTIG) in a dose of 250 IU intramuscularly should be considered for those with questionable immune status.

Treatment of diagnosed tetanus has a number of aspects. The offending organism must be removed by local debridement, after the patient's spasms are controlled by benzodiazepines. Penicillin or metronidazole is usually administered to kill the bacteria, but may not be a necessary adjunct in therapy. Although penicillin has been historically considered to be the drug of choice, it has been speculated that penicillin could have an adverse effect by acting synergistically with tetanospasmin. Metronidazole is currently recommended, and there is some evidence that it is associated with an improved prognosis. HTIG is injected intramuscularly: dosage recommendations vary from 500 IU in a single intramuscular injection to 3000–6000 IU injected intramuscularly in several sites. Supportive measures, such as respiratory assistance and intravenous fluids, are often critical to patient survival. Recommended treatment includes benzodiazepines, such as diazepam (Valium). Analgesics that will not cause respiratory depression should be used, and include codeine, meperidine (Demerol), and morphine. Adequate nutritional support should be provided and should consider that the patient's nutritional needs are extraordinarily great.

In cases of clean, minor wounds, tetanus toxoid should be administered only if the patient has not had a booster dose within the past 10 years. For more serious wounds, toxoid should be administered if the patient has not had a booster dose within the past 5 years. All patients who have a reasonable potential for contracting tetanus should receive injections of tetanus immunoglobulin, including those recovering from diagnosed cases of tetanus.

Rabies infection

Signs and symptoms

Rabies is a viral illness spread via the saliva of an infected animal. This occurs usually through biting a human or another animal. Transmission can also occur through saliva touching an open wound or touching mucous membranes.

The average incubation period (time from infection to time of development of symptoms) in humans is 30-60 days, but it may range from less than 10 days to several years.

Most people first develop symptoms of pain, tingling, or itching shooting from the bite site (or site of virus entry).

Nonspecific complaints of fevers, chills, fatigue, muscle aches, and irritability may accompany these complaints. These symptoms may appear similar to those of the flu. Early on, these complaints may seem like any virus, except for the shooting sensations from the bite site.

Gradually, however, the affected individual becomes extremely ill, developing a variety of symptoms, including high fever, confusion, agitation, and eventually seizures and coma.

Typically, people with rabies develop irregular contractions and spasms of the breathing muscles when exposed to water (this is termed hydrophobia). They may demonstrate the same response to a puff of air directed at them (termed aerophobia). By this point, they are obviously extremely ill.

Eventually, the various organs of the body are affected, and the person dies despite support with medication and a respirator.

The disease can then take two forms:

1. With paralytic rabies (approximately 20% of cases), the patient's muscles slowly become paralyzed (usually starting at the site of the bite). This is the less common form and ends in comaand death.
2. With furious rabies (about 80% of cases), the patient exhibits the classic symptoms of rabies, such as
   - anxiety and confusion (The patient is often overly active.);
   - encephalitis, causing hallucinations, confusion, and coma;
   - hypersalivation;
   - hydrophobia (fear and avoidance of water);
   - difficulty swallowing.

Once the clinical signs of rabies occur, the disease is nearly always fatal.

Treatment for people bitten by animals with rabies

If you've been bitten by an animal that is known to have rabies, you'll receive a series of shots to prevent the rabies virus from infecting you. If the animal that bit you can't be found, it may be safest to assume that the animal has rabies. But this will depend on several factors, such as the type of animal and the situation in which the bite occurred.

Rabies shots include:

A fast-acting shot (rabies immune globulin) to prevent the virus from infecting you. Part of this injection is given near the area where the animal bit you if possible, as soon as possible after the bite.

A series of rabies vaccines to help your body learn to identify and fight the rabies virus. Rabies vaccines are given as injections in your arm. You receive four injections over 14 days.

Treatment to prevent rabies has three essential components if a high probability of viral transmission exists. Depending on the likelihood the animal has rabies and, in some cases, the availability of the animal for observation, your doctor may not initiate the latter two steps involving shots against the rabies virus.

- Wound care involving soap and a virus-killing cleanser (this should always be done for any animal bite)
- A onetime injection of human rabies immune globulin (or HRIG), which is a substance that provides rapid, short-term protection against rabies
- Injection of the first of a series of vaccine doses to provide protection against rabies after an exposure

The decision to treat for rabies: The likelihood of an animal having rabies depends heavily on the species of the animal, its behavior, and where you were exposed to the animal. For example, in some areas of the country, such as the Texas-Mexico border, stray dogs have an extremely high likelihood of being rabid. In other areas, stray dogs may have little chance of being rabid.

Domestic dogs, cats, and ferrets have a well-defined incubation period for the rabies virus. If you have been bitten by one of these three animals, and the animal does not appear overtly ill at the time, then the animal will be observed by local health authorities for 10 days. If the animal remains well during that period, you will not need rabies shots.

If the animal has the potential for rabies and is available for sacrifice and immediate examination by the local health department, then treatment may be withheld pending the results of that test. This would include animals such as any wild animal, or an unwanted stray dog or cat, if you know where the animal is (dead or alive).

If the animal has the potential for rabies and is unavailable for sacrifice and examination, then you will be given rabies shots in the emergency department.

Special situations

Rabies vaccination and pregnancy: Both human rabies immune globulin (HRIG) and the various rabies vaccines are safe in pregnancy.

Immune suppression: If you are taking medicines (such asprednisone or steroids) or have a disease that interferes with the body's immune response to the rabies vaccine, discuss these situations with your doctor. The doctor will then determine if you will need additional blood tests to ensure that an adequate response to the vaccine has occurred and that protection against rabies is developing.

There are two types of rabies vaccine injections

Injection of the human rabies immune globulin (HRIG) for immediate protection is based on your exact weight. This is not a situation where more is better. Therefore, you should not overestimate your weight. If the exact weight is not known, you will be weighed at the hospital.

Once the dose is determined, as much as possible is injected into and around the bite site. If the entire volume does not fit into the tissue in that area (for example, the tip of the finger), then the remaining volume will be injected into some other site in your body, such as the arm, leg, or buttocks. The doctor may use numbing medicine to decrease the pain associated with injection of HRIG into the tissues at the bite site.

If you have been previously immunized adequately against rabies, then the HRIG is not needed. You would need only the vaccine described in the next section.

Injection of the vaccine will begin during this initial visit to the emergency department and will proceed on a schedule over the next 14 days, with a total of four small injections.

There are two different types of rabies vaccines licensed for use in the United States (human diploid cell (HDCV) and purified chick embryo cell culture vaccine (PCECV). If given properly and on schedule, both of types will protect you against rabies.

The dose for each is 1 cc, or milliliter, delivered into the muscle. This vaccine must be delivered into the deltoid, or shoulder muscle, in adults or older children. The front, outside aspect of the thigh muscle is acceptable in younger children. It must never be injected into the buttocks. Injection into the proper site ensures absorption. It must be administered in a site different from the remainder of the immune globulin that is not injected into the bite site.

If you have never been vaccinated against rabies, then vaccine shots will be given on the day of the visit (day zero), and again on days three, seven, and 14. If you have already been adequately immunized against rabies, a series of two booster vaccine injections will be given on day zero and again on day three only. This is sufficient to stimulate your body's immune system, or memory, and provide protection against rabies.

TOPIC: DISLOCATION AND FRACTURES OF THE TEETH, FRACTURES OF THE ALVEOLAR PROCESS

Maxillofacial Examination

For medicolegal purposes, consider preoperative photographs prior to invasive treatment.

Include the following in the patient examination:

- Extraoral soft tissue
- Intraoral soft tissue
- Jaws and alveolar bone
- Teeth (displacement and mobility)
- Percussion and pulp testing

Ensure that the patient is cleaned extraorally with a mild antiseptic soap, while taking care not to further inoculate injury sites with debris or foreign bodies. Consider tetanus prophylaxis, depending on previous immunization compliance and wound presentation.

Thoroughly inspect superficial and deep lacerations, abrasions, or any soft tissue compromise. The mechanism of injury elicited in the history and the soft tissue defect alerts the surgeon to suspect underlying hard-tissue damage, such as to the maxilla, the mandible, the temporomandibular joint (TMJ), and alveolar fractures. Success rates are time-dependent with dentoalveolar trauma, and generally perioral soft tissue lacerations (lips) should be repaired after intraoral treatment, except in cases of poor hemorrhage control. In children, women, and the elderly, if the injury observed fails to correlate well with the history given, suspect and subsequently rule out abuse. Authorities, such as social services representatives, initiate proper legal protocols, if necessary.

Prior to any intraoral manipulations, obtain initial radiographic studies (eg, in the pediatric patient, knowledge of the errant deciduous tooth root to the permanent tooth bud position). The chance of further damage could be exponentially disastrous to both the future eruption and the morphology of the developing permanent tooth. Approach intraoral soft tissue examination with caution. Carefully manipulate and handle traumatized tissues to avoid further compromise. Depending on the mechanism of injury, bone or tooth fragments may have penetrated these delicate areas. Closely inspect hematoma formation or ecchymotic areas. Buccal mucosal lacerations should raise the suspicion for Stensen’s duct or orifice injuries. The lips, the floor of the mouth, and the tongue regions are all areas at risk for penetrating or secondary injury and thus should be inspected accordingly. Account for all fractured or missing teeth and restorations or assume they were swallowed, aspirated, or lodged within adjacent structures. Similarly, arrange for radiographic evaluation of the maxillary and nasal sinuses prior to further treatment. While examining for jaw and alveolar bone fractures, the presence of gross mobility or pericoronal bleeding of the involved teeth may be noted. Sublingual ecchymosis at the floor of the mouth is pathognomonic for an underlying mandible fracture. Step defects, crepitation, malocclusion, and gingival lacerations all raise suspicion of possible underlying bony defects. Assess all fractured teeth for enamel, dentin, and pulpal involvement. Complete mobility of the crown may indicate crown-root fracture. Superficial crazing or infractions may be identified with a direct light source, transilluminating perpendicular to the long axis of the tooth from the incisal edge. Inspect and consider each tooth at risk, even at sites distal to the initial traumatic impact. Indirect trauma of the chin may cause posterior dentition defects, such as vertical or cusp fractures. Check occlusion and note any displacements, intrusions, or luxations. The direction of force is most commonly in a buccal-lingual direction.

Test percussion sensitivity and pulp vitality to rule out periodontal ligament injury or one of the many forms of fractures. Gentle tapping of the injured and noninjured control teeth is the technique of choice. Use the handle of a mouth mirror or a specially designed calibrated percussion instrument. Tactile, auditory, and visual senses are used. Dullness may alert the surgeon to the possibility of a luxation injury or alveolar fracture. The quality of this sound indicates that the teeth are not in optimal contact with the adjacent bony structure. If the enamel is fractured or infraction has occurred, the sound is reminiscent of a “cracked tea cup.” The typical sound of the uninjured tooth is that of solid metallic resonance. Percussion testing, in and of it, can add insult to injury; thus, control and caution are warranted. Evaluate tooth vitality via various pulp testing modalities. Mechanical, thermal, and electrical noxious stimuli are used. These tests use various stimuli to check for conduction disturbances at the sensory receptors of the pulp. The pulp comprises both nonmyelinated and myelinated nerve fibers, which regulate vascular changes and respond to pain stimuli, respectively. As the tooth develops, the pain fibers (ie, myelinated) increase, while simultaneously lowering the electrometric pulp stimulation. This concept sheds light on some of the treatment differences in open and closed apices of the permanent dentition. Pulp testing in the acute phase of dentoalveolar fracture is controversial and heavily based on the cooperation and communication of the patient as well as the repair process of the injured pulp tissue. The fear of possibly experiencing increased pain during testing, especially in children, limits verbal objectivity and may render pulp testing too unreliable. Also, acutely injured teeth may revascularize in approximately 1 month, thus increasing the risk of false-negative results during pulp testing. The development stage of the involvedteeth also plays a significant role in the repair process. Incomplete apical development increases the chances of pulp repair and revascularization. As the tooth matures and apical width constriction starts, the chances of pulp repair decrease. Bacterial invasion in the pulp injury zone increases the risk of total pulp necrosis. Paradoxically, occasionally uninjured teeth may not respond as expected. Even with this controversy in mind, pulp testing continues.

Some of the testing paraphernalia are listed as follows:

- Mechanical stimulation
- Dental probe
- Cavity prepping with drills
- Saline-laden cotton pledget (fractured teeth)
- Thermal test
- Heated gutta-percha
- Ice
- Ethyl chloride
- Carbon dioxide snow
- Dichlorodifluoromethane
- Electrometric test
- Electric pulp testers

Laser Doppler flowmetry (LDF), a relatively new pulp testing apparatus, has shown promise. A laser beam, which is directed at the coronal-labial aspect of the pulp, is scattered by pulp blood cells that in turn produce a Doppler frequency shift. The fraction of light scattered back is detected and processed to elicit a signal. The basic theory is that the pulp revascularization process can be monitored. Studies have shown that, in cases wherein electrometric tests were negative and LDF displayed vascular perfusion, the LDF accuracy of pulp vitality reached 100%. The drawbacks to this form of testing are poor light transmission when blood pigments from discolored teeth are encountered, complexity of equipment use, and poor price containment. To ensure completeness, generate a standardized treatment record during the evaluation process, which systematically culminates in a diagnosis, treatment plan, and prognosis.

Radiographic Examination

Radiographic examination is essential to determine whether any underlying structures are damaged and should include periapical, occlusal, and panoramic radiographs. The periapical radiograph provides the most detailed information about root fractures and the dislocation of teeth. Following treatment, periapical films can confirm the proper positioning of an avulsed or luxated tooth into the alveolus. Occlusal radiographs, however, provide a larger field of view, and the detail is almost as sharp as a periapical radiograph.

When occlusal radiographs or periapical films are used to examine soft tissues for the presence of foreign bodies, reduce the radiographic exposure time. The panoramic radiograph is a useful screening view and can demonstrate fractures of the mandible and maxilla as well as fractures of the alveolar ridges and teeth. In the hospital setting, dental radiographs may not be available. Although not ideal, plain films, such as the mandibular series and the Caldwell views, may reveal tooth and alveolar injuries. In the trauma patient whose tooth has not been accounted for at the accident scene, arrange for chest films to rule out the possibility of aspiration. Abdominal radiographic films can determine whether displaced teeth or prosthetic appliances have been ingested.

Classification of Dentoalveolar Injuries

Once the diagnosis of dentoalveolar injury is made, the injury is classified for ease of communication and treatment planning. Many classification systems have been proposed over the years based on the anatomic site of injury, the cause, the treatment alternatives, or a combination of these.

The two most common systems are those developed by Ellis and Davey and Andreasen. The most commonly used simple and comprehensive classification of dentoalveolar injuries is one that was developed by Andreasen and originally adopted by the World Health Organization system for disease classification, using the International Classification of Diseases codes. The classification can be applied to both permanent and primary dentition. It includes descriptions of injuries to teeth, supporting structures, and gingival and oral mucosa.

Injuries to the teeth and supporting structures are divided into dental tissues, pulp, periodontal tissues, and supporting bone as follows:

- Dental tissues and pulp
- Crown infraction (ie, a craze line or crack in the tooth without loss of tooth substance)
- Crown fracture that is confined to enamel, or enamel and dentin, with no root exposure (uncomplicated)
- Crown fracture producing a pulp exposure (complicated)
- Fracture involving the enamel, dentin, and cementum without pulp exposure (uncomplicated crown root fracture)
- Fracture involving the enamel, dentin, and cementum with pulp exposure (complicated crown-root fracture)
- Root fracture involving the dentin and cementum and producing a pulp exposure (root fracture)
- Injuries to periodontal tissues are divided into six categories and encompass what are commonly referred to as subluxations and avulsions.
- Concussion: defined as an injury to the periodontium producing sensitivity to percussion without loosening or displacement of the tooth
- Subluxation: the tooth is loosened but not displaced
- Luxation (ie, lateral, intrusion, and extrusion) dislocation, or partial avulsion: the tooth is displaced without an accompanying comminution or fracture of the alveolar socket
- Injuries to the supporting bone
- Comminution of the alveolar housing, often occurring with an intrusive or lateral luxation
- Fracture of a single wall of an alveolus
- Fracture of the alveolar process, en bloc, in a patient having teeth but without the fracture line necessarily extending through a tooth socket
- Fracture involving the main body of the mandible or maxilla

Categories of injuries to the gingival or oral mucosa area include the following:

- Abrasion
- Contusion
- Laceration

Treatment of Injuries to the Hard Tissues and Pulp Enamel Fractures (Crown Infraction)

These injuries include fractures, chips, and cracks that are confined to enamel, not crossing the enamel-dentin border but terminating at the border. The cracks or fractures can be seen by indirect light or transillumination.

Treatment involves smoothing the rough edges or repairing with composite resin. It is difficult to predict future pulpal vitality; for this reason, perform pulp testing immediately after the injury and again in 6 to 8 weeks.

Crown Fracture without Pulp Involvement

Crown fractures are the most frequent injuries in the permanent dentition. Crown fractures that expose dentinal tubules potentially may lead to contamination and inflammation of the pulp, eventually resulting in pulpal necrosis if untreated. Luxation injury concomitant to crown fractures, with or without pulp exposure, is the primary source of pulpal complications following injury. Prognosis is better if the enamel-dentin fracture involves a tooth that has not been luxated because the blood supply to the pulp has not been disturbed, and the immunologic defense systems in the pulp will combat bacterial invasion. Treatment is directed at protecting the pulp by sealing the dentinal tubules. Although zinc oxide–eugenol cement has been one of the best agents for producing a hermetic antibacterial seal, it is generally not recommended at the site where a composite resin restoration is placed because the eugenol component may interfere with polymerization, at least with some composites. A similar effect has been seen with a hard-setting calcium hydroxide paste, resulting in bond strength reduction incertain dental-bonding agents. In fractures with dentin exposure only, we recommend a dental bonding agent, followed by a composite restoration. With pulp exposure, the preferred treatment is calcium hydroxide placed directly over the exposure and sealed in place with a glass ionomer cement followed by a dentin bonding agent and composite.

Crown Fracture with Pulp Involvement

Crown fractures involving the enamel, dentin, and pulp are called complicated crown fractures by Andreasen and Class III fractures by Ellis. Prognosis depends on the length of time that has elapsed since the injury occurred, the size of the pulp exposure, the condition of the pulp (vital or nonvital), and the stage of root development. Make every effort to preserve the pulp in immature teeth. Conversely, in mature teeth with extensive loss of tooth structure, pulp extirpation and root canal therapy are prudent before post, core and crown restoration. The prognosis is best for teeth with a vital pulp exposure if the fracture is treated within the first 2 hours. Treatment requires direct pulp capping for small pinpoint exposures. If a patient's tooth has an open apex and a small pulp exposure is seen within 24 hours, it should be directly pulpcapped with calcium hydroxide. Perform calcium hydroxide pulpotomies for larger exposures and for small exposures in teeth with open apices over 24 hours old. The direct pulp cap of calcium hydroxide pulpotomy is designed to allow a tooth with an open apex to complete root development. Teeth that have calcium hydroxide pulpotomies usually require root canal therapy along with a post and core and ultimately coronal coverage. In fractures with a vital pulp and a closed apex, perform a direct pulp cap if there is a small pulp exposure and if the patient is seen within 24 hours. If the pulp exposure is larger then 1.5 mm or if it has been present for over 24 hours, carry out root canal therapy.

Crown-Root Fracture

A fracture that is longitudinal and follows the long axis of the tooth or if the coronal fragment constitutes more than one-third of the clinical root, extraction is generally recommended. However, with a fracture line that is above or slightly below the cervical margin, appropriate forms of conservative therapy can usually be used to restore the tooth. Crown lengthening or orthodontic elevation of the involved tooth may be necessary.

Root Fracture

This type of fracture is limited to fractures involving the roots only (Ellis IV). Most root fractures occur in the apical and middle one-third and rarely in the cervical one-third. Root fractures are not always horizontal; in fact, they are often diagonal in angulation. Radiographs taken immediately after an injury may not show a horizontal or diagonal root fracture. After 1 or 2 weeks when inflammation, hemorrhage, and resorption have caused the fragments to separate, the radiograph will show the damage more conclusively.

Root fractures in the apical or middle one-third are usually not splinted unless there is excessive mobility. Treatment of mobile root fractures consists of apposition of the fractured segments with rigid splinting for 12 weeks. Treatment for cervical one-third–root fractures usually involves extraction of the tooth or orthodontic extrusion of the root.

Periodontal Tissue Injury and Treatment Injury to the periodontal tissue presents itself in many ways. Radiographically, this injury usually involves an evident dislocation or a movement of the tooth, and narrowing or loss of periodontal space may be seen. The fate of the tooth that has sustained a periodontal injury is twofold. Primarily, we see the injury from the localized impact and the late complication of the secondary resorptive process. The likely result of displacement injuries is the development of some type and degree of resorption. Thus, to better treat these types of injuries, it would behoove the surgeon to understand this process, both clinically and conceptually. This process affects both primary and permanent dentition. The etiology and pathogenesis is essentially identical to that seen in avulsion injuries, which we discuss later in this chapter in “Exarticulations (Avulsions).”

Classification of Root Resorption

Root resorption is classified as either root surface resorption or root canal resorption. Root surface resorption, also known as external root resorption, is most commonly seen after intrusive injuries and less in subluxation injuries.

It is classified into three types:

- surface resorption,
- replacement resorption,
- inflammatory resorption.

Root Surface Resorption

Surface resorption indicates that the luxated or avulsed tooth root displays superficial resorption lacunae, which are repaired with newly formed cementum. Although not usually seen on radiographs, these may appear as vague excavations or cavities on the lateral root surface. A normal lamina dura is usually present. This development is a response to localized periodontal ligament and/or cementum injury. The process is less aggressive and self-limiting compared with the other resorption processes.

Replacement resorption

Replacement resorption also known as ankylosis, presents as an indistinguishable merging of bone and root substance. The rootsubstance is being ultimately replaced by bone, and radiographically a loss of the periodontal space and progressive root resorption is seen.

Inflammatory resorption

Inflammatory resorption appears as wellcircumscribed areas of cementum and dentin resorption. The localized adjacent periodontal tissue is markedly inflamed. The onset of inflammation is a result of the infected and necrotic pulp tissue within the root canal. The radiograph shows an appearance of root resorption with lines of adjacent bone radiolucency.

Classification of Root Canal Resorption

Root canal resorption, also known as internal root resorption, presents less often than root surface resorption. Studies found that it appears in both permanent and primary teeth. Radiographic imaging may be equivocal; labial or lingual presentations of surface resorption may be erroneously superimposed over the root canal. To avoid a misdiagnosis supplemental radiographic views are warranted.

Root canal resorption is classified as two types:

- internal replacement resorption
- internal inflammatory resorption.

Internal replacement resorption

Internal replacement resorption shows metaplastic replacement of normal pulp tissue into cancellous bone, resulting in a widened pulp chamber. This is a characteristic process that is seen in root fractures and, to a lesser extent, in luxation injuries.

Internal inflammatory resorption

Internal inflammatory resorption often located at the cervical region of the pulp, presents radiographically as an irregular or oval-shaped radiolucent enlargement within the pulp chamber. This condition relates to the ingression of bacteria via dentinal tubules within a necrotic pulp delineated as the necrotic pulp zone. Possibly, this zone is responsible for the progression of the process. Normal pulp tissue is altered and transformed into granulation tissue with giant cells that resorb the dentinal walls of the root canal, giving the chamber an enlarged appearance. The cessation of this process will require root canal therapy. The potential devastating effects of the resorptive process require immediate and proper treatment of periodontal injuries.

Classification of Periodontal Injuries

Periodontal injuries are classified as concussions and displacements. Displacements include subluxations, intrusive luxations, extrusive luxations, and lateral luxations. Concussion Often this injury is overlooked because no acute clinical or radiographic evidence of trauma is seen. No abnormal mobility, displacement, or bleeding is apparent; only minimal injury to the tissues was acquired. Frequently, the history of the insult guides the surgeon to the suspected tooth or teeth. The hallmark to diagnosis is a marked reaction to percussion in both the horizontal and vertical directions. The discomfort is similar to that of a “hot tooth,” hyperemic quality. Because a concussed tooth may take on a chronic course or exhibit progressive problematic sequelae, it warrants close monitoring. Treatment includes taking the suspected tooth out of occlusion to avoid function. If at all plausible, consider occlusal adjustments on the opposing dentition, thereby limiting further trauma to the involved tooth.

Displacements

Displacement injuries, or luxations, principally involve the primary and permanent maxillary central incisors. The mandibular teeth are less at risk, unless a Class III malocclusion exists. Generally, displacement injuries are more prevalent in primary dentition owing to the increased elasticity and resilience of the bony supporting structures. Conversely, permanent teeth will have an increased risk of tooth fracture. The specific luxation classification depends on the force and direction of traumatic impact. Fifteen to 61% of luxation injuries occur in the permanent dentition and 62 to 73% in the primary dentition. Multiple teeth are usually involved in luxation injuries.

Subluxation

Subluxation injuries occur when there is an injury to the toothsupporting structures that causes abnormal loosening; however, there is no clinical or radiographic displacement of the involved tooth. The tooth is sensitive to percussion testing and occlusal forces. Rupture of the periodontal tissues is usually evident by bleeding at the gingival margin crevice. Treatment is similar to that for concussion injuries with occlusal adjustments and vitality testing. Excessive mobility may necessitate nonrigid stabilization. Continue follow-up evaluation and vitality testing for 6 to 8 weeks. Approximately 26% of injuries with this classification result in pupal necrosis, and endodontic treatment is indicated. Studies show that external resorption willoccur in 4% of these injuries. Subluxation has the lowest frequency of periodontal tissue injury resorption. Intrusive Luxation Intrusive luxations may cause marked displacement of the tooth into the alveolar bone, with possible comminution or fracture of the alveolar socket. Percussion sensitivity is limited, and decreased mobility is noted because the tooth is essentially locked in. A highpitched metallic sound is elicited on percussion, reminiscent of an anklyosed tooth. The intrusive injury is more commonly seen in the maxilla because of its less dense anatomy and irregular premaxillary configuration. The superiorly placed hollow cavities and thin floors of the nasal and maxillary sinuses create a formula for relative ease of dislodgement of teeth to these sites when intrusive forces are encountered. Intrusive injuries are the most severe of the luxation injuries that involve the pediatric patient. The intruded primary tooth may be impinging on the tooth bud of the permanent successors in a buccal-occlusal position. The incidence of pupal necrosis is relatively high (96%). Inflammatory resorption incidence may reach 52% as a result of the necrotic pulp. Treating intrusive injuries depends on root development. If incomplete root development exists, allow the intruded tooth to re-erupt. Continue this process for approximately 3 months. If reeruption does not occur, to facilitate this process, place an orthodontic extruding appliance. If pulp necrosis occurs, seek endodontic therapy. In cases of complete root development with closed apices, re-position the tooth atraumatically, and stabilize with a nonrigid splint. Then, initiate endodontic therapy in approximately 10 to 14 days after injury. Use CaOH as canal filler in this therapy to retard or inhibit the inflammatory or replacement resorption process. In fact, use CaOH in any intrusive luxation injuries that result in the displacement of the tooth in excess of 3 to 5 mm, and initiate within 2 weeks. This, along with instrumentation of the canal, will eradicate the bacterial contamination and allow for the repair of the periodontal ligament. Replace the CaOH filler if it resorbs during the healing process. Arrange for frequent radiographic follow-up at 3-month intervals, and continue for 6 to 12 months. Perform conventional root canal therapy with gutta-percha obturation when signs of resorption have ceased.

Extrusive Luxation

Extrusive luxations are the partial displacement of the tooth out of the socket in a coronal or incisal direction with lingual deviation of the crown. This results in the rupture and severance of the neurovascular and periodontal ligament (PDL) tissues, respectively. There is gross mobility and bleeding at the gingival margin. Further, radiographically, the PDL space is widened. A dull sound is heard on percussion testing. Pulp necrosis occurs approximately 64% of the time, and a relatively low frequency of external resorption is seen at 7%. It is treated by delicately placing the extruded tooth back into the proper position in the socket. Check and re-check occlusion to ensure no rotation has occurred. Then, stabilize the tooth with a nonrigid splint for approximately 2 to 3 weeks. If signs of pulp necrosis occur, employ endodontic therapy.

Lateral Luxations

Lateral luxations may result from traumatic forces that displace the tooth, or teeth, in many directions; however, the lingual direction appears to be the most prevalent. These luxations often involve the bony alveolar socket. The radiographic appearance is similar to the extruded tooth on occlusal views, with the PDL space widening in the apical direction. Linear or comminuted fractures are the norm. Lingual and buccal plate expansion may render the tooth mobile. Localized soft tissue compromise is often apparent. When bony defects exist beneath the gingiva, it is common to see complex lacerations and step defects. Because the tooth is often locked in an errant position, the percussion resonance and mobility resemble the intruded tooth. The key to treatment is to reestablish preinjury occlusion. Delay soft tissue repair until this is completed. Manipulate the tooth or teeth back into the socket. If an alveolar segment is involved, reposition it. Digitally apply buccal and lingual pressure in cases of traumatic bony expansion to ensure early PDL repair. Apply a nonrigid splint that is extended to and is supported by the presumably uninjured adjacent teeth. Leave the splint in place for 2 to 8 weeks, depending on bony healing, which may require longer stabilization time. Avoid the use of disimpaction devices, such as forceps or hemostats, while attempting to reestablish proper alignment of teeth or segments. Excessive fulcruming forces may further compromise the tooth and/or supporting structure. In persons who may have experienced delayed treatment in excess of 48 hours, reestablishing occlusion may be difficult and traumatic. Consider spontaneous or orthodontic realignment. Continue frequent radiographic follow-up and vitality testing for several months. Adjacent teeth that may have become devitalized warrant vitality testing. Any signs of pulp necrosis should be met with immediate endodontic therapy. Another complication to consider is the loss of marginal bone support in both lateral and intrusive luxation injuries, which can occur as a temporary or permanent condition. It is seen clinically as an ingrowth of granulation tissue at the gingival crevice, resulting in a loss of attachment. This is the normal process of periodontium healing and takes up to 6 to 8 weeks. When this process occurs, continue maintenance of the splint and pay close attention to oral hygiene compliance to prevent further bone loss. The frequency of this bony loss reaches 5% for lateral luxations and 31% in intruded luxations.

Exarticulations (Avulsions)

Seemingly, avulsion injuries are the worst of the dentoalveolar injuries. By definition, these injuries involve tooth, or teeth, that are completely dislodged from the socket for a period of time. Owing to the higher risk of aspiration, supporting structure damage, or actual physical loss of the tooth, these injuries require special attention. Old ideology and myths still plague the use of newer proven protocols. Avulsion injuries occur from 0.5 to about 16% in the permanent dentition and occur less in the primary dentition (7 to 13%), with children ages 7 to 9 years being most associated with this injury. These injuries usually involve a single tooth, with the maxillary central incisor most often at risk, which is due to the relative instability of the periodontal ligament during the progressive eruption of these teeth. The treatment of such injuries must be geared toward early reestablishment of periodontal ligament cellular physiology. The fate of the avulsed tooth depends on the cellular viability of the periodontal fibers that remain attached to the root surface prior to reimplantation. Although extraoral time is a factor, newer physiologically compatible solutions are available that can maintain and/or replenish periodontal ligament cell metabolites. Two such solutions are Hank's balanced salt solution and ViaSpan. Both Hank's solution and ViaSpan are physiologic with compatible pH and osmolality. ViaSpan is the solution of choice for organ storage during transport for transplantation. The relative availability and cost effectiveness of Hank's solution makes it the medium of choice in storage of avulsed teeth. Commercially available by Phoenix Lazarus Inc., Save-A-Tooth, an emergency tooth preserving system that contains Hank's solution as its active ingredient, is a mainstay in many athletic first aid kits. Other methods for temporarily storing an avulsed tooth are milk, saliva, and saline; however, their ability to replenish cellular metabolites has not been documented. Milk is a readily available medium for the lay person, and, because time is of the essence, it is the medium of choice in the absence of Hank's solution or ViaSpan. Milk will only prevent further cellular demise; thus, it is used specifically when teeth have been extraoral for < 20 minutes. Any periodontal ligament extraoral exposure > 15 minutes will deplete most of the cell metabolites; for this reason, a longer period of extraoral time limits milk's effectiveness to maintain cellular viability. Unlike Hank's solution and ViaSpan, which can store avulsed teeth and replenish cellular metabolites for 24 hours and 1 week, respectively, milk as a storage medium becomes ineffective after approximately 6 hours. Treatment considering the root maturation, the extraoral time, and the generalhealth of the tooth preinjury determines the route of treatment. The idea of early or immediate replantation should be adopted. Teeth that are in poor condition from a hygiene standpoint are generally not replanted. Those that present with moderate to severe periodontal disease, gross caries involving the pulp, apical abscess formations, infection at the replanting site, and bony defects and/or alveolar injuries, in which supporting bone is lost are less likely to be considered for replantation. To optimize success of treatment, replant and stabilize avulsed teeth within 2 hours (120 minutes); periodontal ligament cells become irreversibly necrotic after this time frame. Attempt to salvage avulsed teeth, even if the critical 2-hour period has passed, but the prognosis becomes progressively worse. Teeth with open apices > 1 mm diameter have a prognosis that is much better than that of the more mature or closed-root apex. Treat the tooth with an open root within the 2-hour time frame by placing it in Hank’s solution for about 30 minutes. Next, place the tooth in a 1 mg/20 mL doxycycline bath for 5 minutes, followed by immediate replantation and splint stabilization. If radiographic or clinical evidence of pathology is noted, perform an endodontic apexification procedure with a CaOH filling. The CaOH should be periodically replaced until the apex is closed, followed by conventional root canal therapy. Contrary to CaOH, MTA provides a hard-setting nonresorbable surface with cavity adaptation. It provides excellent tissue biocompatibility and allows for immediate apical seal. The increased potential for reestablishment of pulpal circulation in teeth with open apices has been shown to improve prognosis of survival of the pulp and PDL in the avulsed tooth. This revascularization process is optimized by the topical application of doxycycline. Individuals who have avulsed teeth with mature or closed apices and who present within the 2-hour time frame are treated by placing the tooth in Hank’s solution for about 30 minutes, followed by replantation and splinting for 7 to 10 days. Carry out endodontic cleansing and shaping of the canal, and place a CaOH filling just prior to splint removal. Final gutta-percha obturation is contingent on resolving canal and/or root pathology (6 to 12 months). Late failure of the replantation process is manifested as either inflammatory or replacement resorption owing to a necrotic pulp or compromised PDL, respectively. In individuals who experience an extraoral period that exceeds 2 hours, apical root morphology plays little role in the success rate. Eliminate the necrotic periodontal ligament strands manually or chemically in a sodium hypochlorite wash for approximately 30 minutes. Perform root canal therapy extraorally with conventional cleansing and shaping of the canal. Withhold final obturation until the canal, dentinal tubules, and root surface have been treated with various chemicals in a stepwise fashion. First, a citric acid bath for 3 minutes, followed by rinsing with 0.9% NaCl, will open and debride the dentinal tubules, thus allowing unimpeded ingrowth of connective tissue to the root surface. Second, the tooth should be moved to a 1% stannous fluoride solution for 5 minutes. This will decrease the risk of the resorption process. Finally, set up a 5-minute bath of 1 mg/20 mL doxycycline, which will rid the root surface of residual bacterial remnants and facilitate pulpal revascularization. Complete the final obturation with gutta-percha. The tooth is then replanted into preinjury alignment and splinted for 7 to 10 days.

Treatment Summary for Avulsed Teeth (< 2 h, open apex)

1. Replant immediately if possible
2. Transport in Hank’s solution or milk
3. Present to nearest qualified facility (decrease time call first)
4. Check ABCs; evaluate for associated injuries
5. Store in Hank’s Solution for about 30 min
6. Transfer to a 1 mg/20 mL doxycyline bath for about 5 min
7. Perform radiography (posteroanterior, occlusal, panoramic, chest)
8. Initiate local anesthesia
9. Irrigate socket with saline solution
10. Perform tetanus prophylaxis as needed
11. Initiate antibiotic coverage
12. Replant tooth
13. Splint for 7–10 d
14. Perform apexification with CaOH in cases of pathosis closed apex
15. Store in Hank’s solution for about 30 min
16. Replant
17. Splint for 7–10 d
18. Perform endodontic cleansing and shaping of canal at time of splint removal
19. Fill canal with CaOH (6–12 mo)
20. Perform final gutta-percha obturation (~6–12 mo)

Treatment Summary for Teeth Avulsed (> 2 Hours)

1. Replant immediately, if possible
2. Transport in Hank’s solution or milk
3. Present to nearest qualified facility (decrease time call first)
4. Check ABCs; evaluate for associated injuries (history and physical examination)
5. Bathe tooth in sodium hypochlorite for ~30 min vs manual débridement of the periodontal ligament
6. Perform extraoral RCT
7. Bathe tooth in citric acid (~3 min)
8. Bathe tooth in 1% stannous fluoride (~5 min)
9. Transfer to a 1 mg/20 mL doxycyline bath for ~5 min
10. Perform radiography (posteroanterior, occlusal, panoramic, chest)
11. Initiate local anesthesia
12. Perform tetanus prophylaxis as needed
13. Initiate antibiotic coverage
14. Replant tooth
15. Splint for 7–10 days

Splint Requirements

The splint should:

1. Be able to be applied directly in the mouth without delay owing to laboratory procedures
2. Stabilize the injured tooth in a normal position
3. Provide adequate fixation throughout the entire period of immobilization
4. Neither damage the gingiva nor predispose to caries and should allow for a basic oral hygiene regimen
5. Not interfere with occlusion or articulation
6. Not interfere with any required endodontic therapy
7. Preferably fulfill esthetic demands
8. Allow a certain mobility (nonrigid) to aid periodontal ligament healing in cases of fixation after luxation injuries and replacement of avulsed teeth; however, after root fracture, the splint should be rigid to permit optimal formation of a dentin callus to unite the root fragments
9. Be easily removed without re-injury to tooth

Splinting Protocol and Technique Splinting after avulsion and displacement injuries immobilizes the tooth or segment into proper preinjury alignment and allows for the initial pulpal revasculature and periodontal ligament healing course. Several techniques have been advocated in the past; however, the acid-etch/resin splint (or variants of this technique) is the treatment of choice. This technique fulfills the requirements of acceptable splint utilization in a maxillofacial traumatic injury. The acid-etch technique is the only system that most closely adheres to these recommendations. The arch bar, self-curing, Essig, intracoronal, and circumferential splints may rarely present with an indication but are not routinely recommended. Each has been demonstrated to violate one or many of the basic splint requirements. The arch bar, in particular, produces an eruptive or extrusive force because of the placement of the wire beneath the height of contour of the tooth. Also the rigid nature of these techniques will facilitate the external resorption process.

Treatment of Fractures of the Alveolar Process

Owing to the exposed anatomy, alveolar fractures usually occur at the incisor and premolar regions. Treatment involves early reduction and stabilization of the involved segments. Depending on the fracture'sseverity, use either an open or closed technique. Digital manipulation and pressure, along with rigid splint stabilization, will usually be sufficient in the closed technique. Leave the splint in place for approximately 4 weeks. A gross displacement and/or impedance to reduction may necessitate the open technique. Inability to freely reduce fracture segments may be due to root or bony interferences or impaction (apical lock). Access to the area involves an incision that provides adequate exposure and is located apical to the fracture lines. The segment is then disimpacted or freed up. Proper alignment and occlusion are then attained, and the segments are stabilized with suitable transosseous wire or a small (2.0 mm) monocortical plate. Ensure that the closure of the wound is meticulous to prevent exposure of bone and/or hardware to the ingress of bacteria. Stabilize teeth that may be mobile in the fractured segment with an appropriate secondary splint after bony stabilization. Likewise, avoid removing teeth that are considered nonsalvageable and that are within the bony segment until the bony healing phase is completed (~ 4 weeks). Obvious infection and inadequate bony envelopment indicate early removal. Successful treatment of alveolar fractures is associated with the pupal healing after the injury. When the fracture level is apical to the root tips, the vascular supply to the pulp is less at risk; however, if the line of the fracture and root apices are in contact, the teeth in the alveolar segment are at a higher risk for internal or external resorption. In concomitant injuries, such as maxillary or mandibular fractures, early maxillomandibular fixation is accomplished with a technique that will allow for dual treatment of the dental and/or alveolar injury and the jaw injury (eg, arch bars and maxillomandibular fixation). Perform the more invasive open reduction if indicated. Avulsive injuries will often expose bone and jeopardize tooth support. Aim treatment at soft tissue coverage in the form of judicious mucosal advancement flaps. Consider early removal for teeth without bony support.

Treatment of Trauma to the Gingiva and Alveolar Mucosa

Traumatic injury to the oral soft tissue mainly consists of abrasion, contusion, and laceration. If these injuries are not addressed, they can place the underlying bony tissue at risk for devitalization. Frequently these injuries may alert the surgeon to underlying trauma. The ultimate goal of treatment is to reestablish vital soft tissue bony coverage.

Abrasion

An abrasion is a superficial wound wherein the epithelial or gingival tissue is rubbed, worn, or scratched. Treatment consists of local cleansing with a mild disinfectant soap for the skin and saline rinsing and/or irrigation of the gingiva. Antibiotic coverage is seldom necessary. Inspect the wound for possible foreign body (asphalt) accumulation, whichcould lead to unsightly accidental tattooing. If present, carry out meticulous removal within 12 hours, with care not to further inoculate the patient. The removal process includes a technique that aligns the surgical blade perpendicular to the direction of the abrasion. Contusion A contusion, a hemorrhage of subcutaneous tissue without laceration or break of overlying soft tissue, is similar to a bruising injury caused by blunt trauma. Treating gingival contusion includes local cleansing and observation. This injury may be associated with an underlying hematoma or ecchymotic formation, which is generally self-limiting. Antibiotic coverage is usually unnecessary.

Laceration

Lacerations are the most common form of facial injury. Gingival lacerations may involve an underlying bony defect. Treatment involves early cleansing and reapproximation. Remove devitalized tissue in a conservative manner, and suture in a manner that limits wound tension. Consider antibiotic and tetanus prophylaxis. More serious avulsive gingival wounds warrant close inspection of remaining tissue and underlying bony integrity. Exposure of any underlying bony defect may indicate localized keratinized sliding or advancement flaps. If nonkeratinized tissue is used for coverage, future grafting may be indicated.

TOPIC: BURNS AND FROSTBITE OF THE FACE

Burns of the face

Facial burns vary from relatively minor insults to severe debilitating injuries. Over 50% of burn injuries involve the head and neck region and can be caused by flame, electrical current, steam, hot substances, and chemicals.

It should be noted that burn injuries may take the skills of multiple specialties in a burn center. Objectives for reconstruction following a facial burn include restoration of function, comfort, and appearance. Functional concerns in these patients include airway patency, protection of the cornea, oral continence, and neck mobility. Burns may impart a tight masklike sensation to the face, distorting features and limiting facial expression. Appearance is altered by contractures, scarring, and pigmentary changes. The goal of the reconstructive surgeon is to minimize final deformity by restoring the patient to a near-normal appearance.

Anatomy

Before caring for a patient with facial burns, a thorough knowledge of skin anatomy and burn classification is essential. Skin covers the entire external surface of the human body and is the principle interface between the body and the surrounding environment. Skin serves as a protective barrier, preventing internal tissues from being exposed to trauma, ultraviolet radiation, temperature extremes, toxins, and bacteria. Other important functions include sensory perception, immunologic surveillance, thermoregulation, and control of insensible fluid loss.

The skin is composed of 3 principal layers: the epidermis, the dermis, and an underlying fatty subcutaneous layer. The epidermis and dermis are 2 mutually dependent layers that rest on the subcutaneous layer. The epidermis contains no blood vessels and is entirely dependent on the underlying dermis for nutrient delivery and waste disposal via diffusion through the dermoepidermal junction. The primary function of the dermis is to sustain and support the epidermis.

Epidermal appendages are intradermal epithelial structures lined with epithelial cells with the potential for division and differentiation. These serve an important role as the source of epithelial cells that are responsible for reepithelialization if overlying epidermis be removed or destroyed in certain situations, such as partial-thickness burns, chemical peeling, dermabrasion, or traumatic abrasions. Epidermal appendages include sebaceous glands, sweat glands, apocrine glands, mammary glands, and hair follicles. Sebaceous glands are in highest concentration on the face and scalp (as many as 900 per cm^2). Epithelial appendages are located deep within the dermis. In the face, they may even lie in the subcutaneous fat beneath the dermis. The deep location of these structures and their density in the face account for the remarkable ability of the face to reepithelialize even the deepest cutaneous wounds.

Skin varies in thickness based on anatomic location and on the sex and age of the individual. Skin is thickest on the palms and soles (approximately 1.5 mm thick), while skin is thinnest on the eyelids and postauricular region (approximately 0.5 mm thick). The skin of men is characteristically thicker than the skin of women in all anatomic locations. Children have relatively thin skin that progressively thickens until the fourth or fifth decade of life when thinning begins.

A burn involves the destruction of skin cells and sometimes the underlying structures of fascia, bone and muscle. A burn occurs when these structures absorb more heat than their capacity to dissipate. The injury to skin triggers inflammatory responses, and a variety of local cytokines cause a rapid accumulation of extravascular fluid.

Classification

Heat – The depth of the thermal injury is related to contact temperature, duration of contact of the external heat source, and the thickness of the skin. Because the thermal conductivity of skin is low, most thermal burns involve the epidermis and part of the dermis. The most common thermal burns are associated with flames, hot liquids, hot solid objects, and steam.

Electrical discharge – Electrical energy is transformed into heat as the current passes through poorly conducting body tissues. Electroporation (injury to cell membranes) disrupts membrane potential and function. The magnitude of the injury depends on the pathway of the current, the resistance to the current flow through the tissues, and the strength and duration of the current flow.

Chemical Burns – is irritation and destruction of human tissue caused by exposure to a chemical, usually by direct contact with the chemical or its fumes.

Burns are classified by degree or depth of injury.

A first-degree burn (superficial) involves minimal tissue damage and is confined to the epidermis. It can be caused by sunburn, scald, or flash flame. The appearance is dry and without blisters, with a pink color that is usually painful. Healing occurs over 5-10 days. Usually, no permanent scar occurs, but the tissue may discolor.

A second-degree burn (partial thickness) destroys the epidermis and a portion of the dermis and is typically painful. These burns can be caused by contact with hot liquids or solids, flash flame, or chemicals. The appearance is hyperemic, but may be pale, and moist with blisters. Although a variable depth of skin is initially lost, most partial-thickness burns, if treated appropriately, spontaneously reepithelialize from epithelial cells residing in remaining epithelial appendages. These burns usually heal in 10-14 days and no grafting is needed. A deeper partial-thickness burn may take more than 30 days to heal and can convert to a full-thickness injury if it becomes infected.

Third-degree burns (full-thickness burns) destroy both the epidermis and the entire dermis and are insensate because of the loss of sensory nerve endings. These burns can be caused by contact with hot liquids/solids, flames, chemicals or electricity. A full-thickness burn is dry and leathery with a gray, white, or translucent color; it turns brown or black in color, characteristic of an eschar. Most of these wounds do not heal spontaneously unless they are very small, and the resulting scars may be quite disfiguring. Operative intervention is indicated in full-thickness facial burns and in some partial-thickness burns.

Initial Evaluation

An initial evaluation and treatment should follow the same systematic approach as for all trauma patients: a primary survey, a secondary survey, followed by definitive treatment. For head and neck burns, the airway is a major concern because of potential inhalational injuries. A recent study looked at all of the risk factors for inhalational injuries and the need to intubate, and they concluded that patients who present with soot in the oral cavity, facial burns, or body burns should undergo fiberoptic laryngoscopy to look for laryngeal edema because they are much more likely to require intubation.

Facial burns are frequently associated with other morbidities. Assess the full extent of injury, including the total body surface area involved and the depth of wounding. Keep in mind that the head and neck represent 9% of the total body surface area in adults when using Wallace's rule of 9s. In neonates, the surface area is approximately 19%, and for each year of age 1% is subtracted from the head and given to the thighs until age 10. This consideration is important in that it informs future decisions regarding the determination of available donor sites for subsequent reconstruction and necessity for other procedures by different specialists. Adequate documentation, including photographs, is essential because facial burn reconstruction is typically a lengthy process involving multiple procedures.

Consider the circumstances surrounding the injury, patient age, significant comorbidities, drug and alcohol use, and available family and friend support structure. These factors influence patients' ability to cope with the severe psychological and social impairment often experienced following severe facial burns.

An open and honest discussion with patients and their families should detail the extent of injury and the goals of surgical intervention. Portray results realistically, including what can and cannot be accomplished. This is typically the beginning of a long-term physician-patient relationship and requires compassion, understanding, and guidance. Ensure that patients understand that dedication and patience will be required to endure a long reconstructive process that may take several or more years to complete. Finally, patients must realize that, however successful the reconstruction, they will not be returned to their pre-injury appearance.

Initial Treatment

For patients with severe burns, fluid resuscitation needs to be initiated using the Parkland or modified Brooke formulas to ensure that urine output is maintained at 0.5-1 mL/kg/hr. Ensure patients have appropriate pain control with acetaminophen, narcotics, and/or anxiolytics on a continuous basis. Patients with burns greater than 30% should be transferred to a burn unit. Those patients with burns of 15% or less or third degree burns of less than 2% may be treated on an outpatient basis if all other circumstances are favorable. Others should be treated as in-patients.

The aim of initial therapy is to prevent complications. Gently, under appropriate anesthesia, mechanically débride wounds. Do not perform sharp debridement initially, since it is often difficult to determine the exact depth of injury, and such action may injure underlying muscles or nerves. Then apply an antibiotic ointment such as silver sulfadiazine, mafenide (Sulfamylon), silver sulfadiazine, or 0.5% silver nitrate solution; nitrofurazone soluble dressing (Furacin) or a bilaminar temporary skin substitute (Biobrane) is also an option. The early application of Biobrane decreased pain significantly in one study.

Most authors recommend conservative therapy consisting of local wound care for approximately 10 days after wounding. Many facial wounds heal rapidly during this period because of the excellent blood supply and high density of epithelial appendages. Initially, estimating wound depth is often difficult. If wounds have not reepithelialized by 10 days or are clearly third-degree burns at initial assessment, no benefit is obtained by further temporizing.

Wounds unhealed within the first 2 weeks have a much higher likelihood of developing hypertrophic scarring and should undergo excision and grafting. Other considerations during this initial period include:

– flap coverage of exposed bone or cartilage;
– protection of exposed corneas;
– release of the mouth or neck to allow eating, communication, and access for subsequent anesthetic administration.

Operative Strategy

Appropriate timing of surgical intervention is essential to a successful outcome. Establishing priorities, placing functional concerns before cosmetic concerns, and rationing potential donor sites are key elements of this operative strategy. Allow the patient (rather than the surgeon) to prioritize reconstructive procedures, with the surgeon providing counseling regarding expected benefits and appropriate timing of procedures. Together, an overall master plan may be developed that may need modification as treatment progresses and patient status changes. Whenever possible, reduce anesthetic events by combining complementary procedures and coordinating the various surgical disciplines involved.

Generally, surgical intervention may be categorized as early or delayed; but, in reality, patient treatment is a continuum beginning at the time of injury. The earliest essential concern is to achieve a closed wound. This is necessary before entertaining any thoughts of reconstruction.

Techniques

Certain aesthetic principles are important when approaching reconstruction of the burned face. Reconstruction should proceed within facial aesthetic units. Treat each region (ie, forehead, eyes, nose, cheeks, ears, upper lip, lower lip and chin, neck) as an individual entity. Place scars at the boundaries of 2 aesthetic units. Often, sacrificing normal skin and replacing an entire aesthetic unit is better than replacing part of an aesthetic unit and having a scar coursing across that unit. Orientation of scars parallel to relaxed skin-tension lines (RSTLs) is also important. Some authors even stress symmetry to the point of recommending the same procedure for each side of the face. This may seem reasonable if the entire face were involved but is unnecessary if only part of the face is burned.

Primary repair is elected over waiting for scar maturation and secondary release of contractures that always results in a patchlike appearance on the applied grafts. Hypertrophic scarring seems to occur in wounds that take longer than 2-3 weeks to heal. Scar revision or staged excision may still be necessary later, and the patient should understand this at the outset.

Preoperative treatment includes daily antiseptic bath with chlorhexidine 0.05% and antibiotic ointment applied daily. Continue until day 10 and address those wounds that are not likely to heal within 2 weeks of injury. Once diagnosed with a deep injury, definitive treatment must be considered. Options include healing by granulation, primary closure, free skin grafts, local regional, or distant skin flaps. Burns that are clearly full-thickness are scheduled for excision and grafting within the next 7-10 days.

A careful operative technique is essential for a successful outcome. Perioperative antibiotics are administered. Use ocular lubricating ointments or corneal protectors prior to starting the procedure. After administering an appropriate level of anesthesia, prepare the wound for reconstruction. Start with wound cleansing with saline or dilute Betadine, judicious debridement, and achievement of meticulous hemostasis. Good hemostatic control can be gained with ligation, gentle pressure, application of a topical vasoconstrictor (eg, epinephrine), or bipolar electrocautery. Minimize electrocautery, as it creates devitalized tissue. Use of topical or injected epinephrine at the donor or recipient sites does not compromise outcome or tissue survival.

Early excision and grafting is the treatment of choice, it may be a 1-2 stage technique. Other options include AlloDerm (from cadaver skin, removing all cell components that cause rejection), TransCyte (human fibroblast-derived temporary skin substitute), or Integra (a temporary silicone epidermal substitute and an artificial dermal layer. If Integra is used, the neo-dermis forms in 2-4 weeks. The silicone layer is then removed and replaced with an ultrathin split-thickness skin graft (0.004 in). For larger burn defects, one might consider regional flaps, free flaps, and tissue expansion in addition to skin grafting.

A study by Li et al indicated that an integrated approach to total facial reconstruction can be beneficial in patients with postburn full facial deformities. Flap prefabrication, skin overexpansion, and reshaping of the facial contours through multistaged revisions were performed, with the prefabricated flap used to resurface the whole defect. Two patients underwent bone marrow mononuclear cell transplantation. Improvements in aesthetic and functional status scores were achieved, and the investigators reported that patients had good skin compliance, normal contours, and emotional expression.

Prioritized Specific Procedures

Skin grafting

Grafting is frequently the preferred choice when large full-thickness or deep partial thickness defects are involved. The propensity of split-thickness grafts for contracture is attributable to their limited content of dermal tissue.

Skin grafting is the simplest way to replace burned facial skin. Setting aside the face's donor site before all donor sites are used to resurface other body parts is important. Although even the best full-thickness graft loses some of its innate skin qualities, and although textural and pigmentary changes may persist, skin grafts advantageously lack bulk; therefore, they do not mask facial expression. Donor sites for full-thickness grafts are limited, and preoperative planning must consider the most efficient use of these donor sites. Match thickness, texture, pigmentation, and hair coverage as closely as possible. When grafting in children, remember that donor sites (eg, groin, axilla, thigh, chest) sprout hair at puberty, and this hair growth may be undesirable at the new location.

Full-thickness grafts

Choose donor sites for full-thickness grafts that are inconspicuous and that are easily closed primarily. Full-thickness grafts may be harvested from the upper eyelid, nasolabial fold, preauricular and postauricular regions, and the supraclavicular fossae. These donor sites are most often used to close facial or neck wounds or to resurface an existing scarred region.

When harvesting from the face, harvesting bilaterally to maintain facial symmetry is often aesthetically preferable, even if more skin than necessary is removed to cover the defect. Full-thickness donor sites used less frequently include hairless groin skin, dorsum of the foot, wrist-flexion creases, and elbow creases. Scars from skin grafts harvested from the wrist-flexion crease may resemble those seen with suicide attempts and should probably be avoided. Darker-pigmented grafts may be obtained from the prepuce, scrotum, and labia minora. Locations with avulsed or surgically removed skin are potential donor sites that are often overlooked.

Split-thickness grafts

Only use split-thickness grafts if harvested very thick with a significant portion of dermis included. This is because secondary contraction often occurs after split-thickness skin grafting, and the amount of secondary contraction is inversely proportional to the amount of dermis included in the graft. The scalp is a great donor site. For split-thickness grafts 0.018-0.025 inch, the scalp is then shaved. After complete hemostasis is achieved, the autografts are placed in aesthetic units.

Regional flaps

Regional and transpositional flaps introduce neighboring tissue with organic blood supply to the defect and have the advantage of providing skin of similar quality and pigmentation as the burned site. Transposition flaps are generally thin and pliable, although not as thin as skin grafts.

Free-tissue flaps

Free-tissue transfer may be required to cover exposed bone or cartilage or to introduce extra tissue when local tissues or skin grafts are inadequate or contraindicated. Defects greater than two-thirds of the anterior neck require distant tissue and are usually not amenable to local flaps. The most commonly used free-tissue flaps for facial reconstruction include latissimus dorsi, temporoparietal fascia, omentum, rectus abdominis, groin, radial forearm, dorsalis pedis, and parascapular flaps. These flaps require microvascular anastomosis and often subsequent debulking because of their large size. Free-tissue transfer for head and neck burn flap does have a success rate of 94%.

A total-face reconstruction was performed with a bilateral free scapula megaflap, but did result in the appearance of facial masking. Advances such as flap prefabrication, prelamination pre-expansion, chimeric flaps, and super-thin flaps have increased the quality of freeflaps. Prefabrication has resulted in thin flaps not requiring debunking in 64% of patients. In addition to the extra tissue, the radial forearm free flaps can be made sensate by incorporating the lateral ante brachial cutaneous nerve and attaching it to a branch of the trigeminal nerve. The free flap is not usually a first-line treatment option with the inherent disadvantages such as the complexity of the surgery with microvascular anastomose but is a valuable adjunct when required.

Tissue expansion

Another reconstruction option is recruitment of additional skin by tissue expansion. Benefits of this technique include generation of tissue that is similar to the defect in color, texture, and thickness. Furthermore, no donor site is created. In addition to standard tissue expansion techniques, tissue expansion has been used to expand the donor sites for full-thickness skin grafts. This allows harvesting of large full-thickness grafts from a relatively small donor area that subsequently may be closed primarily.

Grafts produced in this manner behave like smaller, nonexpanded full-thickness grafts. Tissue expansion and free-tissue transfer are commonly used together in reconstructive facial plastic surgery. Tissue expanders are placed under the subcutaneous place between the deep fascia and the muscle layer. Disadvantages of pre-expansion are the need for 2 operations and a significant time period for the progressive expansion.

Delayed or Secondary Treatments

Scar revision

Hypertrophic scarring, keloid formation, and contracture leading to disability and deformity continue to be challenging late complications of burn injuries and other traumas. Contractures can lead to permanent skeletal deformities and functional deficits. Methods of release include alphabet plasties (eg, Z, W, Y-V), skin grafts, skin expansion, or regional or free musculocutaneous or fasciocutaneous flaps to achieve adequate functional improvement.

Nonoperative treatments include massage, pressure garments, and cosmetic camouflage. Secondary skin grafting is simple, reliable and safe. Full-thickness skin grafts (groin, thighs, abdomen, postauricular, supraclavicular, upper eyelid, shoulder, cubital fossa) are preferred to split-thickness skin grafts (thigh, arms, buttocks) in postburn contracture releases.

Another option is using Integra, which may reduce the number of operations and the time period of treatment required by conventional procedures of multistage scar contracture release. With this technique, allografts were applied to control wound colonization and 48 hours later, the allografts were removed and Integra was applied to the wounds. Eighteen days after Integra application, ultrathin split thickness skin grafts were applied over the Integra dermal layer.–Dermabrasion is a useful technique for revising old burn scars. The ultimate goal for delayed scar revision is symmetry and a relatively normal appearance.

Human partial face allograft (face transplant)

The first of several human partial face allograft transplants occurred in 2005. After 18 months, the surgeons reported the outcome, stating that the patient has sensitivity to light touch and to heat and cold, which returned to normal at 6 months after transplantation. Motor recovery was slower, and labial contact allowing complete mouth closure was achieved at 10 months. Psychological acceptance of the graft progressed as function improved. Rejection episodes occurred twice after transplantation and were reversed. Face transplants still have long-term rejection risks and require immunosuppressants that have inherent risks, such as developing a carcinoma. Potentially, someday this procedure could be of benefit for some severely disfigured burn patients.

Specific Anatomic Sites

Eyelid

Eyelid reconstruction has highest priority because the lid functions to protect the cornea. Initiate reconstruction even in the presence of an immature scar. Scar release and subsequent grafting is the procedure of choice. Split-thickness grafts are a better match for the upper eyelid, and the optimal donor site is the contralateral upper lid if uninvolved with injury. Full-thickness grafts better approximate lower lid thickness, and decreased tendency for secondary contraction minimizes chances of postoperative ectropion. Generally, surgeons are recommended to release upper and lower eyelids in separate operative sessions.

Lip and chin region

This region is second in priority. Complications of burns in this region include drooling, microstomia, eating and communication difficulties, lip eversion, and oral hygiene inadequacy. General anesthesia administration in these patients can be difficult and dangerous. This region is not only important but is also one of the most difficult regions to reconstruct adequately. If at all possible, allow scars to fully mature with continuous pressure application so that tissues are soft and supple.

Often, patience yields equivalent or better results than might be expected with surgery. If operative intervention is necessary, thorough scar release with sufficient skin and soft tissue should be added to the lip and chin area, and this frequently requires a multiple-step operation. Full-thickness grafting is the procedure of choice. Reconstruction of very large defects of the lower lip is difficult. Consider cheek advancement flaps, rotation flaps using the residual lip, doublecross-lip flaps, or nasolabial flaps.

Large flaps that cover the chin and upper neck usually obscure the cervicomental angle and add to the appearance of pseudomicrogenia. Free flaps, such as the free radial forearm flap, are also a consideration, but the thickness of the flap should be reduced to cover the defect of the lower lip and chin area. To improve the cervicomental angle the lower border of the flap can be fixed with space of 1 finger-width from the hyoid bone. To form the labiomental sulcus, a space between the epidermis of the flap and muscle of the defective area was adhered. The addition of an alloplastic chin implant often improves the contour and provides better chin definition. Many authors place a feeding tube at the time of surgery and forbid eating, drinking, or speaking for a week postoperatively.

These are considered major procedures, and patients must receive long-term postoperative care. A modified dynamic mouth splint to minimize microstomia contractures is an important aspect of continued facial burn treatment. Also, compression therapy, scar massage, contact media, exercise, patient education, and neck splinting are used. The prevention and treatment of microstomia is difficult because of the aggressiveness of the contracting tissues and poor patient compliance.

Neck

Neck flexion contractures can result in significant posture alteration and in difficulty eating, communicating, and seeing. As with oral burns, general anesthesia administration in these patients may be difficult and dangerous. Splint the neck in patients suffering burns in the cervical region to prevent contracture and to make subsequent surgical intervention easier. Splints should be worn almost continuously. Refit and reapply splints promptly following each surgical intervention for at least 6 months.

Since correction of neck contractures generally requires multiple interventions, these procedures are usually begun early in the reconstructive plan. They are often complimentary procedures performed in tandem with other procedures.

In general, completely excise and resurface hypertrophic or widely restrictive scars. If less than a third of the neck is involved, local tissue transposition, Z-plasty, or W-plasty may be performed to reorient scars from vertical to transverse. For relatively minor burns involving less than two thirds of the neck, tissue expansion may provide an opportunity to replace all scarred tissue. When more than two thirds of the neck is involved, consider regional flaps, free flaps, and grafts. Skin grafts work well but require longer postoperative splinting and are not as aesthetically pleasing.

Cheek

Treat the burned cheek with a facial-pressure garment to assist in scar maturation. This is unnecessary, however, if the entire aesthetic unit is to be excised and grafted. Other options include tissue expansion of unburned cervical skin and local flaps. Postoperatively, a mask should be worn for 6 months if skin grafts have been applied.

Upper lip

The upper lip actually consists of 3 aesthetic units: the 2 lateral lip elements and the central philtral ridge. Usually, an incision is placed transversely at the root of the nose, allowing the upper lip to fall back into its normal position and maintain fullness. The secondary defect is subsequently skin-grafted. The philtral ridge may be augmented with dermal or cartilage grafts that otherwise might be discarded. Patients agreeable to permanent moustache wear might consider moustache reconstruction. This is performed with a free or islandized scalp flap based on the superficial temporal vessels. Achieving the correct downward-growth orientation of hair follicles is a crucial consideration.

Nose

Nasal reconstruction is of intermediate priority. Airway obstruction may require early intervention with scar release and skin grafting. Cosmetic nose reconstruction is better postponed until scar maturation is complete, but total resurfacing with a skin graft may be performed rather early. Since the nose is the central point of the face, many patients derive significant psychological benefit from satisfactory nasal reconstruction. Nasal reconstruction often restores a sense of humanity to individuals. Alar deformities may be treated with a local turndown flap resurfaced with a full-thickness skin graft or a composite graft from the ear.

Total nasal reconstruction may be performed with a forehead flap; however, many patients reject a forehead flap if the forehead is perceived as the only remaining normal region of the face. In these instances, a dorsalis pedis free flap or a Tagliacozzi tubed pedicled flap may be better alternatives.

Forehead

The forehead may be resurfaced completely as an aesthetic unit with a full-thickness skin graft. Tissue expansion is an alternative for a forehead burned on less than half its area. Exposed bone requires introduction of vascularized tissue (eg, temporoparietal fascial flaps) to accept a skin graft.

Generally, perform scalp reconstruction late in the sequence of events. If extensive bone is exposed early, the entire cranium may be resurfaced with a free flap that subsequently may be skin grafted. Scalp flaps may be used for less extensive injuries. Apply scalp flaps for specific indications and design them carefully, as they often leave donor sites as large as the covered defect. Temporoparietal fascial flaps may also be used as a vascularized surface for skin grafting, as with forehead defects. Punch grafts or hair transplants generally do not survive in previously burned or grafted skin that has a less-than-optimal blood supply. Tissue expansion is optimal if enough unburned hair-bearing skin remains.

Advantages of tissue expansion include:

– no donor site creation;
– donor skin of very similar color, thickness, and texture;
– high hair follicle survival rate.

Patients who reject these procedures may wear hairpieces or wigs.

Eyebrow reconstruction may proceed with strips of hair-bearing scalp transplanted as free grafts. Delicately handle and minimally defat these grafts because significant follicle loss may accompany this technique. Another option for bushier eyebrows is transference of a hair-bearing scalp segment as an island flap based on the superficial temporal vessels, as in upper-lip moustache reconstruction.

Ear

Ear reconstruction is generally performed last because the first choice for reconstruction uses available local tissue that should be allowed to achieve mature scarring. Indications for early intervention include cartilage exposure, in which case a turnover temporoparietal fascial flap may be used as a vascularized surface over which to apply a full-thickness skin graft.

Reconstructive options include local skin and fascial flaps with/without tissue expansion, ipsilateral axial temporoparietal fascial flap, contralateral temporoparietal, or an ipsilateral random fascial flaps. If the entire skin of the temporoauriculomastoid region is lost or badly scared with poor axial vascularity, a bilobed cervical flap or fascial free flap is an option. Rib cartilages are harvested from the right side and sculptured to create an ear frame. Another solution is the osseointegrated auricular prostheses, but it has a high cost and risk of potential complications over the long term.

Postoperative Care

After wound closure, scar hypertrophy may begin within 3 weeks. Scar hypertrophy is most exuberant over the next 3 months and is characterized by raised, red, itchy, tight scars. These scars then slowly regress during the following 12-24 months. At completion of this process, a mature scar remains that is characteristically white, soft, and flat. If possible, depending on clinical condition and patient insistence, exercise an observation protocol and patience until scars mature.

Postoperative care includes the continuous use of pressure garments or masks fitted early after injury. Regularly reassess, remold, and, as necessary, replace these garments to ensure continuous uniform pressure over the entire scarred area. Silicone pads are also sometimes used. No pressure should be placed on the auricle. These pressure devices lead to softer, less exuberant scars. Application of splints (Hartford or Larson device) to oral commissures and the neck may help prevent contracture development. Appropriate early measures may render subsequent procedures unnecessary or less difficult. Triamcinolone may be injected if hypertrophic scarring begins to develop.

The patient should begin scar massage as soon as wounds heal. Instruction in use of camouflage makeup may make a substantial impact on the lifestyle of these patients and their willingness to appear in public. Finally, instruct patients to wear moisturizing sunscreen and avoid direct sun exposure for the first 12 months following burn scar healing and skin grafting. These precautions avoid potential hyperpigmentation complications.

Electrical injuries

Electrical injuries have become a more common form of trauma with a unique pathophysiology and with high morbidity and mortality. They encompass several types, as follows: lightning injury, high-voltage injury, and low-voltage injury. Clinical manifestations range from transient unpleasant sensations without apparent injury to massive tissue damage. Some electrocutions are instantly fatal. Familiarity with the mechanisms of injury and the principles of therapy improves patient care.

Four classes of electrical injuries are as follows.

1. True electrical injuries. The person becomes part of the electrical circuit and has an entrance and exit site
2. Flash injuries. Superficial burns caused by arcs that burn the skin; no electrical energy travels through the skin
3. Flame injuries. Caused by ignition of the persons clothing by arc; electricity may or may not travel through the person's body
4. Lightning injuries. A unique type of injury that occurs at extremely high voltages for the shortest duration; the majority of electrical flow occurs over the body

Approximately 20% of all electrical injuries occur in children, with a bimodal peak incidence highest in toddlers and adolescents. Most electrical injuries that occur in children are at home, with extension cords (60-70%) and wall outlets (10-15%) being by far the most common sources in this age group. Electrical burns account for 2-3% of all burns in children that require emergency room care.

In adults, most electrical injuries happen at the workplace and constitute the fourth leading cause of work-related traumatic death. One third of all electrical traumas and most high-voltage injuries are job related. More than 50% of these occupational electrocutions result from power line contact (5-6% of all work-related deaths), and 25% result from using electrical tools or machines. The annual occupational death rate from electricity is 1 death per 100,000 workers, with a male-to-female ratio of 9:1.

Pathophysiology

The 3 major mechanisms of electricity-induced injury are as follows:

1. Electrical energy causing direct tissue damage, altering cell membrane resting potential, and eliciting muscle tetany.
2. Conversion of electrical energy into thermal energy, causing massive tissue destruction and coagulative necrosis.
3. Mechanical injury with direct trauma resulting from falls or violent muscle contraction.

Factors that determine the degree of injury include the magnitude of energy delivered, resistance encountered, type of current, current pathway, and duration of contact. Systemic effects and tissue damage are directly proportional to the magnitude of current delivered to the victim. Current flow (amperage) is directly related to voltage and inversely related to resistance, as dictated by Ohm law ($I=V/R$; where I=current, V=voltage, R=resistance). Of the parameters described by Ohm law, voltage usually can be determined and is used to gauge the potential magnitude of current exposure and, therefore, the magnitude of injury.

Electrical shock is classified as high voltage (>1000 volts) or low voltage (< 1000 volts). As a general rule, high voltage is associated with greater morbidity and mortality, although fatal injury can occur at household current (110 volts).

AC is substantially more dangerous than DC. Contact with AC may cause tetanic muscle contraction, preventing the victim from releasing the electrical source and, thereby, increasing the duration of contact and current delivery. Thoracic muscle tetany involving the diaphragm and intercostal muscles can result in respiratory arrest. The repetitive nature of AC increases the likelihood of current delivery to the myocardium during the vulnerable recovery period of the cardiac cycle, which can precipitate ventricular fibrillation. In contrast, DC usually causes a single violent muscle contraction, often thrusting the victim away from the source. Lightning is a unidirectional massive current that lasts from 1/10 to 1/1000 of a second, but often has voltages that exceed 10 million volts.

The most important difference between lightning and high-voltage electrical injuries is the duration of exposure to the current. Body tissues differ in their resistance. In general, tissues with high fluid and electrolyte content conduct electricity better. Bone is the tissue most resistant to the flow of electricity. Nerve tissue is the least resistant, and together with blood vessels, muscles, and mucous membranes offers a path of low resistance for electricity. Skin provides intermediate resistance and is the most important factor impeding current flow. Skin is the primary resistor against electrical current, and its degree of resistance is determined by its thickness and moisture. It varies from 1000 ohms for humid thin skin to several thousand ohms for dry calloused skin.

The current pathway determines which tissues are at risk and what type of injury is observed. Electrical current that passes through the head or thorax is more likely to produce fatal injury. Transthoracic currents can cause fatal arrhythmia, direct cardiac damage, or respiratory arrest. Transcranial currents can cause direct brain injury, seizure, respiratory arrest, and paralysis.

Electrothermal tissue injury results in tissue edema; therefore, the development of a compartment syndrome can occur in any body compartment. The leg is the site most commonly involved for the development of compartment syndrome.

The current intensity will have a probable effect in the body, determined by the factors discussed above. There may be individual variation on the energy dose for a specific effect. Less energy is generally required in children, who have more water content and thin skin and, hence, better conductivity and less resistance, and in patients under moist conditions.

Presentation

Clinical presentations range from a tingling sensation to a widespread tissue damage and even to instantaneous death. The external resistance to electric flow is offered primarily by the skin and appendages and by the internal resistance of all other tissues, including nerves, blood, muscles, tendons, bone, and fat. Since the skin resistance can be affected by moisture, electric current can be transmitted to deeper structures before it causes significant skin damage. Electric current may be retained in the bones, causing heat and leading to necrosis and coagulation of small- to medium-sized vessels within the muscles and other tissues, almost completely sparing the skin.

Often, the main symptom of an electrical injury is a skin burn. A specific type of burn, called the "kissing burn," occurs at the flexor creases and is related to the current flowing through the opposing skin at the joint when the flexor muscles contract due to tetany. Not all electrical injuries cause external damage; high-voltage injuries may cause massive internal burns and coagulation necrosis along with edema and compartment syndrome. Lightning injury usually causes superficial surface burns.

The heart can be damaged by direct necrosis to the myocardium and by cardiac dysrhythmias. Cardiac arrhythmia can occur and range from benign to fatal. High voltage or DC current usually causes asystole, and AC current usually causes ventricular fibrillation. Ventricular fibrillation is the most common fatal arrhythmia, occurring in up to 60% of patients in whom the current pathway goes from one hand to the other hand. The most common abnormalities seen on an electrocardiogram (ECG) are sinus tachycardia, nonspecific ST- and T-wave changes, heart blocks, and prolongation of the QT interval.

The most common electrical injury seen in children younger than 4 years is the mouth burn. These burns may cause facial deformities and growth problems of the teeth, jaw, and face. If a current travels close to the eyes, it may lead to cataracts. Cataracts can develop within days of the injury or years later.

Acute renal failure can complicate the hospital course due to acute tubular necrosis secondary hypovolemia from third spacing and huge volume shift. Rhabdomyolysis that results from massive tissue necrosis can also cause pigment-induced renal failure.

About two thirds of patients struck by lightning have ruptured eardrums. Autonomic dysfunction can cause pupils that are fixed and dilated or asymmetric, and this finding should not be used as a reason to stop resuscitation. The "locking-on" phenomenon refers to a refractory state of neuromuscular stimulation with tetanic contractions that prevents the victim's hand to release the electrical source.

Electric burn patients may present with bone fractures from either severe muscle contractions (eg, avulsion fractures) or as a result of falls. This is more commonly seen in upper limb long bones and in vertebrae.

Chemical Burns

A chemical burn is irritation and destruction of human tissue caused by exposure to a chemical, usually by direct contact with the chemical or its fumes. Chemical burns can occur in the home, at work or school, or as a result of accident or assault. Although few people in the United States die after contact with chemicals in the home, many substances common in both living and storage areas can do serious harm.

Many chemical burns occur accidentally through misuse of products such as those for hair, skin, and nail care. Although injuries do occur at home, the risk of sustaining a chemical burn is much greater in the workplace, especially in businesses and manufacturing plants that use large quantities of chemicals.

Most chemical burns are caused by either strong acids or strong bases (for example, hydrochloric acid or sodium hydroxide). Acids damage and kill cells by coagulating cells while bases liquefy cells. Prolonged exposure can severely damage human tissues and, if the patient survives, leads to scarring and disability. Other chemicals like oxidants and certain metals may also produce similar chemical burns. Limiting the time of exposure to any of these chemicals can greatly reduce their damaging effects.

Unfortunately, some chemical burn agents are designed to harm people (chemical agents used in wars and in terrorist attacks). It is not the scope of this article to cover these agents.

Chemical Burn Causes

Most chemicals that cause burns are either strong acids or bases. A glance at the medical information on the labels of dangerous chemicals usually confirms the expected toxicity. Common sense precautions and consumer education can reduce the risk of injury.

A variety of common household products that may cause chemical burns are as follows:

1. Bleach
2. Concrete mix
3. Drain or toilet bowl cleaners
4. Metal cleaners
5. Pool chlorinators
6. Chemical Burn Symptoms

All chemical burns should be considered potential medical emergencies.

Most chemical burns occur on the face, eyes, hands, arms, and legs. Usually a chemical burn will be relatively small and will require only outpatient treatment. Chemical burns can be deceiving, however. Some agents can cause deep tissue damage that is not readily apparent when people first look at it.

- Tissue damage from chemical burns depends on several factors:
- The strength or concentration of the agent
- The site of contact (eye, skin, mucous membrane)
- Whether swallowed or inhaled

- Whether or not skin is intact
- With the quantity of the chemical
- The duration of exposure
- How the chemical works
- The length of time to washing (decontamination)
- Signs and symptoms of chemical burns include the following:
- Redness, irritation, or burning at the site of contact
- Pain or numbness at the site of contact
- Formation of blisters or black dead skin at the contact site
- Vision changes if the chemical gets into the eyes
- Cough or shortness of breath
- Vomiting

In severe cases, a person may develop any of the following symptoms:

- Low blood pressure
- Faintness, weakness, dizziness
- Shortness of breath or severe cough
- Headache
- Muscle twitching or seizures
- Cardiac arrest or irregular heartbeat

Chemical burns can be very unpredictable. Death from a chemical injury, although rare, can occur.

Chemical Burn Diagnosis

In the emergency department, a person can expect the following:

- Initial evaluation and stabilization
- Rapid evaluation of the chemical's ability to damage tissue
- Determination of the extent of injury
- Blood tests and other studies to determine if the patient should be admitted to the hospital
- Determination of additional injuries and treatment

Specific medical treatment depends on the chemical that the patient was exposed to. Some of the general steps taken to medically treat chemical burns are as follows:

IV fluids may be needed to normalize blood pressure and heart rate as any type of burn (fire, chemical, sun exposure) often results in dehydration of the patient.

The IV access may also be used for any medications needed to treat pain or protect against infection.

Decontamination will begin (likely water irrigation).

Some people may be an antidote to counteract the chemical, if appropriate.

Antibiotics often are not needed for minor chemical burns.

Wounds will be cleaned and bandaged with medicated creams and sterile wraps as needed.

Consultation with other medical specialists may be done if indicated.

Pain in a burn can often be severe. Adequate pain control will be addressed by the doctor.

If there is any indication of breathing problems, a breathing tube may be placed in the patient's airway to help maintain the airway and provide adequate ventilation.

Frostbite

Frostbite occurs when tissues freeze. This condition happens when you are exposed to temperatures below the freezing point of skin. Hypothermia is the condition of developing an abnormally low body temperature. Frostbite and hypothermia are both cold-related medical emergencies.

Although frostbite used to be a military problem, it is now a civilian one as well. The nose, cheeks, ears, fingers, and toes are most commonly affected. Everyone is susceptible, even people who have been living in cold climates for most of their lives. Some groups of people at greatest risk for frostbite and hypothermia include those:

- who spend a lot of time outside, such as the homeless, hikers, hunters, etc.;
- under the influence of alcohol;
- who are elderly without adequate heating, food, and shelter;
- who are exhausted or excessively dehydrated;
- who are mentally ill.
- What Causes Frostbite?

In conditions of prolonged cold exposure, the body sends signals to the blood vessels in the arms and legs telling them to constrict (narrow). By slowing blood flow to the skin, the body is able to send more blood to the vital organs, supplying them with critical nutrients, while also preventing a further decrease in internal body temperature by exposing less blood to the outside cold.

As this process continues and the extremities (the parts farthest from the heart) become colder and colder, a condition called the hunter's response is initiated. The body's blood vessels are dilated (widened) for a period and then constricted again. Periods of dilatation are cycled with times of constriction in order to preserve as much function in the extremities as possible. However, when the brain senses that the person is in danger of hypothermia (when the body temperature drops significantly below 98.6 F [37 C]), it permanently constricts these blood vessels in order to prevent them from returning cold blood to the internal organs. When this happens, frostbite has begun.

Frostbite is caused by two different means: cell death at the time of exposure and further cell deterioration and death because of a lack of oxygen.

In the first, ice crystals form in the space outside of the cells. Water is lost from the cell's interior, and dehydration promotes the destruction of the cell.

In the second, the damaged lining of the blood vessels is the main culprit. As blood flow returns to the extremities upon rewarming, it finds that the blood vessels themselves are injured, also by the cold. The vessel walls become permeable and blood leaks out into the tissues. Blood flow is impeded and turbulent and small clots form in the smallest vessels of the extremities. Because of these blood flow problems, complicated interactions occur, leading to inflammation that causes further tissue damage. This injury is the primary determinant of the amount of tissue damage that occurs in the end.

It is rare for the inside of the cells themselves to be frozen. This phenomenon is only seen in very rapid freezing injuries, such as those produced by frozen metals.

Three frostbite stages are:

1. The first degree – irritates the skin
2. The second degree – blisters but has no major damage
3. The third degree – involves all layers of the skin and causes permanent tissue damage

Varieties of frostbite classification systems have been proposed. The easiest to understand, and perhaps the one that gives the best clues to outcome, divides frostbite into two main categories: 1) superficial and 2) deep.

In superficial frostbite, you may experience burning, numbness, tingling, itching, or cold sensations in the affected areas. The regions appear white and frozen, but if you press on them, they retain some resistance.

In deep frostbite, there is an initial decrease in sensation that is eventually completely lost. Swelling and blood-filled blisters are noted over white or yellowish skin that looks waxy and turns a purplish blue as it rewarms. The area is hard, has no resistance when pressed on, and may even appear blackened and dead.

The affected person will experience significant pain as the areas are rewarmed and blood flow reestablished. A dull continuous ache transforms into a throbbing sensation in 2 to 3 days. This may last weeks to months until final tissue separation is complete.

At first, the areas may appear deceptively healthy. Most people do not arrive at the doctor with frozen, dead tissue. Only time can reveal the final amount of tissue damage.

There are milder conditions related to frostbite, including frostnip, chilblains, and trench foot.

Frostnip refers to the development of tingling sensations (paresthesias) that occur due to cold exposure. They disappear upon rewarming without any tissue damage.

Chilblain (or pernio) refers to a localized area of tissue inflammation that appears as swollen and reddish or purple. These develop in response to repeated exposure to damp, cold conditions above the freezing point. Chilblains may itch or be painful.

Trench foot was described in World War I as a result of repeated exposure to dampness and cold and exacerbated by tight boots. The affected feet are reddened, swollen, painful or numb, and may be covered with bleeding blisters. This condition is still observed in some homeless persons today.

The doctor will take a history in order to gather information on the events of the exposure and the medical condition prior to the cold injury.

The doctor will take note of the vital signs, including temperature, pulse, blood pressure, and respiratory rate in order to exclude or treat any immediate life threats such as hypothermia or severe infection.

X-rays or other imaging studies may be performed, but they probably will be deferred until weeks later when they are more useful to the treatment team.

The doctor will collect data in order to classify the injury as superficial or deep and the prognosis as favorable or poor.

A good prognosis is heralded by intact sensation, normal skin color, blisters with clear fluid, the ability to deform the skin with pressure, and the skin becoming pink when thawed.

Blisters with dark fluid, skin turning dark blue when thawed, and an inability to indent the skin with pressure indicate a poor prognosis.

The first step for a person who may have frostbite is to call for medical help. If you are in an area that has an emergency medical alert system such as 911, while attending to the injured person, have someone call 911 and best explain the condition of the patient. Remove all wet clothing from the affected area, and elevate the area higher than the heart if possible to avoid swelling. Keep the person dry and warm. If they are immobile, and unable to walk try to keep the person busy with conversation. Keep the body warm and dry if possible.

After initial life-threatening problems are excluded or managed, rewarming is the highest priority in medical care.

This is accomplished in the hospital rapidly in a circulating water bath heated to 40^0 C to 42^0 C and continued until the thaw is complete (usually 15 to 30 minutes).

Narcotic pain medications may be given because this process is very painful.

Because dehydration is very common, IV fluids may also be given.

After rewarming, post-thaw care is undertaken in order to prevent infection and a continuing lack of oxygen to the area.

The clear blisters are debrided (dead tissue is removed) while the bloody ones are often left intact so as not to disturb the underlying blood vessels.

When there is a great risk of damage enough to require amputation (for example, multiple digits, or proximal amputation), tPA (tissue plasminogen activator) may be given into an artery to reduce the incidence of blood clots. This can only be given to people who are not at risk for significant bleeding complications.

A tetanus booster is given if needed.

People with frostbite are hospitalized for at least 1 to 2 days to determine the extent of injury and to receive further treatment.

Aloe vera cream is applied every 6 hours, and the area is elevated and splinted.

Ibuprofen or a similar anti-inflammatory OTC medication may be given to decrease inflammation.

For deep frostbite, daily water therapy in a 40 C (104 F) whirlpool bath will be performed in order to remove any dead tissue.

There are a number of experimental therapies for frostbite, many of which aim to further treat the inflammation or decreased blood flow seen in frostbite. As of yet, none of these treatments has proven beneficial.

Symptoms follow a predictable pathway. Numbness initially is followed by a throbbing sensation that begins with rewarming and may last weeks to months. This is then typically replaced by a lingering feeling of tingling with occasional electric-shock sensations. Cold sensitivity, sensory loss, chronic pain, and a variety of other symptoms may last for years.

The treatment of frostbite is done over a period of weeks to months. Definitive therapy such as surgery may not be performed for up to 6 months after the initial injury. Therefore, it is important to establish a working relationship between you and your doctor that will continue throughout the healing process.

TOPIC: MANDIBULAR FRACTURES

Causes

The causes of maxillofacial fractures have changed over the past decades and will continue. Different societies and cultures show different patterns of facial trauma. Varying socioeconomic conditions combined with behavioral differences, however, make the aggregate comparison of mandibular fractures difficult. Obtaining analysis data from various regions can increase the understanding of facial trauma and allow for optimization of treatment. Demographic information on maxillofacial injuries has changed with the onset of motor vehicle seat belt and airbag laws, reduced speed limits, and increasing urban violence. Many countries, including Brazil, India, and China, are contributing to this demographic data, along with reports from the United States, England, Germany, the Netherlands, and the Scandinavian countries. The main causes of mandibular fractures worldwide include motor vehicle accidents, interpersonal violence, falls, and sports-related injuries. Past data from industrialized or developed nations with large numbers of vehicles indicated that multiple mandibular fractures were occurring with severe concomitant facial fractures and associated nonmaxillofacial injuries, situations that required extensive treatment. In contrast, assaults and falls have become the predominant mechanism for facial injuries. It is important to note, however, that local laws and socioeconomic conditions in developed versus developing countries create mixed results for case by case studies. Work-related accidents, firearms, and pathologic conditions are also causative factors.

Location of mandibular fractures

In the cases evaluated for fracture location, the incidences were as follows: angle (30%), condyle (23%), symphyses (22%), body (18%), ramus (2%), and coronoid process (1%). As noted, there are many variables, but generalization fractures that occur in the symphyses, condyle, and angle do not differ much in incidence, and fractures of the ramus and coronoid process are rare.

Classification of mandibular fractures

Dictionary classification

For the sake of this discussion, the following fracture terms have been adopted from Dorland's Illustrated Medical Dictionary:

1. Simple or closed: A fracture that does not produce a wound open to the external environment, whether it be through the skin, mucosa, or periodontal membrane

2. Compound or open: A fracture in which an external wound, involving skin, mucosa, or periodontal membrane, communicates with the break in the bone

3. Comminuted: A fracture in which the bone is splintered or crushed

4. Greenstick: A fracture in which one cortex of the bone is broken, the other cortex being bent

5. Pathologic: A fracture occurring from mild injury because of preexisting bone disease

6. Multiple: A variety in which there are two or more lines of fracture on the same bone not communicating with one another

7. Impacted: A fracture in which one fragment is firmly driven into the other

8. Atrophic: A spontaneous fracture resulting from atrophy of the bone, as in edentulous mandibles

9. Indirect: A fracture at a point distant from the site of injury

10. Complicated or complex: A fracture in which there is considerable injury to the adjacent soft tissue or adjacent parts; may be simple or compound

Classification by anatomic region

Mandibular fractures are also classified by the anatomic areas involved, as follows: symphysis, body, angle, ramus, condylar process, coronoid process, and alveolar process. Dingman and Natvig have defined these regions as follows:

1. Midline: Fractures between central incisors

2. Parasymphyseal: Fractures occurring within the area of the symphysis

3. Symphysis: Bounded by vertical lines distal to the canine teeth

4. Body: From the distal symphysis to a line coinciding with the alveolar border of the masseter muscle (usually including the third molar)

5. Angle: Triangular region bounded by the anterior border of the masseter muscle to the posterosuperior attachment of the masseter muscle (usually distal to the third molar)

6. Ramus: Bounded by the superior aspect of the angle to two lines forming an apex at the sigmoid notch

7. Condylar process: Area of the condylar process superior to the ramus region

8. Coronoid process: Includes the coronoid process of the mandible superior to the ramus region

9. Alveolar process: The region that would normally contain teeth Kazanjian and Converse have classified mandibular fractures by the

presence or absence of serviceable teeth in relation to the line of fracture. They thought that their classification was helpful in determining treatment.

Three classes were defined:

Class I: Teeth are present on both sides of the fracture line.

Class II: Teeth are present on only one side of the fracture line.

Class III: The patient is edentulous.

They thought that class I fractures could be treated by a variety of techniques, using the teeth for monomaxillary or intermaxillary fixation. Class II fractures, usually involving the condyle-ramus angle or partially edentulous body of the mandible, require intermaxillary fixation. Class III fractures require prosthetic techniques, open reduction methods, or both for stabilization.

Rowe and Killey have divided mandibular fractures into two classes: (1) those not involving basal bone; and (2) those involving basal bone. The first class primarily is comprised of alveolar process fractures. The second class is divided into single unilateral, double unilateral, bilateral, and multiple.

Kruger has classified mandibular fractures into simple, compound, and comminuted. Kruger and Schilli took into account many of the aforementioned classifications described and developed four categories of mandibular fractures:

1. Relation to the external environment
    a. Simple or closed
    b. Compound or open
2. Types of fractures
    a. Incomplete
    b. Greenstick
    c. Complete
    d. Comminuted
3. Dentition of the jaw with reference to the use of splints
    a. Sufficiently dentulous jaw
    b. Edentulous or insufficiently dentulous jaw
    c. Primary and mixed dentition
4. Localization
    a. Fractures of the symphysis region between the canines
    b. Fractures of the canine region
    c. Fractures of the body of the mandible d. Fractures of the angle of the mandible in the third molar region
    e. Fractures of the mandibular ramus between the angle of the mandible and sigmoid notch

f. Fractures of the coronoid process
g. Fractures of the condylar process

Diagnosis of mandibular fractures

Patient history

A thorough history is imperative for the proper diagnosis of mandibular fractures. The patient's health history may reveal preexisting systemic bone disease, neoplasia with potential metastases, arthritis and related collagen disorders, nutritional and metabolic disorders, and endocrine diseases that may cause or be directly related to the fractured jaw. The history also reveals significant medical and psychiatric problems that will influence the management of the patient and perhaps even dictate treatment modalities.

A history of temporomandibular joint (TMJ) dysfunction can have significant legal and post-treatment ramifications.

A number of clinicians and radiologists have experienced problems by not obtaining an adequate history of previous mandibular trauma or fracture.

The type and direction of traumatic force can be extremely helpful for the diagnosis. Fractures sustained in MVAs are usually different from those sustained in IPVs. Because the magnitude of the force can be much greater, victims of automobile and motorcycle accidents tend to have multiple, compound, comminuted mandibular fractures, whereas the patient hit by a fist may sustain a single, simple nondisplaced fracture.

The object that caused the fracture can also influence the type and number of fractures. A blow from a broad blunt object (e.g., a two by four piece of wood) may cause several fractures (e.g., symphysis, condyle) because the impact of the force is sustained throughout the bone, whereas a smaller, well-defined object (e.g., hammer, pipe) may cause a single comminuted fracture becausethe impact of the force is concentrated in a smaller area.

Knowing the direction of force can help the clinician diagnose concomitant fractures. An anterior blow directly to the chin can result in bilateral condylar fractures and an angled blow to the parasymphysis may cause a contralateral condylar or angle fracture. A patient with teeth clenched together at the moment of impact is more likely to have dental and alveolar process fractures than basal bone fractures. Even knowing where the patient was sitting in an automobile may aid in the diagnosis of mandibular or other injuries. Chest injuries caused by noncollapsible steering wheels, facial fractures caused by striking unpadded dashboards, and facial lacerations from non–safety glass are a few examples of predictable injuries that have been eliminated by the use of seat belts and more effective automotive safety engineering.

Clinical examination

Initial evaluation is part of the secondary survey when adhering to the Advanced Trauma Life Support (ATLS) protocol. Stabilization of the airway may require tracheotomy in severe crushing injuries, such as the hallmark flail mandible. The mandible should be evaluated after the patient is stabilized and more life-threatening injuries have been addressed. The signs and symptoms of mandibular fractures are as follows.

Change in Occlusion. Any change in occlusion is highly suggestive of mandibular fracture. The clinician should ask the patient whether his or her bite feels different. A change in occlusion can result from fractured teeth, fractured alveolar process, fractured mandible at any location, and trauma to the TMJ and muscles of mastication.

Post-traumatic premature posterior dental contact or an anterior open bite may result from bilateral fractures of the mandibular condyle or angle and from maxillary fractures with inferior displacement of the posterior maxilla. A posterior open bite may occur with fractures of the anterior alveolar process or parasymphyseal fractures. A unilateral open bite may occur because of ipsilateral angle and parasymphyseal fractures . A posterior crossbite can result from midline symphyseal and condylar fractures, with splaying of the posterior mandibular segments.

Retrognathic occlusion is associated with condylar or angle fractures (and forward displaced maxillary fractures); prognathic occlusion can occur with effusion of the TMJ joints and with protective forward posturing of the mandible (also with retropositioning of the maxilla).

These are only a few of the many occlusal disharmonies that can exist, but any change in occlusion has to be considered the primary diagnostic sign of mandibular fracture.

Anesthesia, Paresthesia, or Dysesthesia of the Lower Lip. Although changes in sensation in the lower lip and chin may be related to chin and lip lacerations and blunt trauma, numbness in the distribution of the inferior alveolar nerve after trauma is almost pathognomonic of a fracture distal to the mandibular foramen. Conversely, most nondisplaced fractures of the mandibular angle, body, and symphysis are not characterized by anesthesia, so the clinician must not use lip anesthesia as the sole feature in diagnosis.

Abnormal Mandibular Movements. Most patients with a fractured mandible have limited opening and trismus because of guarding of the muscles of mastication. However, certain mandibular fractures or associated facial fractures result in predictable abnormal mandibular movements. A classic example is deviation on opening toward the side of a mandibular condylar fracture.

Because lateral pterygoid muscle function on the unaffected side is not counteracted on the opposite side by the nonfunctioning lateral pterygoid muscle, deviation results. Inability to open the mandible may be caused by the impingement of the coronoid process on the zygomatic arch from fractures of the ramus and coronoid process or from depression of a zygomatic arch fracture.

Inability to close the jaw can be the result of fractures of the alveolar process, angle, ramus, or symphysis, causing premature dental contact. Lateral mandibular movements may be inhibited by bilateral condylar fractures and fractures of the ramus with bone displacement.

Change in Facial Contour and Mandibular Arch Form. Although facial contour may be masked by swelling, the clinician should examine the face and mandible for abnormal contours. A flattened appearance of the lateral aspect of the face may be the result of a fractured body, angle, or ramus. A deficient mandibular angle can occur with unfavorable angle fractures in which the proximal fragment rotates superiorly. A retruded chin can be caused by bilateral parasymphyseal fractures. The appearance of an elongated face may be the result of bilateral subcondylar, angle, or body fractures, allowing the anterior mandible to be displaced downward. Facial asymmetry should alert the clinician to the possibility of a mandibular fracture. The same holds true for mandibular arch form. If there is a deviation from the normal U-shaped curve of the mandible, fracture should be suspected.

Lacerations, Hematoma, and Ecchymosis. Trauma significant enough to cause loss of skin or mucosal continuity, or subcutaneous-submucosal bleeding, can result in trauma to the underlying mandible. Lacerations should be carefully inspected before closure. The direction and type of fracture may be visualized directly through the laceration, with the clinician thereby obtaining diagnostic information that might be impossible to ascertain clinically or radiographically. The common practice of closing facial lacerations before treating underlying fractures should be discouraged from diagnostic and treatment standpoints. The diagnostic sign of ecchymosis in the floor of the mouth indicates a mandibular body or symphyseal fracture.

Loose Teeth and Crepitation on Palpation. A thorough examination of the teeth and supporting bone can help diagnose alveolar process, body, and symphyseal fractures. A force strong enough to loosen teeth can fracture the underlying bone. Multiple fractured teeth that are firm indicate that the jaws were clenched during traumatic insult, thus lessening the effect on the supporting bone. The clinician should palpate the mandible using both hands, with the thumb on the teeth and the fingers on the lower border of the mandible. By slowly and carefully placing pressure between the two hands, the clinician can detect crepitation in a fracture. However, this simple diagnostic technique is often overlooked in favor of extensive (and expensive) radiologic diagnostic methods.

Dolor, Tumor, Rubor, and Color. Pain, swelling, redness, and localized heat have been noted as signs of inflammation since the time of the ancient Greeks. All these findings are excellent primary signs of trauma and can greatly increase the index of suspicion for a mandibular fracture.

Radiologic examination

The following radiologic studies are helpful in the diagnosis of mandibular fractures:

1. Panoramic radiograph
2. Lateral oblique radiograph
3. Posteroanterior radiograph
4. Occlusal view
5. Periapical view
6. Reverse Towne’s view
7. TMJ, including tomograms
8. CT – high-resolution spiral or helical CT

The single most informative radiologic study used in diagnosing mandibular fractures is the panoramic radiograph, showing the entire mandible, including condyles

The advantages are simplicity of technique, ability to visualize the entire mandible in one radiograph, and generally good detail, but there are several disadvantages:

– the technique usually requires the patient to be upright (machines that allow the patient to be prone are available), which may make it impractical in the severely traumatized patient;

– it is difficult to appreciate buccal-lingual bone displacement or medial condylar displacement;

– fine detail is lacking in the TMJ, symphysis (depending on the type of equipment), and dental and alveolar process regions.

A secondary but important disadvantage is that panoramic radiographic equipment is not available in all hospital radiology departments. In addition, because of the sensitivity of the panoramic radiographic technique, superimposition of structures can lead to interpretation errors.

The lateral oblique view of the mandible can be helpful in the diagnosis of ramus, angle, and posterior body fractures. The technique is simple and can be done in any radiology department. The condyle region is often unclear, as are the premolar and symphysis regions. The Caldwell posteroanterior (PA) view demonstrates any medial or lateral displacement of fractures of the ramus, angle, body, and symphysis.

The condylar region is not well demonstrated on this view, but midline or symphyseal fractures can be well visualized. The anteroposterior view is occasionally used for patients who cannot be placed in the supine position; however, considerable magnification and distortion occur with this view. The mandibular occlusal view demonstrates discrepancies in the medial and lateral position of body fractures and also shows anteroposterior (AP) displacement in the symphysis region . The reverse Towne view is ideal for showing medial displacement of condyle and condylar neck fractures . Transcranial lateral views of the TMJ are helpful in detecting condylar fractures and anterior displacement of the condylar head. Periapical dental films show the most detail and can be used for nondisplaced linear fractures of the body and alveolar process and dental trauma. Plain tomograms can be used in an AP and lateral direction when greater detail is necessary. The traditional CT scan is ideal for condylar fractures that are difficult to visualize; however, greater expense and radiation exposure limit its use to cases that cannot be diagnosed with plain films and panoramic tomography With the advent of newer technology, helical CT (HCT) is faster, more accurate, and less expensive and delivers decreased radiation exposure as compared with conventional CT scans. In summary, as with most other imaging procedures, it is usually optimal to have views of the mandible in two planes oriented at 90 degrees to each another. Ideal imaging includes sections in the axial and coronal planes but practical considerations of time, the patient’s condition, and cost mandate selectivity. Thus, the history, physical examination, and understanding which structures are best shown by certain imaging methods dictate the proper radiologic technique.

General principles of treatment

Throughout this text, specific chapters have been devoted to preoperative, operative, and postoperative treatment of facial fractures. This section presents general principles used in the management of mandibular fractures.

1. The patient's general physical status should be carefully evaluated and monitored before any consideration of treating mandibular fractures.

It must be emphasized that any force great enough to cause a fractured mandible is capable of injuring other organ systems in the body. This is obvious when dealing with massive crush injuries of the face, with concomitant multiorgan system involvement. However, it is easy for the clinician to focus on an obvious isolated mandibular fracture without noting a fractured cervical spine, subdural hematoma, pneumothorax, cardiac tamponade, or ruptured spleen.

2. Diagnosis and treatment of mandibular fractures should be approached methodically, not with an emergency-type of mentality. Patients rarely die of mandibular fractures, so the clinician has time to evaluate the nature and extent of mandibular injuries carefully and thoroughly. Diagnosis on the basis of the history and local physical and radiologic examination should be expedited in an orderly and efficient manner, and treatment should be instituted in a controlled fashion. However, this is not to condone prolonged unnecessary delay, which can increase the potential for infection and nonunion.

3. Dental injuries should be evaluated and treated concurrently with treatment of mandibular fractures.

Teeth are often injured with mandibular fractures and, although the teeth may not have to be restored immediately, dental knowledge is vitally important for determining which teeth can and should be maintained.

a. Fractured teeth can become infected and jeopardize bone union; however, an intact tooth in the line of fracture that is maintaining bone fragments can be protected with antibiotic coverage .

b. A second molar on an otherwise edentulous posterior fracture segment should be maintained to prevent superior displacement of the fragment in intermaxillary fixation.

c. Mandibular canines are the cornerstone of occlusion and should be maintained at all costs.

d. Some teeth are not critical to restoration and can be removed when their prognosis is doubtful and when maintenance may adversely affect fracture treatment. For example, a lone mandibular incisor adds little to future bridge or partial denture construction; however, a single molar tooth in an otherwise edentulous posterior quadrant can be critical to dental rehabilitation.

e. Some fractured teeth cannot be salvaged, no matter how critical they may be. For example, a molar tooth may be split mesially and distally, so reconstruction would be impossible. Maintenance of this tooth during intermaxillary fixation could result in severe discomfort and perhaps infection.

4. Reestablishment of occlusion is the primary goal in the treatment of mandibular fractures.

Probably because of the mandible's excellent blood supply, nonunion of mandibular fragments is rare, so it is apparent that bone fragments do not have to be in tight approximation to heal. In addition, in most cases, facial aesthetics will not be adversely affected by slight fragment displacement. However, function can be seriously compromised when improper treatment results in malocclusion. Impressive appearing radiographic bone adaptation should not be the primary treatment goal.

5. With multiple facial fractures, mandibular fractures should be treated first. The old adage "inside out and from bottom to top" applies to the proper sequence to follow when treating facial fractures. To build a foundation on which the facial bones can be laid, the mandible should be reconstructed first, although with the use of rigid fixation, deviation from this principle can be allowed. All intraoral surgery should be done before any extraoral open reductions or suturing of facial lacerations. Lip and skin wounds that have been meticulously closed in an emergency room are often inadvertently, or even necessarily, reopened during the treatment of mandibular fractures. Gross debridement and control of hemorrhage should be combined with temporary measures to reapproximate extraoral wounds, thus allowing definitive treatment to be carried out after the intraoral procedures are completed.

6. Intermaxillary fixation time should vary according to the type, location, number, and severity of the mandibular fractures, the patient's age and health, and the method used for reduction and immobilization. Historically, a period of 6 weeks of intermaxillary fixation has been used to allow healing to occur. However, this length of time is only empirical and should vary with the patient and clinical situation. A simple, nondisplaced, greenstick mandibular fracture in a healthy child would certainly require less intermaxillary fixation time than multiple, grossly comminuted, compound mandibular fractures in an older unhealthy patient. With the advent of rigid fixation techniques, intermaxillary fixation may be eliminated or maintained with light elastics for short periods.

7. Prophylactic antibiotics should be used for compound fractures.

The benefit of pre- and perioperative antibiotics has been extensively analyzed and verified to lower postoperative infection rates. Numerous literature studies have demonstrated the advantages of antibiotics in the management of compound mandibular fractures; despite the number of new antibiotics, however, penicillin remains the agent of choice. However, much debate remains in regard to assessing the efficacy and duration of a postoperative antibiotic regimen.

8. Nutritional needs should be closely monitored postoperatively.

Excellent reduction and fixation techniques may fail in a patient who has undergone notable weight loss and has a catabolic nutritional status.

9. Mandibular fractures can be treated by closed reduction.

With enthusiasm for open reduction and rigid fixation in the treatment of mandibular fractures, it is important to remember that closed reduction techniques have a long history of success. Although open techniques have advantages, such as more exacting bone fragment reapproximation and earlier return to function by the patient, significant disadvantages exist as well. These may subject the patient to prolonged anesthesia, increase the risk of infection and metal rejection, cause damage to adjacent teeth and nerves, result in intraoral or extraoral scarring, and increase hospitalization time and cost. The following sections present relative indications for open or closed techniques.

Treatment of mandibular fractures

Hundreds of techniques are available for treating mandibular fractures, as noted earlier in the section on history. However, state of the art treatment methods are currently limited and well defined. It is important to realize that establishment of occlusion is imperative for the successful treatment of mandibular fractures. There are Closed Reduction and Open Reduction with following fication of fractured Mandible.

Indications for closed reduction

Nondisplaced Favorable Fractures. The simplest means possible should be used to reduce and fixate mandibular fractures. For the reasons specified earlier, open reduction can carry an increased risk of morbidity, so closed techniques should be used for treatment. If possible.

Grossly Comminuted Fractures. Because of the excellent blood supply to the face, small fragments of bones will coalesce and heal if the associated periosteum is not disturbed. Comminuted fractures should be managed as a so-called bag of bones, with the clinician using closed techniques to establish normal occlusion without violating integrity.

Fractures Exposed by Significant Loss of Overlying Soft Tissue. Fracture repair is somewhat dependent on soft tissue coverage and vascular supply. Soft tissue coverage should be established by rotational flaps, microvascular grafts or, if the area is small, secondary granulation. Wires, screws, and plates may decrease the chance of successful bone union by disrupting the covering soft tissue further.

Edentulous Mandibular Fractures. These fractures present a special challenge because the inferior alveolar vascular supply to the bone is severely compromised, there is little cancellous bone (with associated osteoblastic endosteum) for repair, and the fractures usually occur in older adults, in whom the normal healing potential can be retarded. Open reduction requires stripping of the covering periosteum, which further inhibits osteogenesis. Closed reduction with the use of a mandibular prosthesis held in place by circummandibular wires offers a more conservative approach if amendable to the clinical presentation. If open reduction is necessary, a supplemental bone graft across the fracture site should be considered in addition to minimal periosteal stripping. In severely atrophic edentulous ridges, open reduction with primary bone grafting may be indicated, because proper alignment of the fractured ends of bone may be impossible because of the small cross-sectional diameter of the mandibular body.

Mandibular Fractures in Children With Developing Dentition. Open reduction with wires or plates carries the risk of damage to developing tooth buds, which occupy a major portion of the mandible in children. If open reduction is necessary because of gross displacement of the fragments, fine wires should be placed at the most inferior border of the mandible, engaging only the cortex. Closed reduction is indicated with special wiring techniques (continuous loop or Risdon wire) or fabricated acrylic splints maintained by circummandibular wiring. A special concern in children is fractures of the mandibular condyle. Damage to the condylar growth center can result in retarded growth of the mandible and facial asymmetry. Intracapsular condylar fractures in children can also lead to ankylosis of the joint, so early mobilization (after 7 to 10 days of intermaxillary fixation) is indicated.

Coronoid Process Fractures. Fractures of the coronoid process are rarely isolated and are usually simple and linear, with little displacement, although with extreme trauma the bone may be displaced into the temporal fossa. Isolated fractures of the coronoid process cause trismus and swelling in the region of the zygomatic arch. There may be swelling in the retromolar area and a lateral crossbite. Treatment is usually initiated only if the occlusion is compromised or if the fractured coronoid process impinges on the zygomatic arch, inhibiting mandibular movement.

Condylar Fractures. Most condylar fractures can and should be treated via closed techniques if the occlusion is not compromised. Early jaw mobilization and physical therapy are indicated to prevent ankylosis or limited jaw movements.

Indications for open reduction

The indications for open reduction as outlined in the following examples are frequently based on the anatomy of the fracture rather than the severity of the injury or the amount of displacement. Control of the edentulous or proximal segment is the overriding factor in selecting this surgical option. Additional factors such as the necessity of early mobilization or medical considerations such as seizure disorders may dictate open reduction treatment.

Displaced Unfavorable Fractures Through the Angle of the Mandible. Open reduction is indicated for this fracture when the proximal fragment is displaced superiorly or medially and reduction cannot be maintained without intraosseous wires, screws, or plating.

Displaced Unfavorable Fractures of the Body or Parasymphyseal Region of the Mandible. The mylohyoid, digastric, geniohyoid, and genioglossus muscles may further displace the fragments. When treated with closed reduction, parasymphyseal fractures tend to open at the inferior border, with the superior aspects of the mandibular segments rotating medially at the point of fixation. With medial rotation of the body of the mandible, the lingual cusps of all premolars and molars move out of occlusal contact. If the constriction is not corrected, masticatory inefficiency and abnormal periodontal changes occur.

Multiple Fractures of the Facial Bones. In multiple fractures of the facial bones, open fixation of the mandibular segments provides a stable base for restoration.

Midface Fractures and Displaced Bilateral Condylar Fractures. With midface fractures and displaced bilateral condylar fractures, one of the condylar fractures should be opened to establish the vertical dimension of the face. If this procedure is not done, any type of suspension wiring, such as that from the frontozygomatic suture area to the mandible, would tend to collapse and telescope the fractures of the midface and condyles, resulting in a foreshortened facial appearance.

Fractures of an Edentulous Mandible With Severe Displacement of the Fracture Fragments. In fractures of an edentulous mandible with severe displacement of the fracture fragments, open reduction should be considered to reestablish continuity of the mandible. The technique is especially helpful with a nonatrophic mandible when there are no dentures, so the occlusion is not an immediate concern. In this situation, plating of the mandible without IMF should be a strong possibility. As the mandible becomes extremely atrophic, consideration must be given to the status of blood supply to the bone and the effect of an open surgical procedure on the compromised vascularity. Supplemental bone grafts have to be considered for extremely atrophic mandibular fractures.

Edentulous Maxilla Opposing a Mandibular Fracture. When a maxilla opposing a mandibular fracture is edentulous or contains insufficient teeth to allow intermaxillary fixation, open reduction should be considered. Open reduction with rigid fixation of the mandibular fractures would eliminate the need for IMF. However, if the patient's condition warrants closed reduction, a prosthesis could be constructed for the maxilla, it could be stabilized with palatal screws or circumzygomatic wires, and routine IMF could be used to treat the fractured mandible.

Delay of Treatment and Interposition of Soft Tissue Between Noncontacting Displaced Fracture Fragments. When treatment has been delayed and soft tissue becomes interposed between noncontacting displaced fracture fragments, open reduction should be used. There are cases in which the treatment of mandibular fractures is delayed because of head injury or other serious medical problems so, with time, connective tissue grows between the bone fragments, inhibiting osteogenesis. When treatment is finally initiated, scar tissue must be removed and treatment completed via an open approach.

Malunion. When a poor result is obtained after mandibular fracture treatment, various types of osteotomies will have to be done via open surgical approaches to correct the deficiencies.

Special Systemic Conditions Contraindicating Intermaxillary Fixation. There are situations in which mandibular functional movement is necessary, and open rigid fixation techniques can provide that option. For example, patients with difficult to control seizures, psychiatric or neurologic problems, compromised pulmonary functions, and eating or gastrointestinal disorders could benefit from open rigid fixation techniques.

Closed reduction and fixation procedures

Bridle Wire. Hippocrates was the first to advocate the use of wires for the reduction of mandibular fractures. His technique of placing a single wire around the teeth adjacent to the fracture still has its place in modern oral surgery. A simple bridle wire placed around the adjacent teeth of a mandibular fracture can temporarily stabilize a flailed mandibular segment. This in itself helps prevent further soft tissue damage, aids in protecting the airway, helps alleviate pain from the two segments moving against each other, and assists in preventing the muscle cramping that is associated with unstable segments.

Equipment Needed

- Local anesthetic
- Needle driver or needle holder
- 24- or 26-gauge stainless steel wire

Procedure. After adequate local anesthetic has been administered, the two segments are manually reduced.

The wire is passed around the necks of the teeth and the fracture loosely approximated. While manually stabilizing the fracture, the operator achieves further reduction by tightening the wire in a clockwise fashion. In the event that the adjacent teeth are loose, decayed, or avulsed, the operator can use the nearest stable teeth.

Ivy Loops. Ivy loops are a quick and easy way of obtaining maxillomandibular fixation. The loop is constructed of 24-gauge wire and passed interproximally to two stable teeth. The ends of the wire are first brought around to the mesial and distal sides of the teeth. The distal wire is then delivered under the loop and tightened to the mesial wire in an apical direction. The loop is then tightened to adapt it into the interproximal space.

Maxillomandibular fixation between Ivy loops can then be achieved by various methods. A smaller gauge wire can be passed through the loops and tightened. To obtain adequate fixation with this method, the loops should be short enough that they do not overlap and create an unstable fixation. Another method involves passing a smaller gauge wire around the lugs created by the loops

Arch Bars. There are a variety of arch bars available for achieving maxillomandibular fixation. Placement of arch bars can be a difficult task, depending on the dentition present and their stability in the traumatized mandible. Here we discuss placement of arch bars on a full intact dentition for reasons of simplicity and instruction. Later, we shall discuss the placement of arch bars on some different combinations of tooth loss (edentia).

Equipment Needed

- Local anesthetic
- Arch bars

• 24- and 26-gauge wire

• Needle drivers

Procedure. The first step in placement is measurement of the arch bar. The bar is usually placed two teeth proximal from the fracture. The bar is traditionally placed from a point distal to the first molar to a point distal to the first molar on the opposite side.

Wire is the next consideration; 24-gauge wire is recommended for the circumdental wires, and 26-gauge wire is used for the box wires that provide the maxillomandibular fixation. The first circumdental wires placed are usually on the second premolars. The measured arch bar is then placed in the loops of the wires and the wires are loosely secured. Wiring then takes place from midline to posterior to avoid excess arch bar in the anterior of the arch.

After placement of the circumdental wires and gross reduction of the fractured segments, they are tightened in the same fashion – from midline to posterior. Some have suggested that the mobile fracture segment should be tightened last, after maxillomandibular fixation. Adaptation of the arch bars to the interdental spaces helps maximize tooth to arch bar contact and helps prevent loosening of the arch bar. The box wires are placed and occlusion is obtained. The circumdental wires are tightened and the rosettes are formed. Box wires are then fully tightened and maxillomandibular fixation is achieved. The goal is to have proper occlusion before complete tightening of interdental wires. The Erich arch bar and, occasionally, eyelet (Ivy loop) and continuous loop (Stout) wiring are used for intermaxillary fixation. Other modalities more rarely used are the Risdon wire and intermaxillary fixation screws.

Risdon Wire. Use of a Risdon wire can be advantageous when treating primary and early mixed dentition. Often, because of the anatomy of the teeth at this stage of development, the design of conventional arch bars does not allow for secure maxillomandibular fixation. There are two separate methods for using the Risdon wire, one continuous cable used on the entire arch or two separate cables joined at the midline of the arch.

A 24- or 26-gauge wire is passed around the distalmost strong tooth, leaving both ends on the buccal side. The wire is then twisted to create a cable, which is fastened in the same manner to the contralateral distalmost strong tooth. Alternatively, a separate 24- or 26-gauge wire can be twisted around both distal strong teeth and the two separately twisted strands can then be crossed at the midline and twisted around each other. Then, as noted earlier, a circumdental wire is passed above and below the twisted cable to ligate each tooth independently. A small rosette is formed and elastics are used to create maxillomandibular fixation

Intermaxillary Fixation Screws. The application of arch bars and insertion of wires in the interdental spaces increase the chance of accidental skin puncture, hence increasing the chance of transmission of HIV and viral hepatitis. The advantages of these intermaxillary fixation screws are as follows:

1. Ease of application
2. Decreased operating time hence diminished overall cost.
3. Decreased risk of disease of transmission.
4. No impingement of cervical gingiva

Equipment Needed

- Local anesthetic
- 24-gauge wire • IMF screws
- Screwdrivers
- Needle drivers

Procedure. This procedure can be performed under local anesthetic or, if the patient cannot tolerate it, under IV sedation or general anesthetic. The mucosa just medial to the canine region is injected with local anesthetic in the upper and lower dentitions. Next, the selftapping IMF screw, 8 to 12 mm in length, is inserted in the bone in a transmucosal fashion. Alternatively, the mucosa overlying the site can be incised with a no. 15 blade and pilot holes can be drilled to facilitate insertion. Close attention should be paid to the location of the mental nerves and root apices to avoid inadvertent iatrogenic injury. Next, IMF is obtained using 24-gauge wires. The disadvantages of IMF screws are the lack of tension band effect and interference with internal fixation plates.

Therefore, we recommend their use only in minimally displaced fractures. The screws can be removed under local anesthetic in the office. Usually, the mucosa around the screw will grow to cover the head of the screws, necessitating an incision and a minimal amount of dissection to retrieve them.

Although this technique can be used for dentulous or edentulous patients, it is most valuable in the following situations: (1) in edentulous cases, to maintain space where bone is missing because of severe trauma or resection; (2) in severely comminuted fractures; and (3) when intermaxillary or rigid fixation edentulous patients with fractures is presented in

Open reduction and fixation procedures

Just as there are a multitude of techniques for closed reduction and fixation, there are also many methods of open reduction and types of orthopedic hardware available for establishing bone approximation. As noted, open reduction should be used in specific situations because of the morbidity attendant with open procedures.

Surgical approaches

Before operating on a mandibular fracture, the surgeon should include the angle of the mouth in the operating field to monitor facial nerve activity and ensure that the anesthesiologist has not paralyzed the patient for an extended period of time. Factors used to establish the location of incision include fracture location, skin lines, and nerve position.

Submandibular Approach. The skin incision is 4 to 5 cm in length, 2 cm below the angle of the mandible. Optimally, the skin incision should be positioned in an existing skin crease to hide the scar and should be made at right angles to the skin surface .The subcutaneous fat and superficial fascia are dissected to reach the platysma muscle. The platysma is sharply dissected to reach the superficial layer of the deep cervical fascia. The marginal mandibular branch of the facial nerve lies just deep to this layer, so it is important to know its course. The dissection to bone is carried through the deep cervical fascia by the surgeon, carefully using a nerve stimulator. The dissection is continued beneath the fascia to the inferior border of the mandible. The submandibular gland and its capsule will become evident, and the lower pole of the parotid may be encountered. The dissection is carried to the masseter muscle, with the surgeon taking care to retract the nerve fibers superiorly. Once the muscle is encountered, it is sharply divided at the inferior border to expose the bone. The muscle, periosteum, and soft tissue are retracted superiorly to expose the body, ramus, and fracture site. If facial vessels cannot be retracted successfully, they may be divided and ligated.

Typically, the submandibular lymph node can be identified adjacent to the facial vessels. Exposure can be increased and closure enhanced by dissecting the medial pterygoid and stylomandibular ligaments from the inferior and posterior borders. Further exposure can be obtained by distracting the angle and inferior border with a wire or bone forceps. The submandibular gland and its capsule are usually located just inferior to the inferior border of the mandible. The parotid gland is generally posterior to the ramus but may wrap around the inferior angle. The capsules of both should be avoided during dissection. Disruption of gland parenchyma may lead to sialoceles or salivary fistulas.

Retromandibular Approach. This approach was basically a variation of the submandibular approach except the incision was approximately 3 cm above the submandibular incision.

The incision is also described by curving behind the angle of the mandible. The incision is made to encounter the parotid, masseteric, and deep cervical fascia The dissection is then extended anteriorly through the deep cervical fascia with the surgeon using nerve stimulation. The incision to bone through the masseter muscle is usually between the marginal mandibular and buccal branches of the facial nerve. The muscle and periosteum are incised over the angle instead of the inferior border. The soft tissue and nerve fibers are then retracted superiorly. This incision gives superior access to the ramus and subcondylar region of the mandible.

Preauricular Approach. This allows for exposure of the TMJ and is easily extended to allow access to temporal anatomy. The incision commonly encounters the superficial temporal vessels, which cartilage.

The incision is made sharply in the preauricular folds, approximately 2.5 to 3.5 cm in length, as described by The incision extends at a 45-degree angle to the zygoma, from the superior aspect of the ear to the inferior attachment of the ear and cheek. Care should be taken not to extend the incision inferiorly because it might encounter the facial nerve as it enters the posterior border of the parotid gland. The incision and dissection should extend just superficial to the perichondrium.

If this plane is followed, it is unlikely that the temporal vessels will be encountered and retracted anteriorly. The dissection is carried anteriorly until the temporal fascia is encountered at the superior portion of the incision. Care should be taken not to perforate into the internal auditory canal as it courses anteromedially. The superficial temporal fascia is continuous with the subcutaneous musculoaponeurotic system above the zygomatic arch and extends superiorly to the galea.

The undersurface of this layer is where the superficial temporal vessels, auriculotemporal nerve, and facial nerve are found. All these layers should be retracted anteriorly with the soft tissue flap. The facial nerve has been described as crossing the zygomatic arch 0.8 to 3.5 cm (mean, 2 cm) anterior to the concavity of the auditory canal. A total of 50% have been found to cross at a point less than 2 cm. The distance from the auditory concavity and lateral articular tubercle is also 2 cm (1.6 to 2.5 cm) and should be the anteriormost point of dissection needed for access.

The temporal fascia divides 2 cm above the zygomatic arch and is an extension of the parotidomasseteric fascia. The lateral layer is contiguous with the lateral periosteum and the deep layer is contiguous with the medial periosteum. Between these two layers is fat, temporal vessels, and the zygomaticotemporal branch of the maxillary nerve. A sharp incision is made through the periosteum 2 to 3 cm anterior to the skin incision. The periosteum, vessels, and nerves are then retracted anteriorly.

The temporal and zygomatic branches of the facial nerve lie within the lateral periosteum-fascia layer. This dissection is carried anteriorly to the lateral eminence, exposing the joint capsule. Fracture location dictates whether the capsule is opened.

Endaural Approach. The endaural incision is started in the skin crease between the anterior helical cartilage and tissue, extended downward in the cleft between the tragus and helix and inward approximately 5 mm along the roof of the auditory canal. As the incision deepens, it is carried anteriorly through the tragal cartilage. After the tragal cartilage is released, the incision is deepened along the auricular cartilage, similar to the preauricular approach described earlier.

Intraoral Access

Symphysis and Parasymphysis. Anterior mandibular fractures may be accessed via intraoral incisions. First, the incision region is infiltrated with local anesthetic and vasoconstrictors. The lip is retracted and a curvilinear incision is made perpendicular to the mucosal surface. It is important to carry the incision out into the lip, leaving at least 1 cm of mucosa attached to the gingiva. The mentalis muscle, now visible, should be incised perpendicular to bone, leaving a flap of muscle attached to bone for closure. The dissection is carried subperiosteally to identify the mental neurovascular bundle, approximately midway between the alveolar ridge and inferior border, below the second premolar or slightly anterior. The fracture site is identified and reduced. The surgical site is closed in layers. The mentalis muscle is secured to the remaining pedicle with nterrupted sutures while an assistant reduces the wound with finger pressure from below. The mucosa is then closed and an adhesive bandage is applied to the chin to support the mentalis muscle and thus prevent drooping.

Body, Angle, and Ramus. After administration of adequate local anesthetic and vasoconstrictors, the cheek is retracted laterally. The mucosa is incised to the bone with the blade positioned perpendicular to the bone to avoid the mental nerve. The incision is made approximately 5 mm from the mucogingival junction to allow adequate mobile tissue for closure. The proximal portion of the incision should be carried along the external oblique ridge, only as high as the mandibular occlusal plane.

Extending the incision higher predisposes the buccal fat pad to prolapsing onto the surgical field. The anterior surface of the ramus can then be exposed by stripping the buccinator and temporal tendon with a notched angled retractor and periosteal elevator. Once the coronoid is exposed, a curved Kocher clamp can be applied, which will act as a self-retaining retractor. Dissection in this manner, subperiosteally, keeps the buccal fat pad out of the field. The masseter muscle can then be elevated with periosteal elevators and J strippers. The entire ramus and subcondylar region can now be exposed with the placement of Bauer retractors in the sigmoid and antegonial notches. The masseter can also be retracted with the placement of LeVasseur-Merrill retractors.

Wire Osteosynthesis. Historically, open techniques were performed through a skin incision or laceration, and wires were generally used to maintain the fracture fragments. With the advent of modern orthognathic surgery, intraoral open surgical approaches have become the standard and, although wire osteosynthesis is still widely used in the United States, techniques developed in Europe that use bone plates and screws for rigid fixation have become widely accepted here. Wires may be simpler to place and usually will maintain the bone fragments and prevent bone displacement by muscle pull until healing occurs.

However, wires lack rigidity, directional control, and surface to bone surface contact area to maintain rigidity under function, so IMF must be used. On the other hand, with screw or plate rigid fixation, IMF is usually unnecessary.

Wire osteosynthesis is most commonly used for angle fractures at the superior border of the mandible; the wire is placed via an intraoral approach. Concomitant removal of an impacted third molar allows excellent access and easy placement of the wire. Parasymphyseal or midsymphyseal fractures are also often reduced and fixed, with wire osteosynthesis placed via an intraoral approach. The wire is positioned beneath the teeth, inferior alveolar canal, and mental foramen. As noted, fractures in this area are often oblique, causing telescoping. In this case, the wire must be placed through both segments of bone, which may be millimeters away from the buccal fracture line. However, if the fracture is perpendicular to the buccal surface of the mandible, wire should be placed in a figure-eight fashion to cradle the lower border and to bring the two edges of the fracture together.

Endoscopic repair. Medical uses of endoscopy date back to the era of Hippocrates. With the advent of the flexible endoscope and fiberoptics, endoscopic techniques have revolutionized a number of approaches in the surgical field. The endoscopic approach to the treatment of abdominal and gynecologic disorders has been well established. Initial applications in craniomaxillofacial surgery were for aesthetic procedures, such as the brow-lifting procedure and forehead recontouring. Uses were expanded within the field to treat orbital wall fractures, distraction osteogenesis, zygoma fractures, frontal sinus fractures, cranial synostosis, and subcondylar fractures. By using the endoscopic technique, the risk of damage to the facial nerve and visible scarring is minimized. For our purposes here, the endoscopic technique is mostly used for the repair of subcondylar fractures (discussed elsewhere in this text), but it can also be used for the repair of high ramus fractures. The surgical approach is via a transoral sagittal split like incision or an extraoral submandibular incision.

TOPIC: MAXILLARY FRACTURES

The maxilla represents the bridge between the cranial base superiorly and the dental occlusal plane inferiorly. Its intimate association with the oral cavity, nasal cavity, and orbits and the multitude of structures contained within and adjacent to it make the maxilla a functionally and cosmetically important structure. Fracture of these bones is potentially life-threatening as well as disfiguring. Timely and systematic repair of these fractures provides the best chance to correct deformity and prevent unfavorable sequelae.

Maxillary fractures account for approximately 6-25% of all facial fractures.

Etiology

Maxillary fractures often result from high-energy blunt force injury to the facial skeleton. Typical mechanisms of trauma include motor vehicle accidents, altercations, and falls. With increased legislation requiring seat belt use, injuries from driver impact with the steering wheel have shifted from chest trauma to facial trauma.

Pathophysiology and classification

Much of the understanding of patterns of fracture propagation in midface trauma originates from the work of René Le Fort. In 1901, he reported his work on cadaver skulls that were subjected to blunt forces of various magnitudes and directions. He concluded that predictable patterns of fractures follow certain types of injuries. Three predominant types were described.

Le Fort I fractures (horizontal) may result from a force of injury directed low on the maxillary alveolar rim in a downward direction. The fracture extends from the nasal septum to the lateral pyriform rims, travels horizontally above the teeth apices, crosses below the zygomaticomaxillary junction, and traverses the pterygomaxillary junction to interrupt the pterygoid plates.

Le Fort II fractures (pyramidal) may result from a blow to the lower or mid maxilla. Such a fracture has a pyramidal shape and extends from the nasal bridge at or below the nasofrontal suture through the frontal processes of the maxilla, inferolaterally through the lacrimal bones and inferior orbital floor and rim through or near the inferior orbital foramen, and inferiorly through the anterior wall of the maxillary sinus; it then travels under the zygoma, across the pterygomaxillary fissure, and through the pterygoid plates.

Le Fort III fractures (transverse), also termed craniofacial dysjunctions, may follow impact to the nasal bridge or upper maxilla. These fractures start at the nasofrontal and frontomaxillary sutures and extend posteriorly along the medial wall of the orbit through the nasolacrimal groove and ethmoid bones. The thicker sphenoid bone posteriorly usually prevents continuation of the fracture into the optic canal. Instead, the fracture continues along the floor of the orbit along the inferior orbital fissure and continues superolaterally through the lateral orbital wall, through the zygomaticofrontal junction and the zygomatic arch. Intranasally, a branch of the fracture extends through the base of the perpendicular plate of the ethmoid, through the vomer, and through the interface of the pterygoid plates to the base of the sphenoid.

In reality, the Le Fort classification is an oversimplification of maxillary fractures. The amount of force impacted during a motor vehicle accident is much greater than Le Fort took into consideration during his work in the late 19th century. In most instances, maxillary fractures are a combination of the various Le Fort types. Fracture lines often diverge from the described pathways and may result in mixed-type fractures, unilateral fractures, or other atypical fractures. In addition, in very high-energy blows, maxillary fractures may be associated with fractures to the mandible, cranium, or both.

Two types of non–Le Fort maxillary fractures of note are relatively common. First, limited and very focused blunt trauma may result in small, isolated fracture segments. Often, a hammer or other instrument is the causative weapon. In particular, the alveolar ridge, maxillary sinus anterior wall, and nasomaxillary junction, by virtue of their accessibility, are common sites of such injury. Second, submental forces directed superiorly may result in several discrete vertical fractures through various horizontal bony supports such as the alveolar ridge, infraorbital rim, and zygomatic arches.

Unlike with fractures in other bones, muscle forces do not play a significant role in the final position of the broken bony segments. While several muscles attach to the maxillary framework, they typically insert to skin and therefore do not lead to additional deformity. Instead, the basis for the patterns of maxillary fractures depends on 2 predominant factors.

First, as Le Fort described, the location, direction, and energy of the impact result in different injuries. Second, the anatomy of the mid face is oriented to provide strength and support to protect against injury. Vertical and horizontal bony bolstering in the face absorbs the energy of traumatic force. This serves to protect the more vital intracranial contents from damage during trauma. Knowledge of the characteristics of the traumatic blow combined with an understanding of the anatomic bolstering in the face can help the clinician approach such injury in a logical and systematic fashion.

Relevant Anatomy. The maxillary bones are paired pyramidal bones that in many ways serve as the cornerstones of the facial skeleton. In a superoinferior direction, the maxilla bridges the cranial-frontoethmoid complex above to the mandible and to the occlusal plate below. In a transverse plane, it bridges the 2 zygomaticoorbital complexes.

Each individual maxilla can be conceptualized as a 5-sided structure, the base of which makes up the lateral nasal wall. The remaining 4 sides of the pyramid are composed of the orbital floor superiorly, the alveolar ridge inferiorly, the front wall of the maxillary sinus anteriorly, and the anterior face of the pterygopalatine fossa posterolaterally.

The maxilla and the associated bones of the mid face are oriented to resist the vertical forces of mastication. This is accomplished through 3 paired vertical buttresses (from anteromedial to posterolateral): the nasomaxillary buttress, the zygomaticomaxillary buttress, and the pterygomaxillary buttress. An additional unpaired midline support is the frontoethmoid-vomerine buttress. These pillars serve to diffuse the vertical forces of mastication over the broad cranial base. They are also effective shock absorbers for a vertically oriented impact to the facial skeleton.

The nasomaxillary buttress transmits force from the maxillary canine area through the lateral pyriform rim and frontal process of the maxilla and to the superior orbital rim. The zygomaticomaxillary buttress transmits forces from the zygomatico-alveolar crest through the zygoma to the posterior aspect of the superior orbital rim and temporal bone. The pterygomaxillary buttress conducts force through the palatine bone to the pterygoid plates and sphenoid base.

The superior and inferior orbital rims and alveolar ridge constitute a group of weaker horizontal buttresses. While these structures provide some protection against horizontal forces, they can withstand much less force than the vertical buttresses. Therefore, vertical impact tends to be better absorbed within the facial skeleton, which resists fracture, while horizontal impact tends to overcome the weaker horizontal buttresses and shear through the vertical pillars. In a surgical approach to maxillary fractures, attempts should be made to restore the continuity of these support buttresses.

Several important nonosseous structures are closely associated with the maxilla and thus may be damaged during injury or repair.

The infraorbital nerve travels through the inferior orbital fissure and exits from the infraorbital foramen just below the inferior rim. Therefore, anesthesia of the cheek and upper teeth is commonplace in persons with extensive fractures.

Branches of the internal maxillary artery provide much of the vascular supply to the mid face. In 2009, Bozkurt et al reported a case of pseudoaneurysm of this artery after a subcondylar fracture. In patients with extensive epistaxis or bleeding, ligation of this artery may be necessary. Redundant anastomoses from other branches of the internal and external carotid circulation ensure that vascular insufficiency of the maxilla is unlikely, even with internal maxillary artery ligation.

Orbital contents such as the lacrimal system, extraocular muscles, optic nerve, and globe are in close proximity to the maxilla and are therefore susceptible to injury.

Physical examination

Evaluation of the maxilla and facial bones should be undertaken only after the patient has been fully stabilized and life-threatening injuries have been addressed. In particular, airway considerations and intracranial injuries must take immediate priority.

In general, patients with facial fractures have obscuration of their bony architecture with soft tissue swelling, ecchymoses, gross blood, and hematoma. Nonetheless, observation alone may be informative. Focal areas of swelling or hematoma may overlie an isolated fracture. Periorbital swelling may indicate Le Fort II or III fractures. A global posterior retrusion of the mid face creates a flattened appearance of the face. The so-called dish-face or pan-face deformity may occur after an extensive Le Fort II or Le Fort III fracture. The maxillary segment is displaced posteriorly and inferiorly. This may cause premature contact of the molar teeth, resulting in an anterior open bite deformity. In severe cases, the upper airway may be compromised. In such a situation, disimpaction forceps may need to be placed into the nasal floor and hard palate to pull the bony segment forward to restore airway patency.

The face and cranium should be palpated to detect for bony irregularities, step-offs, crepitus, and sensory disturbances. Mobility of the mid face may be tested by grasping the anterior alveolar arch and pulling forward while stabilizing the patient with the other hand. The size and location of the mobile segment may identify which type of Le Fort fracture is present. If only an isolated segment of bone is mobile, a small alveolar or nasofrontal process chip fracture may be present. With high-impact force, the maxilla may be comminuted or impacted, in which case the bony framework is displaced or crushed but immobile.

A thorough nasal and intraoral examination should be completed. The nasal bones are typically quite mobile in Le Fort II fractures, along with the rest of the pyramidal free-floating segment. Intranasal examination may reveal fresh or old blood, septal hematoma, or cerebrospinal fluid rhinorrhea. The intraoral examination should assess occlusion, overall dentition, stability of the alveolar ridge and palate, and soft tissue. Finger palpation of the maxillary contour intraorally may provide additional information about the integrity of the nasomaxillary buttress, anterior maxillary sinus wall, and zygomaticomaxillary buttress.

During examination of the eyes and orbit, search for integrity of the orbital rims, orbital floor, vision, extraocular motion, position of the globe, and intercanthal distance. Unlike Le Fort II fractures, Le Fort III fractures are associated with lateral rim and zygomatic breaks. Visual changes may signify a disturbance of the optic canal, problems within the globe or retina, or other neurologic lesions. Disturbances of extraocular motion or enophthalmos may signify a blowout in the orbital floor. An increased intercanthal distance implies displacement of the frontomaxillary or lacrimal bones or avulsion of the medial canthal ligament. For extensive involvement of the orbit or globe, consultation with an ophthalmologist is appropriate.

Tools that may assist the examiner in the evaluation of maxillofacial trauma include a headlamp or mirror, tongue blades, a suction device, a nasal speculum, an otoscope, and a ruler. Early photographs may be helpful in preoperative planning and patient counseling.

Imaging Studies

Plain radiographs

– Plain films are limited by their ability to penetrate through extensive soft tissue edema and to help distinguish between multiple planes of complex bony framework.

– Historically, plain radiographs with Waters and submental-vertical views of the paranasal sinuses, lateral skull, and lateral cervical spine were used as screening examinations.

– Sinus films provide information regarding the zygomatic arches, nasal bones, lateral and anterior sinus walls, and orbital rims. Most of the anatomic bolsters described above should be visualized.

– Lateral skull films provide information about the global anteroposterior position of the mid face and the integrity of the inner and outer tables of the frontal sinus.

– Cervical spine films are important to exclude injury to the vertebral column. Otherwise, standard radiographs are little used for craniofacial injuries.

– For more information, see eMedicine Radiology article Le Fort Fractures.

CT scans

– CT scan images are the imaging modality of choice for facial fractures.

– CT imaging is superior to plain films for delineating multiple fractures, evaluating associated cartilaginous or soft tissue injury, and assessing for the presence of impingement into the optic canal.

– Thin (2-mm) cuts in both the coronal and axial planes are needed to obtain adequate detail of fractures.

Three-dimensional CT scans are highly recommended for the treatment planning of fractures of moderate or greater complexity.

Treatment

Medical Therapy

Stabilize the patient and treat serious insults to the airway, neurologic system, cervical spine, chest, and abdomen prior to definitive treatment of the maxillofacial bones.

Address emergencies related to maxillofacial trauma prior to definitive treatment. These include airway compromise and excessive bleeding. If the airway is compromised and orotracheal intubation cannot be established, the midface complex may be impacted posteroinferiorly, causing obstruction. Disimpaction may be attempted manually or with large disimpaction forceps around the alveolar arch and premaxilla. If the segments do not move readily and the airway is obstructed, an emergent tracheotomy or cricothyrotomy may be necessary. Severe bleeding may occur from soft tissue lacerations or intranasal structures. A combination of pressure, packing, cauterization, and suturing may be useful in such situations.

Surgical Therapy

Fixation of unstable fracture segments to stable structures is the objective of definitive surgical treatment of maxillary fractures. This principle, while seemingly simple, becomes more complex in patients with extensive or panfacial fractures.6 In isolated maxillary fractures, the stable cranium above and occlusal plate below provide sources of stable fixation. One goal of treatment is to restore proper anatomic relationships. In particular, attempt to normalize the integrity of the support bolsters of the facial skeleton, the midfacial height and projection, and dental occlusion and masticatory function.

Preoperative Details

After all other more critical medical problems have been stabilized, the patient may be considered for repair of maxillofacial injuries. Have adequate plain film and CT imaging available in the operating room for intraoperative guidance. A complete maxillofacial plating set must be available.

Prior to surgery, inform the patient of the implications of the anticipated procedures. Counsel the patient regarding the limitations and duration of maxillomandibular fixation (MMF). Additionally, the patient must understand the risks and possible complications of the procedure, including temporary or permanent paresthesia, cerebrospinal fluid leak, meningitis, sinus infection or mucocele, anosmia, malocclusion, infection of implants, osteomyelitis, malunion or nonunion, external deformity, plate exposure, tooth injury, and the possible need for additional surgery.

Intraoperative Details

Perform repair of any significant maxillary fracture requiring reduction and fixation in the operating room with the patient under general anesthesia. Because of the need for MMF, intubate the patient with a nasotracheal tube.

In general, attempt to complete restoration of dental occlusion with MMF prior to reduction and fixation of other segments of the maxilla. MMF accurately restores the position of the base of the maxilla, allowing for correct reconstruction from inferior to superior. If the mandible is also fractured, reduction and fixation of the mandible must be completed first, followed by MMF, and then definitive repair of maxillary fractures. Disimpaction of the free maxillary segments can be performed manually or with disimpaction forceps. Perform this procedure carefully because injury to the nasolacrimal duct, inferior orbital nerve, and extraocular muscles may be involved in middle and high maxillary fractures.

In patients with Le Fort III fractures, exclude the presence of bony segments in the optic canal prior to aggressive attempts at disimpaction. MMF is typically performed with arch bars and stainless steel 25- or 26-gauge interdental wires. For edentulous patients, surgical splints or dentures secured to the underlying bone with screws or with circummandibular and circumzygomatic wiring may serve as the basis of stabilization.

Once the proper occlusal plane is restored, definitive reduction and fixation of the maxillary fractures may be undertaken. Suspension and intraosseous wires have largely been abandoned more because of suboptimal immobilization than other reasons. Both miniplates and external fixation have been applied successfully to the treatment of midface fractures.

Le Fort I fractures surgical treatment

For stable, nondisplaced Le Fort I fractures, MMF alone may suffice to provide stable restoration of bony support. Partial or segmental alveolar ridge fractures can likewise be treated with MMF alone after proper reduction. However, unstable fractures require an additional means of fixation. Some surgeons prefer to place additional fixation even to nondisplaced fractures, with the goal of allowing earlier removal of MMF and return to mastication.

The method of choice for fixation is through miniplates placed via an open approach. Make gingivolabial incisions through mucosa 5-10 mm labial to the apex of the sulcus to preserve a cuff of untethered mucosa for closure. Carry the incision down to alveolar bone from one molar region to the other. Elevate the periosteum superiorly to expose the fracture lines. Take care to not injure the infraorbital neurovascular bundle. Expose the nasomaxillary and zygomaticomaxillary buttresses, piriform aperture, and premaxilla and nasal spine. Then, contour vertically oriented miniplates using a malleable template to span the fracture line. For true Le Fort I fractures, one plate across the nasomaxillary or zygomaticomaxillary buttress on each side is usually adequate for stable fixation. The most common method is low-profile titanium plates secured with monocortical self-tapping screws.

An alternative method of fixation uses suspension wiring. In this method, a 25- or 26-gauge wire is looped around the temporal aspect of the zygomatic arch, retrieved intraorally, and tightened to an intermediate wire loop connected to the arch bar.

The wire may be passed using a specialized awl with a hole at the tip. Insert the awl into the skin at the superior aspect of the junction of the body and arch of the zygoma. Pass it medially to the arch, and direct it toward the first molar or second premolar. The awl then pierces the mucosa, and a strand of wire is twisted securely around the hole in the awl tip. Withdraw the awl tip to a point just above the zygomatic arch, and then direct it downward, but lateral, to the arch. Once inferior to the arch, direct the awl toward the original tract to the same mucosal opening created by the first pass. Untwist the wire from the awl, remove the awl, and secure the 2 free ends of wire, tightening them to the arch bar using an intermediate wire loop. This method is useful for edentulous patients, in which case the free ends of wire are secured to an intermediate wire loop that passes through holes drilled through the patient's dentures (which are used as the basis for MMF).

Take care to not overtighten suspension wires because the zygomatic arch resides somewhat posterior to the true vertical plane of the maxilla. Overaggressive pulling may result in superoposterior displacement of the inferior fracture segment.

Le Fort II fractures surgical treatment

Just as for Le Fort I fractures, disimpaction, MMF, and sublabial incisions and exposure of maxillary bone and fracture lines are performed. Additional exposure is often necessary superiorly for adequate exploration of the orbital rim. This may be achieved through subciliary or transconjunctival incisions. More extensive degloving of the soft tissue envelope through exposure of the piriform aperture and frontomaxillary region may be facilitated by columellar-septal transfixion incisions.

In general, the pyramidal free maxillary segment is stabilized to the intact zygoma. Because rigid fixation is a traumatic procedure, do not perform it until reduction is optimized. Fixation may be completed directly using noncompression miniplates that span the break in the region of the zygomaticomaxillary buttresses. If instability persists, additional plates may be placed in the nasomaxillary buttresses or inferior orbital rim. Any plating must be placed in areas of adequately robust bone (ie, buttresses). Accurate contouring of the plates using malleable templates is important for precise reduction and fixation. Monocortical, self-tapping screws are ideal. Place plates so that at least 2 screws holes are on each side of the fracture. Thus, if needed, additional screws can be placed for more support.

An alternative to miniplates is interosseous wiring. In this method, place small holes into the appropriate bony segments on either side of the fracture line with a minidriver. Then, pass 28-gauge steel wire through the hole on one side of the fracture and retrieve it outward from the gap between the bony segments. Pull the free end of the wire through the opposite drill hole with a loop of 30-gauge wire. Tighten the 2 free ends of wire. In general, place wires from stable to unstable segments. Because this method is less stable than miniplating, perform several areas of fixation (eg, nasomaxillary, zygomaticomaxillary, inferior orbital rim buttresses). If this method is used, implement a longer duration of MMF than with plating.

Circumzygomatic suspension wiring of Le Fort II fractures has been described. While this method may be effective for clean, true Le Fort II fractures, it is discouraged for 3 main reasons. First, these injuries often have multiple segments, in which case comminution and compression of the maxilla may follow efforts to pull the maxilla en bloc. Second, reduction depends on a vector force that is imperfect. In most patients, the vector from the classic fracture line of Le Fort II fractures to the zygomatic arch is at least 15° askew from the ideal axis for fracture reduction. Finally, other methods have the advantage of more precise application of fixation forces immediately at the site of fracture, minimizing micromotion, maximizing bone healing, and allowing for earlier return to mastication.

Le Fort III fractures surgical treatment

In repairing Le Fort III fractures, stabilize the mobile segments of bone to the stable mandible below and cranium above. Initially, the maxilla must be disimpacted and MMF implemented. Soft tissue incisions may be made in the same locations as for Le Fort II fractures. Lateral brow incisions, glabellar fold incisions, or bicoronal scalp flaps can be used for additional exposure to the frontozygomatic buttress.

The bicoronal flaps may be extended to achieve access to the zygomatic arches. The bicoronal flap must be designed cautiously to avoid injury to the frontal branch of the facial nerve. The plane of dissection is between the galea and pericranium. Once the soft tissue flap is rolled over the superior orbital rims, the pericranium may be incised just above the rims to preserve the supraorbital and supratrochlear vascular supply to the flap.

Laterally, perform dissection just superficial to the temporalis fascia. In approaching the zygomatic arch, incise the temporalis fascia well above it. Develop a plane deep to the fascia down to the fractured zygomatic arch. The fracture can then be levered into reduction with a rigid elevator. If impacted or comminuted, direct fixation may be required. Do not use the bicoronal flap in situations in which soft tissue flaps based on the superficial temporal arteries are needed. A receding hairline also may prompt the surgeon to use other incisions.

Prior to fixation of the involved maxillary fractures, reduce and stabilize any mandibular and cranial fractures. Once this is performed and the fractured maxillary segments are exposed, fixation may be undertaken.

Miniplate fixation is currently the most reliable and rigid method. Use malleable templates; accurate contouring of plates; and monocortical, self-tapping screws. Use plates that span the involved major buttresses. For true Le Fort III fractures, bilateral zygomaticofrontal fixation may suffice.

However, more commonly, additional points of fixation are needed (eg, nasomaxillary, nasofrontal, inferior orbital rim, zygomatic arch). Use as few plates as possible to achieve fixation; excessive plating is not necessary.

Interosseous wiring and suspension wiring have been described for Le Fort III fractures but are less reliable than miniplate fixation because vectors of forces to maintain reduction are less accurate and micromotion is increased.

Extraskeletal fixation is not usually necessary for simple Le Fort fractures. In patients with more extensive panfacial fractures, external fixation may be the only means of stabilization. If possible, avoid this method because it can place excessive or misdirected force onto the fracture segments and therefore cause shortening or further deformity of the mid face.

For all maxillary fractures, suspension of the soft tissue of the mid face should be performed prior to closing the intraoral incisions with 3-0 chromic suture and closing the skin incisions with absorbable subcutaneous sutures and permanent skin sutures. Bicoronal flaps may be closed with skin staples.

Postoperative Details

To minimize postoperative edema, a light pressure dressing consisting of gauze and a head wrap may be placed over the operated areas. If the dressing remains dry, it may be removed after 2-5 days.

Surgeons' opinions are divided regarding the need for postoperative antibiotics. If the original fracture sites were open to the external environment or in communication with intraoral or intranasal spaces, implement prophylactic antibiotics covering gram-positive and anaerobic organisms for 5-10 days.

After surgery, observe patients overnight for bleeding, airway problems, and vomiting. If wire fixation was used for MMF, place wire cutters near the patient at all times in the early postoperative period to allow the patient to expel vomited material. Remove wires or rubber bands if the patient begins to feel nauseated.

Prior to discharge, instruct patients on how to remove the MMF in case of vomiting. Also, counsel patients regarding limiting their diet to pureed or liquid intake.

Follow-up

Perform a follow-up evaluation at 5-7 days (skin sutures may be removed at this time), 2-4 weeks, and then at 3-8 weeks for removal of the

MMF. Longer-term follow-up care may be needed to monitor postoperative complications or deformity.

The most important goal during the early postoperative period is maintaining a state of immobilization. Depending on the age and general health of the patient, the extensiveness and displacement of the fractures, and the repair technique used, this period may range from 4-8 weeks. This requires that MMF be maintained during this period. During this period, emphasize to the patient to maintain oral hygiene with diligent teeth and arch bar brushing and oral rinses with saline or antiseptic mouthwash each morning and evening and after each meal.

Throughout the postoperative course, the stability of the facial skeleton may be tested by palpating the patient's maxillary teeth during clenching and relaxing of the muscles of mastication. Minimal conducted motion is acceptable, but excessive mobility may indicate poor healing. Postoperative films (ie, mandible series, Panorex dental views, facial series, CT scan) may be helpful in patients in whom malunion is suggested.

Once the facial skeleton is deemed to be well healed and normal occlusion is present, the MMF may be removed. Minimal vertical mobility of the mid face likely resolves with time. Excessive motion indicates that it is too early for the arch bars to be removed or that a problem exists with union. In general, the MMF is removed earlier for fractures repaired with miniplate fixation and later for those repaired with interosseous or suspension wires.

Complications

Soft tissue complications result from technical pitfalls or problems with wound healing. In general, unfavorable scarring may be avoided by closing facial incisions in a 2-layered fashion, with deeper subcutaneous absorbable sutures placed to remove tension from the skin closure. Skin closure should be performed with nontraumatic handling of wound edges and should result in the wound edges being slightly everted.

Intraoral incisions may dehisce partially or completely because of inadequate closure during surgery, poor oral hygiene, local trauma, or excessive motion. When designing the gingivolabial incisions, a cuff of mucosa should be maintained on the gingiva to allow for adequate soft tissue upon which to suture. This may be accomplished by placing the incision slightly labial to the deepest part of the gingivolabial sulcus. If dehiscence occurs, maintaining local hygiene alone allows for eventual healing.

Lower lid ectropion may follow a subciliary approach to the maxilla. This complication may be avoided by performing meticulous dissection between the orbital septum and orbicularis oculi muscle and, for patients in whom laxity is present, superolateral suspension of the muscle to the periosteum of the lateral orbital wall. If severe ectropion occurs, breaking up the scar with Z-plasty or skin grafting from the opposite lid skin may be necessary. Lower lid transconjunctival incisions decrease the likelihood of ectropion and should be considered in high-risk patients.

Nerve injury may have occurred prior to surgery from the initial traumatic insult. Therefore, the status of the main sensory and motor nerves of the face and forehead must be documented prior to surgery. Care should be taken to identify and preserve the supraorbital and infraorbital neurovascular pedicles while the soft tissue flaps are raised. More commonly, supraorbital nerve injury results from nerve stretching in retracting the soft tissue and orbital tissues to gain access to the superior and medial orbital rims. The frontal branch of the facial nerve may be injured from excess traction on the forehead flap.

Anatomic disruption of the nerve may occur if the improper plane is used to access the zygomatic arch. The nerve is known to cross the arch superficially to the superficial layer of the deep temporalis fascia. Therefore, dissection should be performed deep into this layer. The appropriate plane is accessed by incising the temporalis fascia well above the arch and dissecting deeply to fascia down to the fractured arch. Nerve injury is often incomplete and temporary.

Injury to tooth roots from misplaced screw holes may result in nonviable teeth. If fracture lines are low and do not allow an area adequate to avoid teeth when placing plates, suspension or interosseous wire fixation may be considered.

Postoperative infections are more apt to occur in the setting of extensive soft tissue injury, contaminated wounds, open fractures, fractures communicating with intranasal or intraoral spaces, or nonevacuated sinus blood. If empiric antibiotic therapy does not clear the infection, debridement and drainage may be required. Cultures should be obtained if purulent material is encountered, and specific antibiotic treatment should be instituted. Long-term unchecked infection may cause osteomyelitis around the sites of the screws or wires. Removal of these implants and debridement of bone may be necessary if antibiotics are unsuccessful. Sinusitis may occur if fracture lines involve the sinus drainage ostia. In such instances, decongestants and antibiotics should be started; intranasal surgical drainage should be performed for nonresolving cases.

Malunion and resultant malocclusion and deformity occur if reduction is not precise or if loosening of fixation occurs during the postoperative period. This can be avoided with meticulous surgical technique and adequate fixation, preferably with carefully placed miniplates. Patient noncompliance with MMF and early mastication may result in micromotion, which leads to poor bone healing. If malunion is discovered early, attempts to optimize reduction may be made by loosening the MMF tension and adjusting the wire closure forces or elastics in order to normalize occlusion. If this fails, rigid fixation (wires or plates) must be removed and replaced for better stabilization.

For delayed presentations in which the bones have healed into malposition, osteotomies must be performed through or near the original fracture sites and the bones must be repositioned with rigid fixation. In rare instances, bone resorbs as a result of malunion and motion, and osseous interposition grafts or overlay grafts may be required. Split calvarial grafts are well suited for midface work, but rib grafts may be used as an alternative.

Total nonunion is less common than malunion. In most cases, maintaining an extended period of fixation and immobility results in eventual healing. For persistent nonunion, fracture sites must be reexplored, freshened, and refixated. Again, areas of gaps may need to be addressed with osseous grafts.

Outcome and Prognosis

A lack of prospective studies on trauma patients makes assessment of outcome measures for patients treated for maxillary fractures difficult. Repair of simple maxillary fractures typically restores bony aesthetic contour and function; however, complex fractures often leave the patient with some long-term cosmetic and functional deficits. Early and meticulous surgery is most likely to produce results that restore the patient to the pretrauma state.

Future and Controversies

The continuing trend in facial fracture repair is toward rigid osteosynthesis with miniplates and screws. The advantage of this technique is that a higher degree of stability is gained, allowing for earlier removal of MMF and return to mastication.

Opponents of this technique who favor suspension techniques cite the disadvantage that anatomic realignment must be perfect at the time of surgery. Whereas suspension techniques allow postoperative adjustment of segments by changing the MMF to compensate for slight deviations from perfect reduction, rigid techniques are much less forgiving. Unrecognized displacement of midface or mandibular segments results in inevitable malunion. Also cited are the higher cost of the materials, the difficulty in contouring plates to the surface of the bone, and increased surgical time. Despite these disadvantages, rigid techniques are gaining in popularity. As long as surgical technique is proficient, rigid osteosynthesis is generally believed to lead to better long-term results and faster recovery.

Absorbable plating systems composed of polylactic acids have recently become available and are gaining popularity for maxillofacial repair. These systems have the advantage of providing rigid osseous fixation without permanent foreign body implantation. This theoretically reduces the risk of infection and plate exposure. The other main advantage of these systems is the ability to contour plates with thermal manipulation (hot saline sponge or specialized heated instruments) even after the plates have been positioned in situ. This facilitates contouring plates to a precise and appropriate shape across fracture lines.

The use of endoscopically-assisted techniques allows for limited incisions for the reduction of facial fractures. These techniques have been pioneered for use in the reduction of condylar and orbital fractures but have recently been applied to more extensive procedures. The use of endoscopic techniques allows for limited incisions, faster recovery periods, and shorter hospital stays. Despite the advantages afforded with these techniques, the indications for open procedures have not been drastically altered. Facial trauma that involves severely dislocated or comminuted fractures of the facial skeleton and major reconstruction of the facial support structures still requires the use of open techniques and direct visualization.

Reconstruction of the facial skeleton involves the reestablishment of the original contours of the face with the precise alignment of fractures. The advancement of image guidance systems has assisted the surgeon in preoperative evaluation and surgical planning, but its recent introduction into the operative arena allows real-time localization of displaced facial skeletal segments during reduction and internal fixation. The use of this technology can help the surgeon obtain a postoperative result that most closely approximates the pre-trauma skeletal structure. This may be most useful in cases where the adjacent bony anatomic landmarks are also displaced or altered and the continuing incorporation of computer-aided guidance of reduction of facial fractures will help to optimize surgical results.

TOPIC: ZYGOMATIC COMPLEX FRACTURE

The zygoma articulates with the frontal, sphenoid, temporal, and maxillary bones and contributes significantly to the strength and stability of the midface. The forward projection of the zygoma causes it to be injured frequently. The zygoma may be separated from its four articulations. This is called a zygomatic complex fracture. The terms *trimalar* or *tripod fracture* are therefore inaccurate. These terms reflect an inability to easily identify the orbital (zygomaticosphenoid) portion of the injury before the advent of computed tomography (CT). The zygomatic arch may be fractured independently or as part of a zygomatic complex fracture.

Surgical Anatomy

The zygoma has four projections, which create a quadrangular shape: the frontal, temporal, maxillary, and the infraorbital rim. The zygoma articulates with four bones: the frontal, temporal, maxilla, and sphenoid. A zygomatic complex fracture includes disruption of the four articulating sutures: zygomaticofrontal, zygomaticotemporal, zygomaticomaxillary, and the zygomaticosphenoid sutures. All zygomatic complex fractures involve the orbital floor, and therefore an understanding of orbital anatomic features is essential for those treating these injuries. The orbit is a quadrilateral pyramid that is based anteriorly. The orbital floor slopes inferiorly and is the shortest of the orbital walls, averaging 47 mm. It is composed of the orbital plate of the maxilla, the orbital surface of the zygomatic bone, and the orbital process of the palatine bone. The medial and lateral walls converge posteriorly at the orbital apex. The medial wall consists of the frontal process of the maxilla, the lacrimal bone, the orbital plate of the ethmoid, and a small portion of the sphenoid body. The lateral orbital wall is the thickest and is formed by the zygoma and the greater wing of the sphenoid. The orbital roof is composed of the frontal bone and lesser wing of the sphenoid. The zygomatic arch includes the temporal process of the zygoma and the zygomatic process of the temporal bone. The glenoid fossa and articular eminence are located at the posterior aspect of the zygomatic process of the temporal bone. The sensory nerve associated with the zygoma is the second division of the trigeminal nerve. The zygomatic, facial, and temporal branches exit the foramina in the body of the zygoma and supply sensation to the cheek and anterior temporal region. The infraorbital nerve passes through the orbital floor and exits at the infraorbital foramen. It provides sensation to the anterior cheek, lateral nose, upper lip, and maxillary anterior teeth. Muscles of facial expression originating from the zygoma include the zygomaticus major and labii superioris. They are innervated by cranial nerve VII. The masseter muscle inserts along the temporal surface of the zygoma and arch and is innervated by branch of the mandibular nerve. The temporalis fascia attaches to the frontal process of the zygoma and zygomatic arch.

Classification of zygomaticomaxillary complex fractures

It is probably fair to say that classification of zygomatic fractures according to the individual who tries to describe them. The result has been a confusing array of classification systems that try to describe the anatomic position of the displaced bone or to classify fractures using position and criteria for postreduction stability.Whether a patient receives better treatment from being classified into one system or another is doubtful, and one should not dwell on the many classification systems available. Asis true for many other aspects of surgery, it is extremely rare to find two patients who have exactly the same condition.

In 1990, Manson et al 40 published a classification of midfacial fractures that was based on the amount of energy dissipated by the facial bones secondary to the traumatic force. Their classification of high-, moderate-, or low-energy fractures was based on findings on computed tomography (CT) scans. High-energy fractures had extreme displacement, comminution of the articulations, and segmentation of the bones. They noted that these required extensive exposure and fixation for a satisfactory outcome. On the other hand, lower energy fractures were characterized by displacement but without comminution of bony articulations. They noted that these could be treated by less aggressive means. Using preoperative CT findings may be the most useful way to decide how much intervention may be required before surgery.

It behooves clinicians to evaluate each case individually. Whether they choose to prescribe treatment based on the experience of others for a given class of fracture is their choice; however, with proper surgical management, the nature of the treatment should depend more on the preoperative imaging analysis and surgical findings than on statistical prescription.

Diagnosis of zygomaticomaxillary complex fractures

The diagnosis of zygomatic fractures is primarily based on clinical and radiologic examination, although the history frequently raises a strong suggestion of the possibility that a fracture may exist and gives an indication about the nature, direction, and force of the blow. It should be stressed that the clinical examination is frequently difficult to perform adequately because of the nature of the patient’s mental state and/or the amount of facial edema and pain. The swelling may conceal facial deformity that appears only after the swelling has subsided. If the examination can be performed immediately following the injury and before the onset of edema, more information can be obtained from the clinical examinations. Because there are no sensitive indicators of zygomatic fractures (e.g., those that the teeth provide in maxillary or mandibular fractures), and because the concomitant soft tissue edema and contusion that frequently accompany zygomatic injuries can obscure clinical examination, the use of imaging and clinical findings is important in the diagnosis of ZMC fractures.

Clinical examination

After the clinician has ascertained the neurologic status of a patient with suspected ZMC fracture, the first priority is determination of the visual status of the involved eye. A thorough ocular and funduscopic examination should be performed, with complete documentation of the findings. Ocular injuries, such as vitreous hemorrhage, hyphema, globe laceration, severance of the optic nerve, and corneal abrasions, were found in 4% of patients with midfacial trauma by Turvey and in 5% of zygomaticoorbital fractures by Livingston et al.

Ophthalmologic consultation was deemed necessary in approximately 5% of 2067 cases of zygomaticoorbital injuries reported by Ellis et al. Ioannides et al found significant ocular and adnexal injuries in 26% of orbital fractures. Al-Qurainy et alprospectively performed ophthalmologic examinations in 363 patients who had sustained midfacial fractures. Minor or transient eye injuries, such as corneal abrasion, chemosis, mild impairment of accommodation and visual acuity, and orbital emphysema, were found in 63% of patients. Moderate injuries, such as enophthalmos, conjunctival abrasion, traumatic pupillary changes, iridodialysis, lens damage, macular edema, and moderate to severe impairment of accommodation and visual acuity, were noted in 16% of patients. Severe ophthalmic disorders, such as gross proptosis, retrobulbar hemorrhage, corneal laceration, hyphema, angle recession, severe reduction or loss of vision, visual field loss, choroidal tear involving the macula, and optic nerve injuries, were found in 12% of patients. One third of all patients with comminuted ZMC fractures suffered a severe ocular disorder. Therefore, if the clinician discovers any significant or questionable findings in patients with midfacial fractures, ophthalmologic consultation should be obtained.

Examination of the zygoma involves inspection and palpation. Inspection is performed from the frontal, lateral, superior, and inferior views. One should note symmetry, pupillary levels, presence of orbital edema and subconjunctival ecchymosis, and anterior and lateral projection of the zygomatic bodies. The most useful method for evaluating the position of the body of the zygoma is from the superior view. The patient can be placed in a recumbent position or recline in a chair. The surgeon inspects from a superior position, evaluating how the zygomatic bodies project anteriorly and laterally to the supraorbital rims, comparing one side with the other. The surgeon should lay his or her index finger below the infraorbital margins, along the zygomatic bodies, pressing into the edematous tissue to palpate and reduce the visual effect of edema simultaneously when performing this examination. The superior view is also helpful for evaluating possible depression of the zygomatic arches. One should not forget to perform an intraoral examination, because zygomatic fractures are often accompanied by ecchymosis in the superior buccal sulcus and maxillary dentoalveolar fractures.Palpation should be systematic and thorough, and one side should be compared with the other. The orbital rims are palpated first. The surgeon palpates the infraorbital rims with the index finger, moving the finger rhythmically from side to side along the rim. The lateral orbital rims are palpated with the index finger and thumb. One should also use the index finger along the inner aspect of the lateral orbital rim because fractures may frequently be detected by palpating inside the orbital rim, asopposed to palpating along the lateral aspect. When fractures are present, palpation frequently is accompanied by exquisite tenderness. The body of the zygoma and zygomatic arch are best palpated with two or three fingers in a circular motion, with the surgeon comparing this palpation with that of the opposite side. The zygomatic buttress of the maxilla is palpated intraorally with one finger, and hematoma or irregularities are sought.

Signs and Symptoms

Several signs and symptoms accompany zygomatic fractures. The presence and magnitude of their severity greatly depend on the extent and type of zygomatic injury. For example, facial flattening is more pronounced in injuries in which the zygomatic body has been greatly displaced, as opposed to those in which the body has not been displaced. Similarly, zygomatic arch fractures may be expected to produce less ocular disruption than ZMC fractures. The following signs and symptoms can accompany zygomatic fractures and therefore should be evaluated.

Periorbital Ecchymosis and Edema

Edema and bleeding into the loose connective tissue of the eyelids and periorbital areas is the most common sign following fracture of the orbital rim.Swelling, often massive, may be present and is most dramatic in the periorbital tissue, where the eyelids may be swollen closed. The ecchymosis may be in the inferior lid and infraorbital area only or around the entire orbital rim.

Flattening of the Malar Prominence

A characteristic sign and striking feature of zygomatic injury is a flattening of the normal prominence in the malar area. An especially common finding in ZMC injuries, this flattening is reported in 70% to 86% of cases, especially those in which distraction of the frontozygomatic suture and medial rotation and/or comminution have occurred. If edema is present, flattening may be difficult to discern soon after injury; however, one can usually gain an appreciation of this sign by depressing the index fingers into the soft tissue of the zygomatic areas and comparing one side with the other from above the patient.

Flattening over the Zygomatic Arch

A characteristic indentation or loss of the normal convex curvature in the temporal area accompanies fractures of the zygomatic arch. Visual and digital comparison with the opposite side is extremely helpful for detection of depressions of the zygomatic arch.

Pain

Severe pain is normally not a feature of zygomatic injuries unless the fractured segment is mobile. Patients do, however, complain of discomfort associated with the attendant bruising. Palpation of the fracture sites also elicits a painful response.

Ecchymosis of the Maxillary Buccal Sulcus

An important sign of zygomatic or maxillary fracture is ecchymosis in the maxillary buccal sulcus. Ecchymosis may occur even with a small disruption of the anterior or lateral maxilla and should be sought in patients with suspected zygomatic fractures.

Deformity at the Zygomatic Buttress of the Maxilla

Intraoral palpation of the anterior and lateral aspects of the maxilla frequently reveals irregularities of the normally smooth contour, especially in the area of the zygomatic buttress of the maxilla. Crepitation from comminuted fragments of bone is also frequently palpable. If no tenderness is experienced during this maneuver, the chances are that no fracture exists. The absence of pain makes a zygomatic fracture unlikely, but its presence does not establish one because the pain can be a result of soft tissue injury and/or maxillary fracture.

Deformity of the Orbital Margin

Fractures running through the orbital rim often result in a gap, or step deformity, if displacement has occurred. This finding is frequently noted at the infraorbital and lateral orbital rims when zygomatic fractures are present. These areas may also be tender to touch.

Trismus

Limitation of mouth opening frequently accompanies zygomatic injuries and is present in approximately one third of cases. This condition occurs with an even higher incidence in isolated fractures of the zygomatic arch (45%). The reason often cited for postfracture trismus is impingement of the translating coronoid process of the mandible on the displaced zygomatic fragments. Whether this contact actually occurs in most cases is doubtful, because the amount of displacement necessary for producing actual mechanical interference is great. A more likely explanation is muscle spasm secondary to impingement by the displaced fragments, especially on the temporal muscle. An associated finding is deviation of the mandible toward the fractured side when the mouth is opened.

Abnormal Nerve Sensibility

An important symptom, present in approximately 50% to 90% of ZMC injuries, is impaired sensation of the infraorbital nerve. Infraorbital nerve paresthesia is more common in fractures that are displaced than those that are not. It is difficult to differentiate true anesthesia from the altered sensation of swollen edematous tissue but, as the swelling decreases, infraorbital nerve anesthesia becomes apparent. Infraorbital anesthesia occurs when the fracture through the orbital floor and/or the anterior maxilla causes tearing, shearing, or compression of the infraorbital nerve along its canal or foramen. Frequently, the entire orbital floor is comminuted, which results in multiple fragments of bone strung together by the infraorbital neurovascular bundle. When the line of fracture is lateral to the infraorbital groove and foramen (less common), the infraorbital nerve is spared. Disruption of the infraorbital nerve causes anesthesia of the lower eyelid, upper lip, and lateral aspect of the nose.A related symptom may be altered sensitivity of the maxillary teeth and gingiva. When this altered sensitivity is present, the clinician should suspect a disruption of the infraorbital nerve within its canal, where the middle and anterior superior alveolar nerves take origin.

Epistaxis

Whenever the sinus mucosa is disrupted, hemorrhage into the sinus is possible. Most fractures through the sinus wall that have had even a minor amount of displacement tear the lining mucosa, producing internal bleeding. Because the maxillary sinus drains into the nose via the middle meatus, unilateral hemorrhage from the nose is possible and occurs in approximately 30% to 50% of ZMC injuries.

Subconjunctival Ecchymoses

Subconjunctival hemorrhage, a frequent finding in zygomatic fractures, is present in 50% to 70% of cases. It may accompany even a hairline crack through the orbital rim if the periosteum has been torn. Its absence does not exclude an orbital rim fracture because if no disruption of the periosteum has occurred, bleeding can accumulate in a subperiosteal location and may not be visible under the conjunctiva. When present, subconjunctival ecchymoses usually have no posterior limit and will be bright red because of the ability of oxygen to diffuse through the conjunctiva to the collection of blood.

Crepitation from Air Emphysema

Fracture through a sinus wall with tearing of the lining mucosa allows air toescape into the facial soft tissue if the pressure within the sinus is greater than that within the tissue. The soft tissue of the periorbital area, especially the eyelids, is prone to inflation with air because of its loose areolar nature. When inflation occurs, one can palpate crepitation, indicating subcutaneous emphysema. Crepitation is most easily appreciated by alternatively rolling two fingers gently over the tissue, which produces a characteristic crackling sensation. It is an uncommon finding following zygomatic fractures, but the potential for air emphysema is constant. When present, however, crepitation can be alarming to the patient. The emphysema disappears spontaneously in 2 to 4 days without treatment. The significance of emphysema is the potential for infection through the communication between the sinus and the soft tissue.

Displacement of the Palpebral Fissure

The lateral palpebral ligament is attached to the zygomatic portion of the orbital rim. Displacement of the zygoma carries the palpebral attachment with it and thus produces a dramatic visual deformity. When the zygoma is displaced in aninferior direction, the lateral palpebral ligament is also depressed, causing a downward slope to the fissure (antimongoloid slant). Because the orbital septum is attached to the infraorbital rim, inferior or posterior displacement of the inferior orbital rim causes depression of the lower eyelid, giving it a shortened appearance. This depression may cause more sclera to be exposed below the iris and an apparent ectropion.

Unequal Pupillary Levels

With the disruption of the orbital floor and lateral aspect of the orbit that frequently accompanies zygomatic fractures, loss of osseous support for the orbital contents and displacement of Tenon's capsule and the suspensory ligaments of the globe permit depression of the globe. This displacement is manifested clinically as unequal pupillary levels, with the involved pupil at a level lower than that of the normal side.

Diplopia

Diplopia is the name given to the symptom of blurred vision. Two varieties of diplopia exist; it is important to distinguish between them. Monocular diplopia, or blurring of vision through one eye with the other closed, requires the immediate attention of an ophthalmologist, because it usually indicates a detached lens, hyphema, or other traumatic injury to the globe. Binocular diplopia, in which the blurring of vision occurs only when the patient looks through both eyes simultaneously,is common and occurs in approximately 10% to 40% of zygomatic injuries.Al-Qurainy et al have found that the severity of diplopia is associated with the severity of midfacial injuries. Almost 30% of patients with comminuted fractures of the ZMC experienced diplopia, 22% of patients with noncomminuted displaced ZMC fractures had diplopia, and only 8% of patients with minimally displaced or nondisplaced ZMC fractures had diplopia. Binocular diplopia that develops following trauma can be the result of soft tissue (muscle or periorbital) entrapment, neuromuscular injury, intraorbital or intramuscular hematoma or edema, or a change in orbital shape, with displacement of the globe causing a muscle imbalance. Enophthalmos and globe ptosis associated with marked displacement of the globe can also cause diplopia. A useful point in differentiating the cause of diplopia is the finding that general edema of the orbit usually causes diplopia in the extremes of upward and downward gaze. Almost complete lack of eye movement in one direction is present with mechanical interference or neuromuscular injury, most commonly muscle entrapment. The diagnosis of diplopia can be difficult in the early stages of an injury, when severe edema of the orbit and eyelids is present. Diplopia of edema or hemorrhagic origin should resolve in a few days, whereas diplopia caused by entrapment of orbital tissue does not.One can determine the presence of entrapment of orbital contents by the fracture through the orbital floor with a forced duction test. Small forceps are used to grasp the tendon of the inferior rectus through the conjunctiva of the inferior fornix and the globe is manipulated through its entire range of motion. Inability to rotate the globe superiorly signifies entrapment of the muscles in the orbital floor. This test should differentiate between entrapment of orbital contents and paralysis as a result of neuromuscular injury or edema. The test should be performed routinely in those who cannot rotate the globe into an upward gaze.

Enophthalmos

If the zygomatic injury has produced an increase in orbital volume, usually by lateral and inferior displacement of the zygoma and/or disruption of the inferior, medial, and/or lateral orbital walls, or has resulted in a decrease in orbital soft tissue volume by herniation of orbital soft tissue, enophthalmos can result. This diagnosis is difficult to make acutely unless the enophthalmos is severe because adjacent soft tissue edema always produces a relative enophthalmos. After the swelling has dissipated, enophthalmos becomes more obvious and is frequently associated with ptosis of the globe. The clinical manifestations of enophthalmos are accentuation of the sulcus of the upper lid and narrowing of the palpebral fissure, causing pseudoptosis of the upper lid. The anterior projection of the globe as viewed from above is reduced on the side of injury. Zygomatic fractures are associated with enophthalmos in approximately 5% of cases before treatment. If enophthalmos is present during the initial examination, it is likely that a great increase in bony orbital volume has occurred.

Radiographic Evaluation

The diagnosis of zygomatic fractures is usually established by history and physical examination. CT scan of the facial bones, in axial and coronal planes, is standard for all patients with suspected zygomatic fractures. Radiographs are helpful for confirmation and for medicolegal documentation and to establish the extent of the bony injury.

CT is the gold standard for radiographic evaluation of zygomatic fractures. Axial and coronal images are obtained to define fracture patterns, degree of displacement, and comminution and to evaluate the orbital soft tissues. Specifically, CT scans allow for visualization of the buttresses of the midfacial skeleton: nasomaxillary, zygomaticomaxillary, infraorbital, zygomaticofrontal, zygomaticosphenoid, and zygomaticotemporal buttresses. Coronal views are particularly helpful in the evaluation of orbital floor fractures. Soft tissue windows, in the coronal plane, are useful to evaluate the extraocular muscles and to evaluate for herniation of orbital tissues into the maxillary sinus.

Treatment

Treatment of zygomatic fractures must be based on a complete preoperative evaluation. This includes a CT scan with axial and coronal images to fully appreciate the nature of the injury. Classification techniques, if they are accepted, are helpful to standardize terminology, to plan treatment, and to predict prognosis. However, the surgeon must individualize treatment based on a combination of history, physical examination, radiographic findings, and sound clinical judgment. Management of zygomatic complex and zygomatic arch fractures depends on the degree of displacement and the resultant esthetic and functional deficits. Treatment may therefore range from simple observation of resolving swelling, extraocular muscle dysfunction, and paresthesia to open reduction and internal fixation of multiple fractures.

Principles in the treatment of Zygomaticomaxillary complex fractures

In the treatment of any ZMC fracture that requires surgical intervention, consideration should be given to each of several steps in a sequential and orderly manner.

Prophylactic Antibiotics

The incidence of infection following ZMC fracture or fracture reduction is extremely low; however, such an infection is difficult to discern because many surgeons routinely use prophylactic antibiotics. This practice also makes it difficult to determine the effectiveness of antibiotics in preventing infection of these fractures. Because the maxillary sinus is involved, ZMC fractures can be considered compound, and prophylactic antibiotics are probably appropriate, especially given the fact that the orbital contents are also frequently violated. The choice of antibiotics should cover routine sinus bacteria (e.g., ampicillin, amoxicillin, clindamycin, cephalosporin).

Anesthesia

For isolated ZMC fractures, general anesthesia with oral intubation is helpful. The anesthesiologist or anesthetist should be positioned so that the surgeon has access to the side of the fracture and head of the table. It is very important to have complete access to the top of the patient's head for visual comparison of one side with the other. (Reduction of isolated zygomatic arch fractures can be performed with the patient under local anesthetic, with or without sedation when the patient is cooperative, and an intraoral or a percutaneous approach is used.)

Clinical Examination and Forced Duction Test

Following induction of general anesthesia, the surgeon should take the opportunity to examine the patient more carefully. With the patient under anesthesia, the surgeon has more freedom in the examination and can use more digital force than is possible with the patient awake. This examination can help confirm previous diagnoses and may reveal new information. It is very important to look at the patient from the superior view and to visualize both zygomas simultaneously. Unless the swelling is marked, one should be able to determine an asymmetry. Laying the index finger across the infraorbital area or on the malar prominence should help discern the asymmetry. A forced duction test should also be performed at this time.

Protection of the Globe

The cornea must be protected from inadvertent trauma. Of the several ways of providing this protection, perhaps the simplest is placement of a scleral shell (corneal shield) after application of an ophthalmic ointment. Temporary tarsorrhaphy can also be used by suturing the dermal surfaces of the upper and lower eyelids together with 5-0 nylon sutures.

Antiseptic Preparation

The type of preparation necessary depends largely on the type of approach(es) that are anticipated. It is good practice, however, to prepare the forehead, both periorbital areas and cheeks to the level of the mouth, and both sides of the preauricular area. Such preparation allows comparison of the affected side with the opposite side during surgery. Another useful suggestion is always to prepare the mouth with throat pack and antiseptic rinse, because an oral approach to the sinus and/or zygoma is frequently useful. If the preoperative clinical and radiographic examinations suggest that a coronal approach may be necessary, the hair and ears are prepared and draped.

Reduction of the Fracture

The fracture should be reduced by whatever means the surgeon deems appropriate (techniques described later).

Assessment of Reduction

The most important step in the management of ZMC fractures is to determine at the table whether the fracture has been properly reduced. The success or failure of reduction will be obvious for those who have opened the fracture at three sites. If exposure at three sites has not been performed, the orbital margins are the areas that should be palpated first to determine reduction. If reduction has been satisfactory, these margins will be smooth and continuous. This finding by itself, however, is inadequate verification that the zygoma is properly positioned. Although the zygomaticofrontal suture area provides the strongest pillar of the zygoma, it is one of the worst indicators of proper reduction of the entire complex, even when surgically exposed and evaluated directly. One should also palpate in the maxillary vestibule. If there is any flatness still visible, the zygoma has not been properly elevated. If there is any doubt about proper reduction, exposure is mandatory. In this case, an incision in the maxillary vestibule offers excellent exposure of the zygomaticomaxillary buttress and the infraorbital rim. For surgeons who have navigation or intraoperative CT scanning available, assessment of the reduction is relatively easy.

Determination of the Necessity for Fixation.

The second most important step in surgically treating zygomatic fractures (following determination of whether the reduction has been satisfactory) is determining whetherthe reduction will be stable by itself or needs some form of fixation. If constant reduction force is necessary for maintaining ZMC position, the ZMC should be stabilized with some form of fixation device(s). If the zygomatic position is deemed appropriate and does not require constant application of reduction force, one should press with moderate pressure on the malar eminence with the fingers and see whether displacement results. If it does not, fixation devices may be unnecessary. Many minimally displaced cases are stable after they have been reduced. However, if there is any doubt about postreduction stability, the application of fixation devices is prudent.

Application of a Fixation Device

The methods of stabilizing the fractured ZMC vary with the imagination and experience of the surgeon. General principles are involved, however (see later).

Internal Orbital Reconstruction

When indicated, reconstruction should be carried out after repositioning and stabilizing the ZMC fracture. In such cases, the orbital floor and walls should be exposed before elevation of the ZMC so that the open orbital rim can also serve as a guide to reduction. However, it is unwise at this point to try to free any trapped tissue, because elevation of the zygoma may separate bone fragments and make this maneuver much easier following reduction. Assessment of the magnitude of the defect to be reconstructed is made following reduction, because the actual defect will then be revealed (techniques described later).In minimally displaced cases in which no ocular signs of entrapment or enophthalmos are noted preoperatively, and in which the fracture is treated by simple reduction, internal orbital exploration and/or reconstruction is unnecessary unless a postreduction forced duction test produces positive findings (rare). In most of these cases, reduction of the zygoma results in adequate alignment of the orbital floor. However, one should never avoid reconstructing the internal orbit for fear of causing harm to orbital tissue. This occurrence is extremely rare. For those surgeons who have intraoperative CT scanning capability, the status of the internalorbit after reduction of the ZMC is known and the decision about the need for internal orbital reconstruction can be made during the surgery.

Surgical Approach to the Zygomatic Arch

In high-energy zygomatic complex fractures or secondary correction of zygomatic deformities, access is limited with conventional incisions. To obtain adequate exposure, a coronal incision combined with a lower eyelid approach is recommended. The initial incision is through the skin, subcutaneous tissue, and galea of the scalp. Elevation of the coronal flap proceeds in the subgaleal loose areolar connective tissue superficial to the pericranium. The temporal and preauricular plane of dissection is along the temporal fascia, which can be identified by its characteristic glistening white appearance. A horizontal periosteal incision is made 2 to 3 cm above the supraorbital rim, and a subperiosteal plane of dissection is developed to the superior and lateral orbit. An incision is made in the superficial layer of the temporal fascia from the posterior zygomatic arch to the previously exposed supraorbital region. The temporal fat pad should be identified. The dissection is extended inferiorly at this depth to the zygomatic arch and anteriorly to the lateral orbital rim. The facial nerve is protected within the flap.

Internal Fixation

Historically, many methods have been used for stabilization of zygomatic complex fractures. These have included antral packing, percutaneous wire fixation, and wire osteosynthesis. It is now accepted that miniplate or microplate fixation provides the best results and minimal complications. Controversy exists regarding the best location for internal fixation and the number and type of plates required. Multiple studies have tried to characterize the forces placed on the zygomatic complex and the amount of fixation required to achieve «stability». These forces include the masseter and temporalis muscles and fascia and soft tissue contracture, which cause rotational movements in multiple axes around the zygomatic buttresses. Internal fixation must provide enough strength to resist these forces. For low- and middle-energy fractures, stable fixation can be achieved at one or more of the anterior buttresses. The location of fixation and number of sites of fixation depends on the fracture pattern, location, vector of displacement, and degree of instability. Occasionally one point fixation may be adequate. More commonly two- or three-point stabilization is required. For high-energy injuries, a fourth point of fixation is required. The zygomatic arch is typically comminuted and laterally displaced. Open reduction and internal fixation is required to restore proper facial width and projection.

Internal Fixation of the Zygomaticomaxillary Buttress

The zygomaticomaxillary buttress provides an ideal location for internal fixation for middle- and high-energy fractures. Anatomic reduction of this fracture assists in restoring malar projection, but is difficult if the buttress is comminuted. The overlying soft tissue is thick, and plate palpability is not a concern. Therefore, this fracture should be stabilized with 1.5 or 2.0 plates.

Internal Fixation of the Zygomaticofrontal Buttress

The zygomaticofrontal buttress contains excellent bone for fixation and can accommodate a 2.0 plate. The reduction and fixation of this fracture will reestablish the vertical height of the zygomatic complex. However, because of its narrow interface, this buttress may not be as helpful in evaluating reduction of a rotated fracture. The thickness of the soft tissue overlying this region is variable. In some instances it may be quite thin and a large plate may be palpable. If stable fixation can be achieved at other sites, a smaller plate may be used.

Internal Fixation of the Infraorbital Rim

Unlike the zygomaticofrontal buttress, the infraorbital rim has poor quality bone for internal fixation. Additionally, the lower eyelid skin is quite thin, and large plates are easily palpable. Despite these concerns, fixation of this site is required to define the orbital volume and facial width. The infraorbital rim is typically displaced posteriorly and inferiorly. The fracture should be mobilized anteriorly and superiorly and stabilized. Typically a 1.0 or 1.5 microplate is used to stabilize the infraorbital rim. A potential pitfall in reduction of this fracture is an unappreciated heminasoethmoid fracture. If the infraorbital rim is secured to this undiagnosed displaced segment, postoperative facial widening may occur.

Internal Fixation of the Zygomatic Arch

Internal fixation of the zygomatic arch is required for high-energy fractures that demonstrate comminution and lateral displacement. Restoration of this sagittal buttress assists in restoring facial projection and facial width. When exposed, the zygomatic arch is often reduced and stabilized first in the sequence of repair of high-energy injuries. Caution must be used in restoring a «straight» arch and not a «curved» arch, which will decrease facial projection. This fracture typically requires a large plate to resist deformational forces.

Postoperative Care

Zygomatic complex fractures violate the maxillary sinus. For this reason, antibiotics and decongestants are recommended. Ampicillin, amoxicillin, clindamycin, or cephalosporin may be used. A decongestant such as pseudoephedrine is also used to clear the airway. Incisions are observed carefully for signs of infection, and the eye is examined to document visual acuity and to rule out complications such as corneal abrasion. Postoperative radiographs (Waters' view and submentovertex view) are obtained to document reduction of the fracture. A CT scan may be obtained in comminuted fractures to evaluate the zygomatic complex reduction and orbital reconstruction.

Complications

Although complications of zygomatic complex and zygomatic arch fractures are uncommon, the surgeon must recognize their signs and symptoms to provide appropriate care. Complications may occur in the early postoperative period or may only become manifest later in recovery.

Complications: infraorbital paresthesia, malunion and asymmetry, enophthalmos, diplopia, traumatic hyphema, traumatic optic neuropathy, retrobulbar hemorrhage, trismus.

TOPIC: COMBINED TRAUMA

The maxillofacial area traumas have specific characteristics arising from their topographic, anatomic-physiology and functional maxillofacial area (MFA) features. Near it, vital organs are located and their damage explains the large complexity and variety in clinical picture. Head and neck are one of the most resilient, but also of most vulnerable topographic area in human body. There is no such other area with so many vital structures, located on so restricted area. Traumas, penetrating or not, regardless of the force and mechanism of injury, can cause life-threatening conditions because of possible damage of brain, eyes, trachea, larynx, esophagus, large blood vessels, cerebral nerves or spinal cord, as well as cervical root damages. Surgeons dealing with these injuries must be aware about anatomic structures of head, maxillofacial area and neck, in order to provide safe, fast and predictable as a result treatment.

Combined trauma have always been interesting for medical thought and practice. At some part of these traumas, except damages in maxillofacial area, there are also serious injuries in other organs and systems, which is one of the reasons those patients to not receive medical aid and therapy of facial trauma in due time. To prevent omissions in diagnostics and to improve prognosis, in conformity with ATLS principles, overall patient's examination is required, as well as early adequate consultations to relevant specialists, as well as dynamic monitoring and re-estimation of their status.

Definition of combined trauma. Per International Classification of Diseases these injuries, in which several anatomic areas are damaged at the same time, are determined as combined traumas. These traumas require interdisciplinary (multi profile) diagnostic and therapeutic approach.

Definition of polytrauma and polytraumatism. A polytrauma is a combined trauma, in which there are two or more severe injuries that affect at least two anatomical regions; rarely, two or more severe injuries in one anatomic area, whereas at least one of these is life-threatening. The term "polytraumatism" that is used in practice, is not actually a synonym of polytrauma, but is directly related to it. Polytraumatism is a concept that requires a complex of diagnostic therapeutic measures and includes solving of serious social problems arising from polytrauma.

Combined injures

To combined injuries refer all the injuries, which appear by the impact on the body of two or more striking factors (shock wave and luminous radiation, gunshot wounds with the impact of chemical war gases etc.).

All the combined injuries, regardless of their origin, have several common features:

- Syndrome of reciprocal complication (the presence of the one type of injury aggravates the course of the other and vice versa);
- Not only the first medical aid gets more complicated, but also the whole process of the following treatment;
- There are often poor functional results of the treatment.

In association with the combined injuries it is necessary to remind about such concepts like degasification and decontamination.

Degasification – neutralization and (or) removal of the toxic substances from the surface or from the volume of the infected objects, done with the purpose of preventing destruction to people.

Decontamination – is the removal of radioactive substances from the surface or from the volume of the contaminated objects, which is led with the purpose of radiation injuries prevention.

Combined radiation injures

The effects of radiation injury on the course of a gunshot wound develop on the following way:

The reparative processes in the wound slow down and get subverted;

The development of infectious complications of local (wound suppuration) and general (sepsis) character becomes more frequent;

The duration of the latent period of the radiation disease decreases;

The gravity of the radiation injury increases;

Threshold of the radiation sickness development against the background of a severe gunshot wound decreases.

By combined radiation injuries the primary surgical debridement of the wound should be done at possibly early terms. The primary surgical debridement of the wound ends with the overlay of a primary suture or conduct of the skin plate. A special role belongs to prophylaxis of the wound infection development; this means the healing takes place under the guise of antibiotic therapy. The syndrome of reciprocal burdening is more clearly manifested in the midst of the radiation disease. That is why there appears the main rule of wound treatment by combined radiation injuries – it is necessary to use the hidden period of the radiation disease to perform the surgical measurements (primary surgical debridement of the wound, primary skin plastic, reconstructive surgery).

The surgical debridement should be done at early terms, in the first 24 hours after the injury if possible, better not later than in 48 hours.

The debridement must be momentary, exhaustive and it must end with the fixation of jaw fragments (if there is a necessity), imposition of primary sutures on the soft tissue wound, local and intravenous antibiotic.

General principles of the surgical debridement of the maxillofacial area wounds are preserved also by combined injuries. The revision of the wound must be done thoroughly, besides for the stop of bleeding in the wound should be used not the usual ligation of vessels, but if possible the sewing of bleeding vessels with soft tissues.

Taking into consideration that the mucosa or skin wounds which were not sewn during the debridement, may turn into big infected necrotic ulcers in the midst of the radiation disease, that why one must aim in any case to close the wound with the simple convergence of the edges or by means of cutting out and moving the flap from the nearby tissues.

Foreign bodies, also metallic ones, are eliminated according to general indications. Metallic prosthesis, inlays and other constructions in the oral cavity may be left during the patient's debridement, if there are no direct indications for their elimination for other reasons (tooth mobility under the crown in the fracture area etc.).

The use of tooth metal tires for the fixation of jaw fragments should be limited; there must be wider used operational methods of immobilization of bone fragments, especially by large doses of radiation.

The surgical debridement of the wounds, which were occasionally infected by radioactive substances, is done by rules, which were adopted in the maxillofacial surgery, but more radically. Metallic foreign bodies, which lie next to the wounded surface, are possibly to be removed, because they can carry on them radioactive particles.

At presence of blind pockets and courses, the last should be split for the removal of foreign bodies, teeth and bone fragments, and also for cleaning and aeration of the wound.

The edges of the wound are brought together, and the intervals are plugged loosely with gauze and are closed with aseptic bandage. These swabs should be changed daily. Later, if the clinical course is favorable, such wounds may be closed by means of a secondary suture.

In the midst of the radiation disease some bleeding and wound sepsis appears different processes in the maxillofacial area join (gingivitis, stomatitis, petechial and drain hemorrhages under the mucosa, ulcerative-necrotic lesions of gums or tonsils etc.). In this period even a drastically made surgical debridement of the wound and the active antibacterial therapy don't usually bring success.

Combined chemical injures

The maxillofacial wounds may be struck by poisoning substances of two types: a) those which have a local and those which have a general effect; b) those which have a general resorptive effect. The local effect of the poisoning substances lies in the development of a marked inflammatory-necrotic process, besides in the more or less important deceleration of the wound cleaning processes and reparatory processes. A severe disturbance of trophic tissue and a decrease of the overall resistance of the organism highly contribute to the development of infectious complications' development. Along with the local effect, which several poisoning substances have, the most typical by wound infection is their general resorptive effect. The character of the general resorptive effect is determined by the particularities of the composition of the used CWF. The absorption of the poisoning substances through the wounded surface takes place quicker, than through the skin. In connection with it by defeat of PS the minimal killing dose reduces significantly.

By *mustard gas* poisoning the wound has a specific odor of burnt rubber or mustard. Sometimes in the wound black oil spots of mustard gas can be seen. In the coming hours after the injury the swelling of its edges is detected. At the end of the day there are bubbles on the congested skin around the wound. And the wound covers with necrotic membrane. In the future the tissue necrosis progresses, an infection develops, and the clearing and wound healing processes are delayed for a long time. The symptoms of the general resorptive effect of the poisoning substances are determined shortly after the injury and express themselves in the general retardation of the patient, loss of appetite, nausea, vomiting, headache, dizziness. In more severe cases there are convulsions and comatose state, not rare with lethal outcome.

By *poisoning with lewisite* the wound smells like geraniums. In the moment of contamination the injured feels a sharp ache, which isn't usual for a trauma. In the first minutes after the contamination the wound tissues get gray color, which is followed by yellowish-brown color. Shortly afterwards around the injury pronounced effects of inflammation will develop, and after 6-8 hours petechial hemorrhages appear in the skin surrounding the wound. At the end of the day bubbles will appear here, which will gradually merge, and by massive contamination the wound edges with get pale yellow color (lifetime fixation of tissues). A high wound bleeding is observed. The bleeding sometimes takes threatening character. In 3-4 days after the injury the wound surface will cover with dry necrotic membrane. Soon an infection will develop. By the contamination of wounds with lewisite the effects of intoxication are seen earlier and they are more pronounced then at mustard gas poisoning. To the number of the typical symptoms of the intoxication refer: weakness, dyspnea, pulmonary edema, collapse.

By *phosphorus poisoning* the wound has a specific odor, which is similar to garlic, and the skin around it is burnt. The injured tissues are covered with grey crust, they are smoking. Sometimes the clothes or bandage inflames. Later a sera-purulent discharge from the wound appears. As a result of the general resorptive effect jaundice appears in the following 2-3 days, there appear bleedings in the intestines and urinary tracts, bleedings into the skin and mucosa. Subsequently on the first plan come the effects of the hepatic failure, which can lead to the development of coma with a lethal outcome in the next hours after the injury.

Primary surgical debridement of the wound is one only after the medical relief action of poisoning substances. There are antidotes for some poisoning substances.

The wound toilet of the maxillofacial area goes together with its *degasification*. By *mustard gas poisoning* the surrounding skin is debrided with 10% solution of chlorine bleach, and the wound with 2% aqueous solution of chloramine. By *lewisite poisoning* the wound is debrided with 5 % solution iodine, and the wound with aqueous solution of Lugol.

By primary surgical debridement of the wound all the foreign bodies and bone fragments must be removed. A careful hemostasis is done. During the operation the wound is periodically washed with 2% aqueous solution of chloramine for the removal of wound detritus and its degasification. The wound is plugged loosely with gauze swabs, which are wetted in a 2% aqueous solution of chloramine. Afterwards on the wound are imposed a delayed primary and early secondary sutures.

The infected bandaging material is folded in geometric receivers and afterwards it is burnt. The decontamination of instruments is done through the way of its careful wiping with cotton, which is wetted with petrol, and it is boiled after it during 25-30 minutes in 2% bicarbonate of soda solution. The infected gloves are mechanically cleaned with soaped water, and after it they are drown for 20 minutes in a 5% solution of chloramine and in conclusion they are boiled (in normal water) during 15-20 minutes.

Antidotes, form, methods of application	Toxic substances
Alloximum lyophilized – ampules of 75 mg, intramuscularly	OPs
Amyl nitrite (propyl nitrite) – ampules of 0,5 ml for enhance	Hydrocyanic acid, cyanides
Anti-cyan – ampules of 1 ml of 20% solution intravenously, 0,75 ml intramuscularly	Hydrocyanic acid, cyanides
Atropine sulfate – ampules of 1 ml of 0,1% solution, intramuscularly, intravenously	OPs
Dicaptolum – ampules of 1 ml, intramuscularly	Hydrogen arsenide, OMC
Trimedoxime bromide – ampules of 5 ml of 10% solution, intramuscularly	OPs
Diaethyximum –ampules of 5ml of 10% solution, intramuscularly	OPs
Cobalt edetate – ampules of 20 ml of 1,5% solution, intramuscularly slowly in drops	Hydrocyanic acid, cyanides
Izonitrozinum – ampules of 3 ml of 40% solution, intramuscularly	OPs
Calcium chloride – ampules of 10 ml of 10 % solution, intramuscularly	Oxalic hydrofluoric acid
Oxygen (inhalation)	Carbon monoxide, hydrogen sulfide etc.
Magnesium oxide – 20-40 mg in 1 liter of water (gastric lavage)	Inorganic acids
Methylene blue – ampules of 20 ml or flacons of 50-100 ml of 1% solution in 25%solution of glucose (“Chromosmon”), intravenously	Hydrocyanic acid, cyanides, aniline, nitrobenzene
Sodium nitrite – ampules of 10-20 ml of 2% solution, intramuscularly in drops	Hydrocyanic acid, cyanides
Sodium thiosulfate – ampules of 10-20 ml of 2% solution, intravenosly	Ferrihemoglobin formers, Hydrocyanic acid, cyanides, mercury, arsenium compounds
Pyridoxine hydrochloride – ampules of 3-5 ml of 5% solution, intravenously, intramuscularly	Hydrazine
Succimerum – flacons of 300 ml, intramuscularly	Mercury
Tetacinum-calcium – ampules of 20 ml of	OMC, arsenium, dichloroethane

10% solution, intravenously in drops in 5% solution of glucose	
Activated carbon (suspension of 20-30 g in water in or for gastric lavage)	By all enteral contaminations
Unithiolum – ampules of 5 ml of 5% solution (1 ml for 10 kg of body weight), intramuscularly	Arsenium, mercury, other heavy metals
Ethanol – 30 % solution inside for 50-100 ml, intravenously (1 ml for 10 kg of body weight per 24 hours in 5% solution)	Methyl alcohol, ethylene glycol

Polytrauma

Management of the multiply injured patient requires a co-ordinated multi-disciplinary approach in order to optimise patients' outcome. A working knowledge of the sort of problems these patients encounter is therefore vital to ensure that life-threatening injuries are recognised and treated in a timely pattern and that more minor associated injuries are not omitted. This article outlines the management of polytraumatized patients using the Advanced Trauma Life Support (ATLS) principles and highlights the areas of specific involvement of the engaged medical team. Advanced Trauma Life Support is generally regarded as the gold standard and is founded on a number of well known principles, but strict adherence to protocols may have its drawbacks when facial trauma co-exists. These can arise in the presence of either major or minor facial injuries, and oral and maxillofacial surgeons need to be aware of the potential problems.

Maxillofacial trauma is without doubt a most challenging area within the specialty of oral and maxillofacial surgery. Despite the many advances in our understanding of tissue healing, biomaterials and surgical techniques, the initial assessment and the timing and undertaking of management of facial injuries in the early stages have remained a difficult area of patients' care. Appropriate and timely management of facial injuries becomes even more challenging following high velocity trauma, when significant injuries elsewhere may, or may not, take priority.

This reinforces the concept of the mechanism of injury, which supports screening for the following injuries.

1. High-velocity impacts (e.g. motor vehicle collision at a pedestrian, ejection from vehicle, airbag deployment, fatalities or severe injuries to other vehicle occupants).

2. Falls from a height (typically greater than the height of the individual or >6 feet).

3. In patients with pelvic fractures or lower limb long-bone fractures.

Familiarity with an algorithm for the assessment and initial treatment of traumatized patients provides the treating physician with the confidence to competently manage polytrauma, and ensures optimal outcome for the patient. Advanced trauma life support (ATLS) is a system of simultaneous assessment and treatment of multiple trauma patients.1 It is based on identification and safe initial management of all injuries. In particular, it prioritizes diagnosis and effective management of life-threatening injuries. Having originally been conceived in Nebraska in the 1970s, its principles are now taught in courses all over the world and form the cornerstone of management of the patient with multiple trauma. Since its inception in 1978, the Advanced Trauma Life Support (ATLS) system of care has generally become accepted as the gold standard in the initial management of the multiply injured patient and is now taught in over 40 countries worldwide.

This approach is based on the three well established principles of:

1. ABCDEs of assessment (Airway maintenance with cervical spine protection, Breathing with ventilation, Circulation with haemorrhage control, Disability; neurological status, and Exposure/ Environment);

2. "primum non nocere" (first, do no harm);

3. treatment of life-threatening injuries within the ‘golden hour’.

Initial evaluation and treatment In the setting of a major trauma unit, the management of seriously injured patients involves the coordinated approach of a multidisciplinary team with simultaneous assessment and treatment of a variety of life-threatening conditions. The care of the patient after hospital admission is divided into the following phases:

1. Primary survey.
2. Adjuncts to primary survey and resuscitation.
3. Secondary survey (head-to-toe evaluation and history).
4. Adjuncts to the secondary survey.
5. Continued post-resuscitation monitoring and reevaluation.
6. Definitive care

A history of the accident and the circumstances may provide important clues to the likely pattern of injury, but in many multiple trauma patients a standard medical history cannot be obtained, due to the severity of the injuries and a reduced level of consciousness.

The mnemonic for the primary survey is given by the letters ABCDE.

• Airway maintenance with cervical spine protection.

• Breathing and ventilation.

• Circulation with haemorrhage control.

• Disability: neurological status.

• Exposure/environmental control – undress the patient but prevent hypothermia

Airway with cervical spine immobilization

If a patient is not able to maintain their own airway, for whatever reason, irreversible cerebral damage can occur in as little as 4 minutes. It is therefore essential that a safe and secure airway is established as the first step. In maxillofacial trauma or other conditions associated with immediate difficulty in gaining an airway, an emergency cricothyroidotomy can be carried out as a temporary measure. In 10–15% of poly-traumatized patients there will have been an associated spinal injury, of which 55% occur in the cervical spine. A proportion will be unstable and injudicious manipulation of the spine, as may be done to secure an airway, runs the risk of spinal cord injury. Therefore an equal priority must be given to the in-line traction applied by hand on either side of the head to maintain stabilization of the cervical spine while trying to secure an airway.

If there is no evidence of airway compromise, or once an airway breathing is spontaneous, the usual procedure moves attention to next issue. The lungs are vital for maintaining oxygenation of the body tissues as well as removing waste carbon dioxide and helping to maintain acidbase balance. There are six causes of life-threatening respiratory compromise: upper airway obstruction, tension pneumothorax, open pneumothorax, flail chest, massive haemothorax and cardiac tamponade. Initial treatment consists of removing the mechanical problem (insertion of an intercostal drain for haemo/ pneumothorax; application of an occlusive dressing for sucking chest wound), providing high-flow oxygen and providing mechanical ventilator support if necessary. In patients with a suspected spinal injury, it is always important to remember the possibility of spinal shock. This occurs in patients who have a cord injury above the level of the thoracic sympathetic outflow. The haemodynamic result is hypotension and bradycardia. It is the bradycardia that tends to differentiate spinal shock from hypovolaemic shock, although patients on â-blockers or with cardiac arrhythmias may also have bradycardia. Treatment of spinal shock involves the judicious use of intravenous fluids combined with vasopressors to increase the resting vascular tone. ATLS teaches that ‘trauma occurring above the clavicle should raise a high index of suspicion for a potential cervical spine injury' and strict application of this principle means all patients with maxillofacial or craniofacial trauma must be included in this group.

Accordingly, maxillofacial trauma patients must be initially managed with a: cervical spine collar until clinical and radiological clearance is confirmed, comprehensive neurological examination including cranial nerves – pecific assessment for cerebrospinal fluid rhinorrhea.

Circulation

Maintaining adequate tissue perfusion and hence oxygenation requires an adequate circulating blood volume, adequate vascular tone and a normally functioning heart. In the vast majority of trauma victims, the main reason for circulatory compromise is acute blood loss leading to haemorrhagic shock. However, some other causes of shock do occur and should also be considered.

These include: cardiogenic shock, tension pneumothorax, neurogenic shock, septic shock. Haemorrhagic shock: while some bleeding may be obvious, frequently the exact source may not be readily apparent.

The common sites for major occult loss are: the chest, in cases of haemothorax; the abdomen, from ruptured viscera; the pelvis, from an unstable pelvic fracture; from multiple closed long bone fractures. The key early signs of haemorrhagic shock are tachycardia and cutaneous vasoconstriction. In healthy young adults, hypotension occurs later and signifies a blood loss in excess of 1500–2000 ml. The initial fluid bolus is 1–2 litres for an adult and 20 ml/kg for a child. This is followed by blood transfusion. Fully cross-matched blood tests is preferable, but in an urgent situation type-specific (ABO and Rh matched blood) can be used. In lifethreatening hypotension, O negative packed cells can be used. The return of blood pressure and pulse to normal are encouraging signs. Restoration of urinary output to 0.5 ml/ kg/hr suggests that adequate renal perfusion has been restored.

Disability (neurological status)

The possibility of brain injury is assessed next. A decrease in a patient's level of consciousness may be due to a primary brain injury. This is assessed very quickly by examining the pupil size and reactivity and by assessing whether the patient is alert, responds to verbal stimuli, responds only to pain or is unresponsive. Unequal pupillary responses may be indicative of local trauma to the eye (traumatic mydriasis) or, more worryingly, an expanding intracranial haematoma. The Glasgow Coma

Scale (GCS) is a useful clinical method of monitoring the status of patients following head injury. This gives a score out of 15 based on the patient's best motor, verbal and eye responses. A score of 15 would indicate a fully alert, cooperative and comprehensible patient, and a score of 8 or less usually indicates the presence of serious cranial trauma. A decreased level of consciousness, particularly in the absence of any signs of external head injury, may represent inadequate cerebral oxygenation and perfusion, prompting a swift re-assessment of A, B and C.

Exposure/environment

To facilitate a thorough whole body examination, all clothing should be removed from a patient. Frequently this requires clothing to be cut off. This allows an adequate assessment of the spinal column, the posterior aspects of the limbs and the perineum. Once the appropriate examinations have been performed, it is important that the patient is covered with blankets to prevent hypothermia. Additional preventative measures include the warming of intravenous fluids and the maintenance of a warm environment in the resuscitation room.

It is vitally important that progress through the assessment and resuscitation sequence does not occur until problems of higher priority are appropriately dealt with. There is no point in trying to put on a pelvic external fixator if the patient cannot maintain his own airway. Equally, the condition of a traumatized patient is constantly changing and frequent reassessment is required to ensure there has been no deterioration. Maxillofacial surgeons should be an integral part of the trauma team for those patients where facial injuries are evident. This involvement is particularly relevant during the management of: the airway, hypovolaemia including facial bleeding, craniofacial injuries, in the assessment of the eyes.

If the patient's blood alcohol levels are high, they are unlikely to fall in the next 12-24 h and the following «hangover» will almost certainly be associated with vomiting. If the patient's consciousness level is of concern, intubation is necessary; also, in these circumstances a CT scan of the brain is indicated.

TOPIC: LOCAL AND GENERAL COMPLICATIONS OF MAXILLOFACIAL INJURES

Facial Nerve Injury

The facial nerve may be encountered in open approaches to the condylar neck and head. The marginal mandibular branch that innervates the depressor anguli oris, depressor labii inferioris, and lower fibers of the orbicularis oris and mentalis may be encountered during standard submandibular or Risdon, low cervical, retromandibular, and preauricular approaches. In standard approaches, when posterior to the facial artery, the marginal mandibular branch of the facial nerve will be roughly 1 cm below the inferior border of the mandible, whereas when crossing the facial artery, the marginal mandibular branch is usually above the inferior border of the mandible. However, some patients may show two, three, or even four branches of the marginal mandibular branch of the facial nerve between the angle of the mandible and the facial artery and vein. The facial vessels lie deep to the facial nerve; therefore, risk to the facial nerve is low once the plane of facial vessels is reached. It has been advocated to dissect to the level of the capsule of the submandibular gland to protect the marginal mandibular branch. Using this approach, the capsule of the submandibular gland is included in the flap that is elevated toward the mandibular border and contains the marginal mandibular branch.

Alternatively, a transmasseteric approach can be performed where an incision is made though the masseter, 10–20 mm above the mandibular basilar edge. In addition, endoscopically assisted fixation of condylar neck fractures, though time consuming, is associated with less morbidity to the facial nerve.

Trigeminal Nerve Injuries

Injuries to the all three branches of the trigeminal nerve can occur following maxillofacial trauma. The prevalence of inferior nerve injury following mandibular trauma approximates 58.5%. Causes of nerve injuries include soft tissue edema, secondary ischemia, transection and crush injuries of the nerve, and when the line of fracture occurs at a foramen and bony fragments impinge on the nerve. The latter cause can lead to permanent anesthesia, parathesia, and dysesthesia if not addressed in a timely manner.

Additionally, disruption of the inferior alveolar canal may cause bony proliferation and stenosis of the canal. Bagheri and colleagues described an algorithm for the approach to the patient with trauma-related trigeminal nerve injury. In the preoperative period, in patients with neurosensory dysfunction, exploration and repair of the nerve should be carried out. However, if microsurgical repair is not possible, open reduction of the fracture should be performed and neurosensory testing should be conducted for 3 months.

Patients with persistent neurosensory dysfunction after 3 months should be referred to a microneurosurgeon. Nerve exploration and repair should take place when there is no improvement after 3 months or if symptoms are not acceptable to the patient after fracture treatment.

Frontal sinus fractures

Frontal sinus injuries are relatively common occurrences, representing approximately 4–8% of all facial fractures. Motor vehicle accidents are the most common etiology, and the fact that a high degree of force is required to fracture the frontal bone (800–2,200 pounds) means that many of these patients will have concomitant injuries that require a multidisciplinary approach. Immediate or delayed complications, some of which can be life threatening, occur in 10–20% of the patients with frontal sinus fractures.

The goals in the treatment of frontal sinus injuries are to provide an aesthetic outcome, restore function, and prevent complications. It has never been completely clear, however, whether the frontal sinus is the culprit in the development of postoperative complications, or a victim of improper or ill-advised surgery. In either case, complications occur both in patients treated surgically as well as in those who are observed. Chuang and Dodson recently attempted to identify the frequency of serious complications of operated patients compared to patients who did not undergo surgery by applying the principles of evidence, based on the existing literature. A Medline search was conducted by identifying pertinent articles from 1980 to 2003 that were related to inflammatory complications associated with frontal sinus injuries. Excluding reviews and single case reports, serious inflammatory complications were reported in sufficient detail to estimate the frequency of such complications in only 25 studies. Study design in these papers was generally considered poor (level 4 evidence), and the inclusion and exclusion criteria were variable or unidentifiable. Despite numerous limitations, it was estimated that the rate of serious complications is approximately 9% (range 0% to 50% with the 95% confidence interval from 0% to 21%). Additionally, in an effort to estimate the rate of complications from untreated frontal sinus fractures, an attempt was made to extrapolate data from the craniofacial surgery literature by reviewing outcomes of procedures that often involve disruption of normal frontal sinus anatomy. They identified nine studies from which comparable data were available and determined the incidence of complications following nonoperative management of iatrogenic frontal sinus injuries to be approximately 3% (range 0% to 12% with a 95% confidence interval ranging from 0% to 14%). While a prospective study directly comparing nonsurgical treatment versus surgical treatment of frontal sinus fractures is neither feasible nor ethical, the current paper suggests that patients with less severe injuries can be safely observed with little risk of short-term complications and that more severely injured patients benefit from surgical repair with a relatively low risk of adverse short-term sequelae.

Infection/Sinusitis

Localized wound infection, hematoma, and/or seroma formation can occur in the immediate postoperative period. These complications can generally be prevented by utilizing suction drains for the first 2 to 3 postoperative days. If they do occur, surgical drainage and appropriate antibiotic therapy will usually resolve the problem without significant long term sequelae. On the other hand, if a postoperative infection extends through a fracture of the posterior wall of the frontal sinus or through rents in the dura, the result may be acute epidural abscess or brain abscess. Meningitis may also result, particularly in patients with severe injuries that involve the anterior and posterior table and who undergo craniotomy. Prompt recognition and treatment with antibiotics and neurosurgical intervention are necessary for successful outcomes.

Complications of Sinusitis

Mucocele and Mucopyocele

Chronic sinusitis that causes inflammation and scarring of the sinus ostia can result in mucocele formation. Mucoceles are found most often in the frontal or maxillary sinuses and are a feared complication of frontal sinus fractures. The danger lies primarily in their propensity to become infected (mucopyocele), which can lead to potential intracranial infection or brain abscess. Frontal mucoceles (or mucopyoceles) should be managed by osteoplastic frontal sinus obliteration or, in rare cases, cranialization (discussed later).

Mucocele formation refers to an expansile mucous-filled lesion of the sinus that occurs due to obstruction of the nasofrontal recess. Continued secretion of mucous causes expansion and an increase in pressure, which leads to osteolysis and devascularization of bone. Osteomyelitis may occur from the compressive forces, or the lesion may extend intracranially or involve the orbits. When the lesion becomes infected, the term "mucopyocele" is used. Once a mucocele becomes infected, it may quickly spread to involve the epidural space or cause a brain abscess. It may also lead to frontal bone osteomyelitis or to orbital cellulitis/abscess.

Diagnosis of a mucocele may be difficult in the postoperative setting, as they may occur long after the initial trauma and repair (1 to 25 years). Symptoms are often nonspecific. If the mucocele is confined to the frontal sinus, frontal headache is the most common presenting symptom. If the orbit is involved, the patient may develop diplopia, proptosis, and limitation in ocular motility. Periorbital cellulitis, with or without ocular symptoms, is a common presentation in the post-traumatic setting. CT imaging is the diagnostic study of choice, which allows for accurate assessment of the size and location of the lesion. Treatment of mucoceles is surgical. Generally this will involve either obliteration of the frontal sinus with autogenous grafting (fat, bone, cement), or cranialization. Care must be taken to ensure that all mucosa is removed, including any remnants of the invaginated mucosa within the foramina of Breshet. As mentioned previously, the nasofrontal duct should be sealed with a robust pericranial flap. If the orbital roof has been destroyed, it must be reconstructed using either autogenous bone or alloplastic materials.

Orbital Complications

Complications can occur as a result of either sinusitis or sinus surgery. The major complications of sinusitis involve the orbit or the intracranial structures. Orbital complications are common and are due to the close proximity of the paranasal sinuses and the thin lamina papyracea separating the ethmoids from the orbit. An additional factor involved in spread of infection to the orbits is related to vascular anatomy. The superior and inferior ophthalmic veins are valveless and allow communication to and from the nose, ethmoids, face, orbit, and cavernous sinus.

Orbital infection has been classified into five categories:

Group 1: Inflammatory edema, characterized by upper eyelid edema, normal extraocular movement, and normal vision

Group 2: Orbital cellulitis characterized by severe, nonsuppurative periorbital edema, often resulting in proptosis, chemosis, impaired extraocular muscle function, or visual impairment

Group 3: Subperiosteal abscess characterized by a collection of pus at the medial aspect of the orbit that causes downward globe displacement, impaired extraocular muscle function, and changes in visual acuity

Group 4: Orbital abscess characterized by an abscess within the orbit, resulting in severe proptosis and complete ophthalmoplegia and visual impairment, often leading to blindness

Group 5: Cavernous sinus thrombosis, which is infection of the cavernous sinus characterized by sepsis, orbital pain, chemosis, proptosis, and ophthalmoplegia.

Treatment for Groups 1 and 2 consists of parenteral antibiotics. Suppurative infections, such as Groups 3, 4, and 5, require urgent surgical drainage, most often via an external approach.

Frontal Osteomyelitis

Osteomyelitis of the frontal bone is occasionally seen as a complication of frontal sinusitis, mucocele or mucopyocele formation. When characterized by subperiosteal abscess and swelling, it is referred to as Pott's Puffy tumor. These entities can be associated with cortical vein thrombosis, epidural abscess, subdural empyema, and brain abscess. The cause of venous thrombosis is explained by venous drainage of the frontal sinus, which occurs through diploic veins, which communicate with the dural venous plexus.

Septic thrombi can potentially evolve from within the frontal sinus and propagate through this venous system. Treatment therefore is by aggressive surgical debridement of the affected bone and soft tissue as well as appropriate antibiotic therapy.

Meningitis

Meningitis generally is regarded as the most common intracranial complication of sinusitis. Diagnosis is made after examination of CSF obtained via lumbar puncture. CSF cultures are used to guide antibiotic therapy that generally consists of high-dose parenteral antibiotics with good CSF penetration.

Intracranial Abscess

Epidural, subdural, or brain abscess is the most feared complication of sinusitis, with mortality rates approaching 20% to 30%. Most abscesses occur in the frontal lobe and present with signs and symptoms such as headache, behavioral changes, fever, and sepsis. CT scan and laboratory tests are diagnostic. Treatment involves prompt neurosurgical consultation, craniotomy, and sinus drainage.

Cavernous Sinus Thrombosis

Cavernous sinus thrombosis results from retrograde spread of infection through the valveless veins of the face, sinuses, and orbit. The presentation is dramatic and is characterized by massive periorbital edema, ophthalmoplegia, proptosis, chemosis, and occasionally visual changes. CT scan may be suggestive of the diagnosis, but angiography is diagnostic. Treatment consists of high-dose parenteral antibiotics, heparinization, and sinus drainage. Mortality is 30% if isolated to the cavernous sinus and 80% with progression to the sagittal sinus.

Aesthetic Deformity

Contour deformities of the forehead may occur as a result of inaccurate reconstruction of the frontal bone or frontal bandeaux, or as a result of infection or debridement of necrotic tissue. If the orbit is deformed and not adequately reconstructed, problems with globe projection and/or vision can also occur. Treatment via secondary reconstruction of the forehead and/or orbital units is usually required.

Distant Complications

1. Hypertrophic scars: Hypertrophic scars are limited to the original scar borders. Treatment involves silicone sheeting, steroid injections, and/or dermabrasion (elevated scars). Scar revisions are performed 6–12 weeks after repair during maximum collagen remodeling. Small scars may be excised (after 6 months), while larger scars may require various soft tissuerearrangements such as Z-plasty, brokenline closure (W-plasty or geometric design repair), and local flaps (advancement, transposition, and rotation).

2. Keloid formation: Keloids extend beyond the original scar borders into the adjacent tissues. The incidence of keloids is increased in Fitzpatrick skin types III–VII (darker skin). Any excision or debulking of a keloid must be combined with other modalities to prevent recurrence. Steroid (triamcinolone) injections and silicone sheets may be used for this purpose.

3. Dyschromias: Minimized with avoidance of direct sunlight.

4. Depressed scars: Minimized with proper eversion of the wound edges during initial tissue reapproximation. Fat atrophy may contribute to the depression of scar tissues. Various implants and aesthetic skin fillers have been used, including alloderm, collagen, fat, and hyaluronic acid.

Key Points

1. All wounds are considered contaminated and must be thoroughly and meticulously irrigated and debrided. Foreign bodies (dirt, glass, and asphalt) not debrided will lead to wound infection, wound dehiscence, and flap necrosis.

2. All complex soft tissue lacerations should be explored to rule out injury to underlying vital structures (neural, vascular, canalicular, and/or ductal injury) prior to closure.

3. The scalp is highly vascularized and can be associated with significant blood loss over a short period of time. The scalp should be closed in a layered fashion to eliminate potential dead space.

4. Signs of lacrimal system dysfunction include persistent epiphora, conjunctivitis, and dacryocystitis. Lacrimal system damage is tested for by utilizing a dacryocystogram and a Jones I/Jones II test. The lacrimal drainage system can be repaired by establishing a new drainage system via a dacryocystorhinostomy.

5. Avulsive injuries to the eyelids up to 25% can be closed primarily. Avulsive injuries to the eyelids of 25% or greater are repaired with tissue grafts (postauricular tissue) or local flaps.

6. An intranasal examination is performed in order to identify septal hematoma formation or avulsion with exposure of the underlying cartilage. Nasal hematomas present as reddish-bluish elevations of the nasal septal soft tissue and should be drained immediately with an incision parallel to the nasal floor. Packings, Doyle splints, or mattress sutures may be used after drainage to prevent the reformation of the hematoma.

7. All ear lacerations should have a complete otoscopic examination to evaluate the tympanic membranes (rupture and hematotympanum), lacerations involving the external auditory canal, and foreign bodies. External ear hematomas should be drained, and a pressure dressing placed. The ear has a tremendous vascular supply and can remain perfused with a small pedicle. Small areas of exposed cartilage are managed with antibiotic impregnated dressings. Large areas of exposed cartilage are managed with skin grafts.

8. The reestablishment of the vermillion border is the key step in repairing lip lacerations. The closure of full-thickness lip lacerations begins from the inside (mucosa) out (skin). Avulsive injuries to the lips of up to 25% can be primarily closed. Defects greater than 25% will require local flaps to minimize microstomia.

9. Neck wounds are divided into three zones. Unstable patients require urgent explorations with general anesthesia. Stable patients require CT angiogram and possible esophagography depending on the zone and depth of injury.

TESTS AND TASKS

TOPIC: DAMAGE OF THE SOFT TISSUES OF THE MAXILLOFACIAL AREA

Tests

1. Outcomes of hematomas with injures of maxillofacial region:
   1. Suppuration of wound;
   2. Complete resorption;
   3. Incomplete resorption;
   4. Encapsulation;
   5. Repeated bleeding.
2. The wound is:
   1. Solution of continuity of the surface layers of the skin;
   2. Damage to soft tissues with solution of continuity of the integument of the body and possible damage to the underlying tissues
   3. Damage of soft tissues without solution of continuity of the integument of the body
3. Types of wound healing:
   1. Primary wound healing;
   2. Secondary wound healing;
   3. Healing by scab;
   4. Superficial.
4. A bruise is damage of soft tissue structures caused by:
   1. Blunt instrument;
   2. With solution of continuity of the skin;
   3. Without solution of continuity of the skin.
5. There are the following types of wounds:
   1. Bruised;
   2. Cut;
   3. Chopped;
   4. Chopped-cut;
   5. All of the above.
6. Signs of a wound:
   1. Bleeding;
   2. Infection;
   3. Hiatus;
   4. Pain;
   5. Disturbince of functions
7. New hematoma has color:
   1. Purple-blue;
   2. Green;

3. Yellow.

8. Possible type of ecchymoma:

1. Forming a cavity;
2. Without cavity formation;
3. With suppuration of surrounding tissues.

9. Superficial mechanical damage of the skin area is called:

1. Contusion;
2. Abrasion;
3. Wound.

Answers: 1 – 1,2,4; 2 – 2; 3 – 1,2,3; 4 – 3; 4 – 5; 6 – 1,2,3,4,5; 7 – 1; 8 – 1,2; 9 – 2.

Tasks

Task 1. Patient B. was injured in everyday life as a result of falling on the glass, delivered to the emergency room. On the side surface of the cheek on the left side there is a linear wound with smooth edges measuring 4 cm long and about 1 cm deep, there is bleeding from the wound.

Make a diagnosis. Describe the steps to help the patient.

Task 2. Patient S. got a face injury in a fight. Describe what tactics of the doctor at the stages of diagnosis?

Task 3. Two men, in a state of intoxication, staged a fight, in which one struck the second with 3 stab wounds, presumably with a nail, 2 in the submandibular region, and 1 in the cheek region on the right.

Objectively: in the area of the cheek there is a point inlet wound hole with a diameter of about 0.3 cm, blood from the wound. When sensing wounds is determined by the message with the oral cavity. On the mucous membrane of the cheeks there is a round wound with a diameter of about 0.3 cm. In the submandibular region, there are 2 entrance wound holes with a diameter of about 0.3 cm, which blindly end in soft tissues. Wound channels about 2cm long.

Make a diagnosis. Describe the steps to help the patient.

Task 4. Patient S., 28 years old, was injured in a sports training session. Consciousness did not lose, nausea, vomiting was not. Objectively: in the right infraorbital region, edema, hematoma of soft tissues. Hematoma is the eyelid of the right eye. Paresthesia of the skin of the infraorbital region, upper lip, wing of the nose on the right. Clinical and radiological data for fractures of the face is not.

Make a diagnosis. Make a treatment plan.

Task 5. The man, intoxicated, slipped and fell on the bench. Dizziness, nausea, vomiting is not observed. In the infraorbital region, on the left, there is swelling, tenderness on palpation. Hematoma of dark blue color with foci of green color on the periphery. Make a diagnosis.

TOPPIC: SURGICAL TREATMENT OF INJURES OF SOFT TISSUES. PREVENTION OF TETANUS, CLOSTRIDIAL INFECTION AND RABIES

Tests

1. At what wounds is it necessary to vaccinate against rabies?
    1. Chopped;
    2. Cut;
    3. Bitten;
    4. Chopped.
2. The second period of the wound process is called:
    1. Regeneration phase;
    2. Phase of inflammation;
    3. Phase of epithelialization.
3. The ends of the ligatures on the skin should be no more than:
    1.0.5-0.8cm;
    2.1.5-2 cm;
    3.3-4 cm.
4. The absorbable suture filaments are:
    1. Chromed collagen;
    2. Shelk braided;
    3. Capron.
5. Primary surgical treatment of a wound does not necessarily involve:
    1. Mechanical pre-treatment;
    2. Antiseptic pre-treatment;
    3. Pre-treatment of the edges of bone fragments;
    4. Layered closure;
    5. Carving (of the edges of the wound).
6. Sequence of suturing with a wound penetrating the oral cavity:
    1. Mucous membrane, muscles, skin;
    2. Muscles, mucous membrane, skin;
    3. Muscle, skin, mucous membrane.
7. Early primary surgical debridement is carried out during:
    1. 12 hours;
    2. 24 hours;
    3. 48 hours;
    4. 72 hours.
8. Late primary surgical debridement is performed after:
    1. 12 hours;
    2. 24 hours;
    3. 48 hours;
    4. 72 hours.

9. Delayed primary surgical debridement is carried out during:
   1. 12-24 hours;
   2. 24-72 hours;
   3. 24-48 hours;
   4. 36-72 hours.

10. Primary surgical debridement of facial wounds can be performed:
   1. No later than 24 hours after the injury;
   2. No later than 48 hours;
   3. No later than 3 days;
   4. At any time.

11. The primary-delayed suture is used:
   1. No later than 24 hours;
   2. No later than 48 hours;
   3. Before the appearance of granulations;
   4. On a granulating wound.

12. Secondary suture is used:
   1. No later than 24 hours;
   2. No later than 48 hours;
   3. Until granulation;
   4. On the granulated wound.

Answers: 1 – 3; 2 – 1; 3 – 1; 4 – 1; 5 – 3,4,5; 6 – 1; 7 – 2; 8 – 3; 9 – 3; 10 – 2; 11 – 2; 12 – 4.

Tasks

Task 1. A patient came to the clinic with a wound in the left buccal region, irregular in shape, up to 3 cm long. The wound is blunt-edged, the depth to the muscle layer, the wound has serous and serum crusts, the edges of the wound are slightly hyperemic, edematous. 2.5 days have passed since the injury.

Make a diagnosis. Select the type of surgical treatment. Make a treatment plan.

Task 2. The patient with the presence of a wound in the left buccal region, a week after PSD, suppuration of the wound occurred, and a week later bleeding began. What causes bleeding? Type of bleeding.

Task 3. The patient with a bitten wound of the face was hospitalized in the department of maxillofacial surgery. According to the patient, he received a wound as a result of an attack by a stray dog. Make a diagnosis. Schedule a treatment plan.

Task 4. A patient with a trauma of the maxillofacial area (transport trauma) was taken to the department. The patient is conscious. From an anamnesis it is found out, that at the moment of collision he has hit about a back of sitting of the car. At the moment of impact, consciousness did not lose, there wasn't vomiting, nausea was also not observed. There was bleeding from the mouth within 30 minutes of the injury. On examination, the edema of the upper lip is determined, on the skin of which there are minor abrasions. On the red border of the lips has a lot of bloody crusts. In the oral cavity, the joining of the teeth is not disturbed.

Make a diagnosis. Outline a treatment and prevention plan for complications.

Task 5. A patient came to the clinic with complaints of swelling of the upper lip, pain in this area. From an anamnesis it is found out, that about 16 hours ago has received blow a blunt subject in area of labiums. At the moment of impact, he briefly lost consciousness. Vomiting and nausea was not. For help did not apply. When viewed from the upper lip swollen, on the mucous membrane of the upper lip from the vestibular side there are many small bruises. The closure of the teeth is not broken.

Make a diagnosis.

TOPIC: DISLOCATION AND FRACTURES OF THE TEETH, FRACTURES OF THE ALVEOLAR PROCESS

Tests

1. Tooth dislocation can be:
   1. Partial;
   2. Incomplete;
   3. Complete;
   4. Impacted;
   5. With tooth extraction.
2. Fractures of the roots of the teeth are:
   1. Transverse;
   2. Longitudinal;
   3. Cruciform;
   4. Oblique.
3. Where are tooth fractures located more often?
   1. On the upper jaw in the anterior region;
   2. On the lower jaw in the anterior region;
   3. On the upper jaw in the lateral division;
   4. On the lower jaw in the lateral division.
4. Where is the incisor root fracture more common?
   1. In the area of the top of the tooth;
   2. Between the middle and apical part;
   3. In the middle of the root of the tooth;
   4. In the neck of the tooth.
5. On a radiograph with a complete dislocation of the tooth is determined by:
   1. Free well
   2. Expansion of the periodontal gap in the apex
   3. Absence of periodontal gap in the apex
   4. The absence of the periodontal gap along the entire length
6. On the radiograph with incomplete dislocation of the tooth it is determined:
   1. Free alveolar socket;
   2. Expansion of the periodontal ligament space;
   3. Absence of periodontal ligament space;
   4. Expansion of the periodontal gap in the apex.
7. On the radiograph of impacted dislocation of the tooth is determined:
   1. Free alveolar socket;
   2. Expansion of the periodontal gap in the apex;
   3. Absence of periodontal gap in the apex;
   4. The absence of the periodontal gap along the entire length.

8. In case of incomplete traumatic dislocation, the apposition of the dislocated tooth is carried out:

1. Using fingers, while they are located only on the dislocated tooth;
2. Using fingers, while they are located on contiguous teeth and capture the dental arch;
3. Using forceps, grabbing the root part of the tooth;
4. With the help of special devices.

9. Is it necessary to conduct anesthesia during reposition of an dislocation of the tooth, if damage to its neurovascular bundle is diagnosed?

1. Yes;
2. No.

10. In the case of complete traumatic dislocation of the tooth, accompanied by a comminuted fracture of the walls of its alveolar socket with the presence of a bone defect, it is necessary:

1. Replantation is not carried out, the tooth socket is suturing;
2. After pre-treatment of the alveolar socket, the pre-pulped tooth is replanted with its immobilization;
3. After treatment of the alveolar socket, the tooth is replanted without pre-filling the canal with the obligatory subsequent EPT.

Answers: 1 – 2,3,4; 2 – 1,2,4; 3 – 1; 4 – 2; 5 – 1; 6 – 2; 7 – 3; 8 – 1; 9 –1; 10 – 1.

Tasks

Task 1. The child is 4 years old. Complains of pain when biting and chewing food. In the words of his mother, he fell down yesterday and hit his face. Loss of consciousness was not. Objectively: the child is active. In the oral cavity: there is an elongation of the crown 5.1 relative to the teeth 5.2, 6.1, 6.2. The mobility of the tooth 5.1 II degree, the tooth is not changed in color, percussion 5.1 painful, the gums in the area of the tooth 5.1 are hyperemic. On the radiography roots of 5.1, 6.1 are formed. There is an expansion of the periodontal gap in the area of the root apex of 5.1.

Make a diagnosis. Make a treatment plan.

Task 2. Parents brought a 2-year-old child, who few hours ago injured 6.1 tooth, falling from a swing. Consciousness did not lose, nausea, vomiting was not. Objective: swelling of the soft tissues of the upper lip. On the red border of the upper lip there is an abrasion, on the mucous membrane of the upper lip are hemorrhage. The 6.1 tooth crown is lose, there is a blood clot in the alveolar socket.

What types of injuries are possible? What additional research methods are needed to make a final diagnosis? Make a treatment plan.

Task 3. Child 1.2 months. According to thewords of his mother, the child fell and hit his face 1 hour ago, the central teeth became shorter than the adjacent teeth. Objectively: swelling of the upper lip, abrasion of the chin skin. Dental crowns 5.1, 6.1 shorter than dental crowns 5.2, 6.2.

Formulate a diagnosis. Make a treatment plan.

Task 4. The child was 1 year and 8 months old, a few hours ago, fell from a chair, did not lose consciousness, was nauseous, did not have vomiting. Objectively: the face is symmetrical. In the oral cavity: in the teeth 7.1, 8.1 there are no medial corners of the crowns, defect of the crowns within the enamel.

Formulate a diagnosis. Make a treatment plan.

Task 5. The father brought a boy of 8 years old, who broke a tooth 1.1 at school in a gym class. The child complains of pain when touching the tooth. Objectively: the crown of tooth 1.1 is broken off by 1/2, the fracture line passes horizontally through the cavity of the tooth. The pulp is naked, red, sharply painful when probing. Percussion of the tooth is painful.

Formulate a diagnosis. What factors will influence the choice of treatment for tooth 1.1?

TOPIC: BURNS AND FROSTBITE OF THE FACE

Tests

1. Periods of burn disease:
    1. Burn shock;
    2. Acute burn toxemia;
    3. Burn septicotomy;
    4. Reconvalescence.
2. The second degree of frostbiteis characterized by:
    1. Tissue necrosis is not present;
    2. The healing takes place without granulation and scarring;
    3. Necrosis of superficial skin areas up to the malpigian layer;
    4. Healing and sclerostenosis.
3. Name phases of the development of burn disease:
    1. Burn shock;
    2. The period of convalescence;
    3. Acute burn toxemia;
    4. Phase septicotoxemia;
    5. All the answers are correct;
4. What is the treatment of the 1st degree burn:
    1. Gently opening of the bubbles in aseptic conditions;
    2. Local antiseptic pre-treatment;
    3. Physiotherapeutic procedures;
    4. Local using of cold;
    5. Superficial treatment using antibacterial ointments.
5. When frostbite, what degree of skin is treated with alcohol and opening the bubbles:
    1. I;
    2. II;
    3. IV.
6. To determine the burn area, the following rules apply:
    1. Palms;
    2. Sixes;
    3. Nines.
7. Burn disease occurs as a result of exposure:
    1. Heat;
    2. Chemical factors;
    3. Physical factors.
8. Is the frolement of the damaged area using during frostbite:
    1. Yes;
    2. No.
9. Types of burns depending on the traumatic factor are:

1. Thermal;
2. Chemical;
3. Beam;
4. All the answers are correct.

10. How many degrees of frostbite are differentiated:
1. V;
2. VI;
3. IV;
4.III.

11. In which areas of the person are the deepest burns formed:
1. Eyelids;
2. Superciliary arches;
3. Nose;
4. Cheeks;
5. Ears;
6. Lips;
7. Zygomatic area;
8. The chin.

12. At what stage of frostbite the skin has a dark-blue or purple-red color:
1. Preactive;
2. Reactive.

Answers: 1 – 1,2,3,4; 2 – 2,3; 3 – 5; 4 – 2.4.5; 5 – 2; 6 – 1.3; 7 – 1; 8 – 2; 9 – 4; 10 – 4; 11 – 2,3,5,6,7,8; 12 – 2.

Tasks

Task 1. The patient is 56 years old, is taken to the hospital admission department with the following clinical picture: severe pain, swelling and cyanotic tissue, paresthesia of tissues, skin of purplish red color. There are small bubbles.

Make a diagnosis. . Define a treatment plan.

Task 2. The patient turned to the hospital with a lesion of the skin of the face and neck. In the region of cheeks, chin, nose, zygomatic bone, there are bubbles filled with serous fluid. The skin is hyperemic, edematous, painful during palpation.

Make a diagnosis. Define a treatment plan.

Task 3. Patient B, delivered by ambulance to VOKB with damage to the lower limbs, ears of the nose, cheeks. From an anamnesis of 10 years suffers from diabetes; a few days was on the street at an ambient temperature of 50C. Objectively: the skin of the face is of a cyanotic color, especially expressed in the region of the nose, cheeks and ears. There are bubbles in these areas filled with a transparent yellowish liquid.

Make a diagnosis. Define a treatment plan.

Task 4. The patient M is 26 years old, is taken to the burn department with burns of head, face, neck. Objectively: the skin of the head, face and neck are hyperemic, swollen, painful. There are no necrosis and blistering.

Make a diagnosis. Define a treatment plan.

TOPIC: COMBINED TRAUMA

Tests

1. ABC's of trauma management
   1. Secure an Airway,
   2. Make sure the patient is Breathing and ventilating properly,
   3. Ensure adequate Circulation by stopping bleeding and providing fluid replacement,
   4. Ensure that no C-spine fracture is present
2. Quickest and easiest method of securing the airway
   1. Endotracheal intubation
   2. Ventilation
   3. Inhalation
3. When will endotracheal intubation not work?
   1. If the patient has a broken leg
   2. If the patient has a broken nose
   3. If the patient has a broken arm
   4. If the patient has a broken neck and massive trauma with distortion of landmarks and bleeding
4. When would a cricothyrotomy not be performed?
   1. If there is concern over a fractured maxilla
   2. If there is concern over a fractured larynx (widened thyroid cartilage, subcutaneous air, neck bruising, hoarseness, coughing up blood)
   3. If there is concern over a fractured maxilla
   4. There is no correct answer
5. What else can be performed if an oral intubation cannot occur?
   1. Fiberoptic nasotracheal intubation
   2. Intubation by tracheostomy
   3. Nasotracheal intubation
   4. There is no correct answer
6. What can hemotympanum signify?
   1. Temporal bone fracture
   2. Occipital bone fracture
   3. Mandibular fracture
7. ABCDE:
   1. Airway, Breathing, Circulation, Disability, Exposure/Environment
   2. Airway, Breathing, Circulation, Dislocation, Exposure/Environment
   3. Airway, Break, Circulation, Dislocation, Exposure/Environment
   4. Airway, Breathing, Circulation, Dislocation, Exit
8. Causes of patient mortality due to maxillofacial trauma
   1. Airway compromise
   2. Exsanguination

3. Intracranial/c-spine injury
4. Meningitis
5. Oropharyngeal infections

9. What can cause cerebrospinal fluid rhinorrhea?
1. Temporal bone fracture
2. Basal skull fracture
3. Mandibular fracture
4. Nose

Answers: 1 – 1-4; 2 – 1; 3 – 4; 4 – 2; 5 – 1-3; 6 – 1; 7 – 1; 8 – 1-5; 9 – 1-2.

Tasks

Tasks 1. Which intubation is better: nasotracheal or orotracheal?

Tasks 2. Trimodal Death Distribution are: Death due to lacerations of brain, brain stem, high SC, heart, aorta and other large bv – 1,

Subdural epidural hematomas, hemopneumothorax, ruptured spleen, lacterated liver, pelvis – 2,

Sepsis and multiple organ failure – 3. Is it true?

Tasks 3. Do the velocity of missile, caliber, and distance from weapon to wounded in penetrating trauma matter?

Tasks 4. Can dentoalveolar trauma include the adjacent soft tissue or is it only injuries that involve teeth and alveolar portion of maxilla/mandible?

Tasks 5. The nerve that is commonly not evaluated upon initial presentation, but whose management depends greatly on the examination at the time of presentation is the _______ nerve.

TOPIC: MANDIBULAR FRACTURES

Tests

1. What are the clinical signs and symptoms of a mandible fracture?
   1. Pain, swelling, limitation of function
   2. Occlusal derangement
   3. Numbness of lower lip
   4. Loose/mobile teeth
   5. Bleeding
   6. Facial asymmetry
   7. Deviation to opposite side
   8. No clinical signs
2. What are the causes of Mandibular fracture?
   1. Trauma
   2. Falls
   3. Sporting injury
   4. Pathology
   5. Iatrogenic
   6. Reconstructive surgery
   7. Industrial injury
   8. All answers are true
3. How can involvement of the surrounding tissue be classified?
   1. Simple
   2. Compound
   3. Displaced
4. What are the possible sites of Mandibular fracture?
   1. Angle
   2. Subcondylar
   3. Parasymphyseal
   4. Body
   5. Ramus
   6. Coronoid
   7. Condylar fracture
   8. Alveolar process
   9. Pterygoid process
   10. floor
5. What factors cause displacement of Mandibular fractures?
   1. Direction of the fracture line
   2. Opposing occlusion
   3. Magnitude of force
   4. Mechanism of injury

5. Intact soft tissue (reduce)
6. age and sex of the patients

6. What radiographic views may be requested for visualisation of Mandibular fractures?

1. Panoramic
2. Occlusal
3. Lateral oblique
4. CT scans
5. Axial
6. Semiaxial

7. What is the treatment of mandible fractures?

1. Control pain and infection
2. Reduction and fixation
3. Non-steroidal anti-inflammatory drugs
4. Sedation therapy

8. What re the options for fixation?

1. Closed reduction and fixation (Intermaxillary *fixation* - IMF)
2. Open reduction internal fixation (ORIF)
3. Fixation by A. Roth
4. All answers are true

9. What are the approaches to open reduction internal fixation?

1. Retromandibular approach
2. Roth approach
3. Preauricular approach
4. Bi-coronal flap
5. Endoscopic reduction and fixation

10. What are the complications of Mandibular fracture?

1. Trismus
2. Osteoarthritis
3. Subluxation
4. Late trismus
5. Deviation to affected side
6. Ankylosis and asymmetry
7. Open bite
8. Exophtalm

11. General criteria for extraction of a tooth in the line of fracture

1. Significant mobility
2. Root exposure in a markedly distracted fracture
3. Interferes with reduction or fixation
4. Gross dental pathology
5. All answers are false

12. What type of fracture are prophylactic antibiotics used for?
  1. Compound fractures
  2. Closed
  3. All answers are false

13. What is closed reduction?
  1. Direct interdental wiring
  2. Indirect wiring
  3. Arch bars
  4. Gunning-type splints/lingual splits
  5. External pin fixation
  6. Open reduction internal fixation

14. Open reduction – advantages:
  1. Early return to function
  2. Promotes primary bone healing
  3. Bone fragments reapproximated via direct visualization
  4. Scarring

15. Open reduction – disadvantages:
  1. Early return to function
  2. More operating room time
  3. Greater operator skill
  4. Scarring

16. Classification of condylar fractures:
  1. Extracapsular
  2. Intracapsular
  3. Subcondylar
  4. Condylar head
  5. All answers are false

Answers: 1 – 1-7; 2 – 8; 3 – 1,2; 4 – 1-8; 5 – 1-5; 6 – 1-4; 7 – 1,2; 8 – 1,2; 9 – 1,3,4,5; 10 -1-7; 11 – 1-4; 12 – 1; 13 – 1-5; 14 – 1-3; 15 – 2,3,4; 16 – 1-4.

Tasks

Task 1. What type of radiographs are taken for mandibular fractures?

Task 2. Which muscle is particularly important in condylar fractures? Why?

Task 3. For thin, atrophic, edentulous mandible what technique can be used if you do not get it back to how it was before?

Task 4. What size plate do you need to use for the mandible? What about elsewhere?

Task 5. Conservative management of a fractured mandible. When is it used? What can it involve?

Task 6. How long is the mandible splinted for before being able to function normally?

Task 7. What are the 3 advantages of the vestibular approach to the mandible?

TOPIC: MAXILLARY FRACTURES

Tests

1. Characteristics of maxillary fractures:
    1. Surgical intervention less common
    2. Need to preserve dorsal periosteum
    3. Preserve oral palatine mucoperiosteum
    4. Potential for oronasal fistula foramtion
2. What options do you have for stablization of maxillary fractures?
    1. Tape muzzle
    2. Pins and wires
    3. Plates
    4. External fixators
    5. Partial maxillectomy
3. Maxillary fractures
    1. LeFort I, II, III
    2. Angular
    3. Mental
    4. Central
4. Complet facial skeleton separation from the skull
    1. Lefort I
    2. Lefort II
    3. Lefort III
5. What is the goal of fixing facial fractures
    1. Resorbtion
    2. Restoraiton of normal function & appearance
    3. Surgery
6. Maxillary fractures. Signs and symptoms:
    1. Loss of bony stability
    2. Difficulty in swallowing
    3. Edema
    4. Blood
    5. Traumatic debris
7. Maxillary fractures. Treatment:
    1. Secure airway
    2. Frequent suctioning
    3. Elevate HOB
    4. Apply ice
    5. Ophthalmology, neurologic consults for Le Fort II, III fxs
    6. Surgery

8. Fixing maxillary fractures
   1. Palatine sutures or wires
   2. Avoid plating if possible
   3. Ex-fix with acrylic connecting columns

Answers: 1 – 1-4; 2 – 1-5; 3 – 1,2; 4 – 1-3; 5 – 1-3; 6 – 1-6; 7 – 1-6; 8 – 1-3.

Tasks

Task 1. Le Fort II fractures are also known as ______ fractures. It involves fracture of the nasal bone, and frontal process of the maxilla. IT can extend laterally to lacrimal bones and floor of the orbit and posteriorly to ptyergoid plates. these peole have "dish face" where midface is sunken in.

Task 2. Le Fort III is also known as ______ ______ where there complete separation of the middle third of the facial skeleton from the cranium. There is a more exxagerated dish face deformity and an anterior ______ and CSF leakage.

Task 3. A _______ fracture occurs when a bone bends and cracks, instead of breaking completely into separate pieces. Can be seen in contrecoup fractures.

Task 4. How often do you repair maxillary fractures? When do you replace maxillary fractures?

Task 5. This type of fracture is among the most severe injuries involving the face. signs and symptoms includes: mobility or displacement of the palate, mobility of the nose in association with the palate, epistaxis, mobility or displacement of the entire middle third of the face.

Task 6. This classification is a low transverse fracture of the maxilla involving the palate, its characterized by mobility of the maxillary dental arch and palate, fracture through: a) aveolar process, b)palate, and c) sphenoid bone, including the pterygoid plates.

TOPIC: ZYGOMATIC COMPLEX FRACTURE

Tests

1. Because of what is it possible to limit the opening of the mouth at the turn of the zygomatic arch with displacement?
   1. Injury of the pterygoid muscles
   2. Pressure of the displaced fragment of the zygomatic arch on the coronoid process and damage to the fibers of the temporal muscle
   3. Hematoma and traumatic swelling of the soft tissues of the zygomatic region
   4. Injury of the chewing muscle
   5. Hemarthrosis of the temporomandibular joint

2. What method of reposition is most suitable for fractures of the zygomatic arch with displacement?
   1. Osteosynthesis with miniplates
   2. Reposition by Limberg's sponsor
   3. Osteosynthesis with Kirchner spokes
   4. Bloody reposition of fragments from the maxillary sinus

3. Specify the mechanism of development of traumatic neuritis of the second branch of the trigeminal nerve with a fracture of the zygomatic bone:
   1. Compression of the infraorbital nerve with bone fragments
   2. Development of hematoma of the zygomatic region
   3. Rupture or compression of the maxillary nerve in the pterygopalatine fossa
   4. Rupture of the nerve trunk in the infraorbital canal
   5. Hemosinus

4. What diseases of the eyes can be observed when there is a fracture of the zygomatic bone?
   1. Limiting the mobility of the eyeball
   2. Exophthalmos
   3. Diplopia
   4. Scleral hematoma
   5. Reduced visual acuity

5. The closed method of repositioning the zygomatic bone by the Limberg rebel is used with:
   1. Fracture of the zygomatic bone without displacement
   2. Fracture of the zygomatic bone with displacement, without hemosinus and comminuted fracture of the anterior wall of the maxillary sinus
   3. Fracture of the zygomatic bone with signs of hemosinus of the maxillary sinus

4. Comminuted fracture of the zygomatic bone

6. The method of bloody reposition of the zygomatic bone is used for:

1. Fracture of the zygomatic bone with displacement, without hemosinus and comminuted fracture of the maxillary sinus
2. Fracture of the zygomatic bone without displacement
3. Fracture of the zygomatic bone with signs of hemosinus of the maxillary sinus
4. Comminuted fracture of the zygomatic bone
5. A fracture of the zygomatic bone without displacement with a fracture of the zygomatic arch with displacement

7. Step deformity along the lower-eyed margin and zygoalveolar ridge is characteristic of:

1. Fracture of the zygomatic arch with displacement
2. Fracture of the zygomatic bone with displacement
3. Fracture of the alveolar process of the upper jaw
4. Fracture of the upper jaw by Le Fort I

8. Step deformity along the lower orbital margin, the outer edge of the orbit and the cheekbone-alveolar crest is characteristic of:

1. Fracture of the upper jaw of Le Fort II
2. Fracture of the zygomatic bone with displacement
3. Fracture of the upper jaw by Le Fort I
4. Fracture of the temporal bone

9. What method of surgical treatment is used for fractures of the bones of the nose with displacement?

1. Reposition of the bones of the nose by bilateral anterior tamponade of the nose
2. Reposition of the bones of the nose by bilateral posterior tamponade of the nose
3. Intranasal instrumental reposition of the bones of the nose with bilateral anterior tamponade
4. Intranasal instrumental reposition of the nasal bones with posterior tamponade
5. Finger reposition of the bones of the nose from the skin with the imposition of | adhesive bandage

10. Fractures of the zygomatic bone and the arc of the total number of fractures of the facial skull are:

1. 5-8%
2. 10-12%
3. 15-20%

11. A characteristic clinical sign of a jaw-maxillary fracture is:

1. Sticking of the middle third of the face on the side of damage

2. Shortening of the middle third of the face
3. Malocclusion

12. A characteristic clinical sign of the jaw-maxillary fracture is a violation of surface sensitivity in the branching zone:

1. The nasal palatine nerve
2. The supraorbital nerve
3. Infraorbital nerve

Answers: 1 – 2; 2 – 2; 3 – 4,1,4; 4 – 2,3,4; 5– 2; 6 – 3,4; 7 – 2,4; 8 – 1,2; 9 – 3; 10 – 2; 11 – 1; 12 –3.

Tasks

Task 1. The patient was diagnosed with an isolated fracture of the nasal bones. Where should he get treatment?

Task 2. A patient admitted to the hospital after a facial injury, was X-rayed. On radiographs of the paranasal sinuses and zygomatic bones in the straight naso-mental (semi-axial) and axial projections, the integrity of the bone tissue in the junction of the zygomatic bone with other bones of the facial and cerebral skull, as well as the darkening of the maxillary sinus from the side of the injury, is determined. Make a preliminary diagnosis.

Task 3. The patient was diagnosed with a fracture of the zygomatic bone. According to x-ray studies of the displacement of fragments and dysfunction there. Prescribe treatment.

Task 4. A patient with a diagnosis of impacted fracture of the bones of the nose was assigned a CT scan of the skull. Explain the reason for the need for this study?

Task 5. The patient after the injury turned into a hospital. Complaints of pain in the place of injury, restriction of mouth opening, marks double vision.

Objectively: there is swelling and hematoma of the soft tissues in the zygomatic region on the right, swelling of the eyelids and hemorrhage into the tissue around the right eye, the eye slit is narrowed. The presence of bleeding from the right nose of the nostrils. Palpation of the zygomatic region is painful, the symptom of the “step” is determined by the lower orbital margin, the upper-outer edge of the orbit, along the zygomatic arch and the zygomatic alveolar crest of the right half of the face. Make a diagnosis, name the missing study.

TOPIC: LOCAL AND GENERAL COMPLICATIONS OF INJURES OF MAXILLOFACIAL REGION

Tests

1. What caused the development of dislocation asphyxia at the fracture of the lower jaw?
   1. The displacement of the lower jaw fragments
   2. Tongue-swallowing
   3. The penetration of blood clots in the upper respiratory tract
   4. Distal chin offset along with the tongue
2. What caused the development of obstructive asphyxia for fractures of the facial bones?
   1. Tongue-swallowing
   2. Swelling of the larynx
   3. The ingress of blood clots or vomit in the upper respiratory tract
   4. Ingress of foreign bodies (teeth, dentures, bone fragments), into the upper respiratory tract
3. What caused the development of stenotic asphyxia in fractures of the facial bones?
   1. Compression of the trachea with hematoma
   2. The ingress of blood clots or vomit in the upper respiratory tract
   3. The displacement of bone fragments
   4. Traumatic edema of the larynx
4. What caused valve asphyxia in fractures of the facial bones?
   1. Compression of the trachea with hematoma
   2. Swelling of the larynx
   3. Closing the entrance to the larynx on the inhale grafts of the damaged mucous membrane
   4. The displacement of bone fragments
5. What is the cause of aspiration asphyxia in fractures of the facial bones?
   1. The ingress of blood clots in the upper respiratory tract
   2. Tracheal hematoma compression
   3. Swelling of the larynx
   4. Closure of the entrance to the larynx by inhalation with flaps of damaged mucous membrane
   5. Vomit entering the upper respiratory tract
6. What is the provision of first aid for dislocation asphyxia?
   1. The removal of foreign bodies and blood clots from the oropharynx
   2. The residence and fixation of the tongue
   3. The conduct of tracheotomy
   4. Filing hanging patches of the oral mucosa
   5. The introduction of the duct

7. What is the provision of medical care for obstructive asphyxia?
   1. Stitching and fixing the tongue
   2. The removal of foreign bodies from the oropharynx
   3. Filing hanging patches of the oral mucosa
   4. The conduct of tracheostomy
   5. The conduct of conicotomy
8. What is the provision of medical care for stenotic asphyxia?
   1. The immobilization of fragments of the lower jaw
   2. Tracheostomy
   3. The removal of foreign bodies and blood clots from the oropharynx
   4. Filing hanging patches of the oral mucosa
   5. The conduct of conicotomy

   Answers: 1 – 1, 2; 2 – 4; 3 – 1,4; 4 – 3; 5 – 1,4,5; 6 – 2,4,5; 7 – 2,5; 8 – 4,5.

Tasks

Task 1. The patient, 31 years old, came to the clinic with complaints about the impossibility of closing the teeth, asymmetry of the face, pain in the temporomandibular joint on both sides, difficulty in chewing food. From the anamnesis, it was revealed that six months ago he had received an industrial injury to his face when working on a building in a remote area, he could not get medical help, he was treated on his own: compresses, antibiotics. He noted the inability to take solid food due to pain in the lower jaw body on the right, while the paramaxillary soft tissues were swollen, painful, and there was an extensive hematoma. He came to the doctor for the first time six months after the injury. From past diseases indicates childhood infections, acute respiratory infections. When a local examination is observed asymmetry of the face due to deformation of the lower jaw in the area of the body of the mandible on the right was found. When palpating the lower jaw on the right, a "step" is defined along the lower edge in the projection of the second premolar. There is no contact between the antagonist teeth on the deformation side, starting from the second premolar. A radiological examination shows a consolidated fracture of the lower jaw body on the right, with the distal fragment shifted down by 2.0 cm.

Make a diagnosis. Schedule a treatment plan.

Task 2. The patient, 23 years old, came to the clinic with complaints of swelling in the left submandibular region, redness of the skin over it, pain in the lower jaw angle to the left, aggravated by chewing. From the anamnesis, it was found out that a month ago, during a fight, he was injured in the lower jaw on the left. I did not go to the doctor; I took pains with analginum. He could not take solid food. A week ago, there was a swelling in the left submandibular region, chills, temperature up to 37.5 ° C, the patient himself took antibiotics at home. From past diseases: children's infections, acute respiratory infections, appendectomy. During a local examination: the face is asymmetrically due to swelling in the left submandibular region, the skin is hyperemic, does not gather in the fold, is determined by a dense infiltrate painful to palpation, 6.0x7.0 cm in size. Palpation is painful. In the oral cavity: the presence of multiple carious cavities of the teeth, the mobility of the fragments in the area of the mandibular coals on the left is determined; from the gingival pocket in the region of the third molar, there is a purulent discharge from the left. At X-ray examination, the shadow of the fracture line is determined in the region of the lower jaw on the left, the divergence of the lower jaw fragments is 0.6 cm, in the area of the fracture there are visible the shadows of three free-lying sequesters, 0.3x0.4 cm in size.

Make a diagnosis. Schedule a treatment plan.

Task 3. The patient, 27 years old, came to the clinic with complaints of redness of the skin and swelling in the lower left cheek area, pain in this area, numbness of the skin of the left half of the lip and teeth in the lower jaw on the left, pain in the lower jaw when eating, general malaise weakness, poor appetite. From the anamnesis, it was found out that a month ago he was injured in the lower jaw on the right and was treated for 10 days in the hospital for a fracture of the lower jaw on the right in the mental opening, then he was outpatiently treated in the clinic at the place of residence, 3 weeks after splinting, bimaksilyarny tires , was discharged to work. A week later, a swelling appeared in the lower jaw area on the left, painfulness, turned to the dentist. From past diseases indicates childhood infections, colds. On local examination: face somewhat asymmetrically due to swelling in the lower part of the buccal region on the left. The skin over the swelling is hyperemic, thickened, palpation is somewhat painful. Clinically, the mobility of the lower jaw fragments on the left in the area of the previous fracture is not determined. X-ray examination determines the shadow in the upper third of the mandible fracture line to the left in the area of the mental opening in the consolidation stage, along the edge of the mandible to the left, the shadow of the sequester is determined, 0.8x0.9 cm in size, free-lying and with the surrounding bone tissue unconnected.

Conduct a rationale for the diagnosis. Make a diagnosis.

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ЧЕЛЮСТНО-ЛИЦЕВАЯ ТРАВМА

MAXILLOFACIAL TRAUMA

учебно-методическое пособие
на английском языке

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