Etiology and Prevention of Nerve Injuries

3

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3.1 Introduction

Dental treatment, surgical operations, and traumatic injuries to the oral cavity and maxillofacial region occur in close proximity to peripheral branches of the three major divisions of the fifth cranial (trigeminal) nerve (TN5), the main sensory innervation to several important structures in the head and neck. Despite detailed knowledge of the regional anatomy and the application of skillful surgical technique, injuries to the TN5 are not always avoidable [79]. In this chapter, situations in which TN5 injuries are known to occur, the mechanism of injury (if known), the local neuroanatomy, and measures or technical modifications that might reduce the risk of trauma to adjacent TN5 branches will be presented.

3.2 Neuroanatomy

The anatomy of the TN5 is complex, and an additional review will be helpful to clinicians [31, 106, 114]. All three divisions of the TN5 are at risk for injury. Those peripheral branches which are most often involved in cases of nerve injury include the supraorbital nerve (SON) and the supratrochlear nerve (STN) from the ophthalmic (first, V1) division of TN5, the infraorbital nerve (IFN) from the maxillary (second, V2) division, and the inferior alveolar (IAN), lingual (LN), mental (MN), and long buccal (LBN) nerves from the mandibular (third, V3) division (Figs. 3.1 and 3.2).

Rarely do injuries occur to the other branches of the TN5, such as the anterior, middle, and posterior superior alveolar, nasopalatine, and greater palatine nerves of V2 and the mylohyoid, auriculotemporal, and incisive nerves of V3, perhaps because

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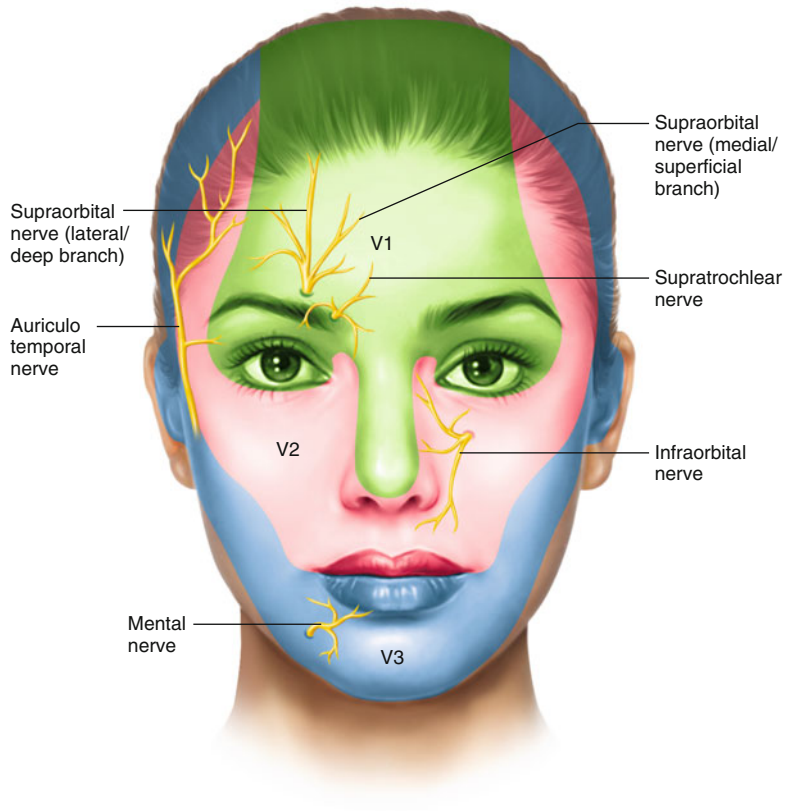


Fig. 3.1 Sensory innervation of the face via branches of the three major divisions of the trigeminal nerve. *V1* ophthalmic division, *V2* maxillary division, *V3* mandibular division

alteration of sensation in the affected areas is not readily perceived by patients and does not seriously interfere with orofacial functions, or merely that the paresthesia resolves rapidly [76]. For example, temporary, but sometimes prolonged, numbness of the palate is common after a LeFort I maxillary osteotomy because of involvement of the nasopalatine and greater palatine nerves. However, it is seldom a long-term patient complaint and does not seem to interfere with speech, mastication, or drinking or swallowing liquids [69]. The buccal and labial gingivae are routinely anesthetic following a LeFort I osteotomy because the terminal fibers of the middle and anterior superior alveolar nerves are severed by the usual circumvestibular incision. Recovery of this sensation occurs within a few weeks or months, and the interval of gingival insensitivity has little or no effect on oral function. The mylohyoid nerve which branches from the IAN in the pterygomandibular fossa provides motor innervation to the mylohyoid muscle and the anterior belly of the digastric muscle. In some patients, it has a sensory component that supplies a small area of skin in the submental area where loss of sensation is not often perceived by the patient. Likewise, the auriculotemporal nerve (ATN) is frequently injured during temporomandibular joint surgery, parotid gland surgery, or rhytidectomy, but the alteration of sensation in the periauricular region generally resolves within a few months and is seldom a problem for the patient. Occasionally, however, injury to the ATN is associated with the development of Frey's syndrome (gustatory sweating, see Sect. 3.5.7) that can be a significant aggravation for the afflicted patient [132]. Also, the incisive nerve is often intentionally sectioned to allow for maximal lateralization or advancement of the IAN during nerve repair surgery after injury or to allow for lateral repositioning for dental implant placement. The resulting loss of sensation in the mandibular labial gingiva and anterior teeth does not present a problem for most patients, although the lack of tactile proprioception in the incisors may be frustrating for some patients. In addition, however, an amputation

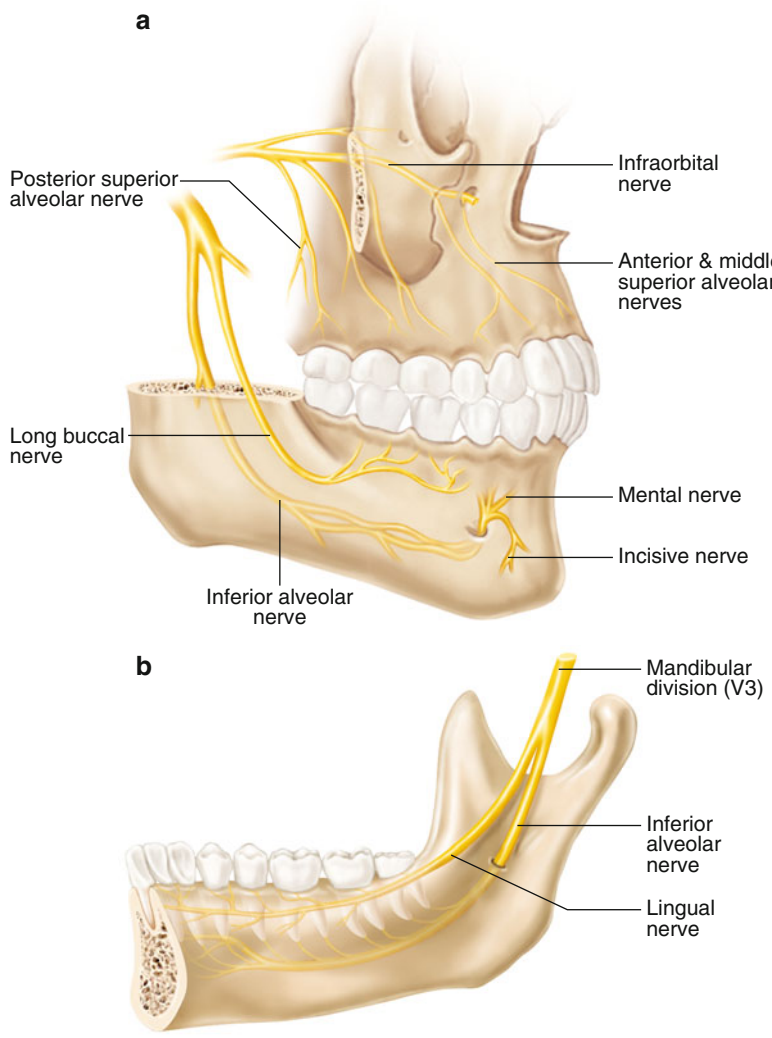


Fig. 3.2 Important sensory branches of the trigeminal nerve in the oral cavity: (**a**) labio-buccal aspect of maxilla and mandible; (**b**) lingual area of mandible

neuroma may develop rarely on the proximal stump of a transected incisive nerve possibly leading to painful neuropathies [14].

3.2.1 Supraorbital and Supratrochlear Nerves

The supraorbital nerve (SON) traverses along the superior orbital fissure above the bony orbit and exits through the supraorbital foramen, or notch, in the superior orbital rim of the frontal bone. From this point, the SON and its branches proceed medially, laterally, and cephalad to supply sensation to the eyebrow, forehead, and anterior scalp. The SON has a "superficial" (lateral) division and a "deep" (medial) division. The superficial division courses superficially over the frontalis muscle and supplies sensation to the skin of the forehead, while the deep division proceeds more cephalad beneath the galea aponeurotica to innervate the frontoparietal region of the scalp [65]. This deep (medial) division has implications in the surgical dissection utilized for a forehead or brow-lift procedure (see Sect. 3.5.9). The supratrochlear nerve (STN) exits from beneath the superior orbital rim about 1 cm medial to the supraorbital foramen and provides branches to

the upper eyelid and lower midportion of the forehead. The patient seldom notices the loss of sensation from the STN alone following forehead injury or surgical procedures.

3.2.2 Infraorbital Nerve

The infraorbital nerve (IFN), the most important branch of V2, traverses the inferior orbital canal below the floor of the orbit and exits via the infraorbital foramen inferior to the inferior orbital rim. From there it divides into several branches as it proceeds peripherally. Its locations within the inferior orbital canal and following its exit from bone make it susceptible to injury from trauma or various surgical procedures. The injured IFN may produce symptomatic neurosensory dysfunction in the upper lip and middle third of the face (see Sects. 3.5.3 and 3.5.4).

3.2.3 Inferior Alveolar Nerve

The inferior alveolar nerve (IAN) leaves V3 in the pterygomandibular space and courses anterolaterally to the medial surface of the mandible into which it enters at the mandibular foramen. From here, its location within the inferior alveolar canal (IAC) can be highly variable, superoinferiorly, between the molar and premolar teeth and the mandibular inferior border and, mediolaterally, between the lateral and medial mandibular cortices (Fig. 3.3). Recognition of this variability of position of the IAN is important in planning a surgical procedure for the removal of mandibular third molars (M3s), correction of mandibular developmental deformities with orthognathic surgery, repair of mandibular fractures, placement of dental implants, and endodontic periapical surgery (see Sects. 3.5.2, 3.5.3, 3.5.4, 3.5.5, and 3.5.7). This location can usually be determined from plain radiographs in most patients [39, 56]; however, in those patients who are suspected of having an intimate relationship between the IAN and an approximating tooth, implant, or other object or structure (based upon plain-film assessment), the availability of newer imaging techniques (computed tomography (CT), cone-beam computed tomography (CBCT)) has made possible the precise and accurate determination of the position of the IAN within the mandible (see Chaps. 5 and 11).

3.2.4 Mental Nerve

The mental nerve (MN) arises from the IAN in the inferior alveolar canal in the premolar region. The MN then courses superiorly and posteriorly to exit the lateral surface of the mandible through the mental foramen (MFN), generally located between and slightly inferior to the apices of the mandibular first and second premolar roots. Vertical or horizontal incisions and submucosal dissections in the mandibular buccal vestibule should be performed with great caution in this area. The level of exit of the MN is generally several millimeters superior to the level of the inferior alveolar canal, a relationship that impacts upon the placement of a horizontal osteotomy for mandibular symphysis repositioning or genioplasty (see Sect. 3.5.9). As it exits the MFN, the MN usually divides into three distinct branches that pass inferior, lateral, and anterior (lower labial branches, LLBs) to supply the lower labial mucosa and skin of the lower lip. Occasionally, there is an anatomic variation in which the MN exits the mandible as two separate branches via two bony mental foramina (Fig. 3.4). Knowledge of the position of the LLBs of the MN [1] aids the clinician in determining appropriate incision designs in the lower labial mucosa for various procedures (such as biopsy of minor salivary glands, excision of submucosal masses, and mandibular symphysis procedures) while minimizing the risk of injury to the MN. In general, the LLBs of the MN proceed in an anteromedial direction at an angle of about 36 % to the horizontal plane of the lower lip, so an incision in the lower labial mucosa for removal of a submucosal mass should parallel the direction of these branches. A U-shaped incision with its lateral aspects parallel to the LLBs should be made to expose the mandibular symphysis (Fig. 3.5). When the patient has lost posterior mandibular teeth and there is

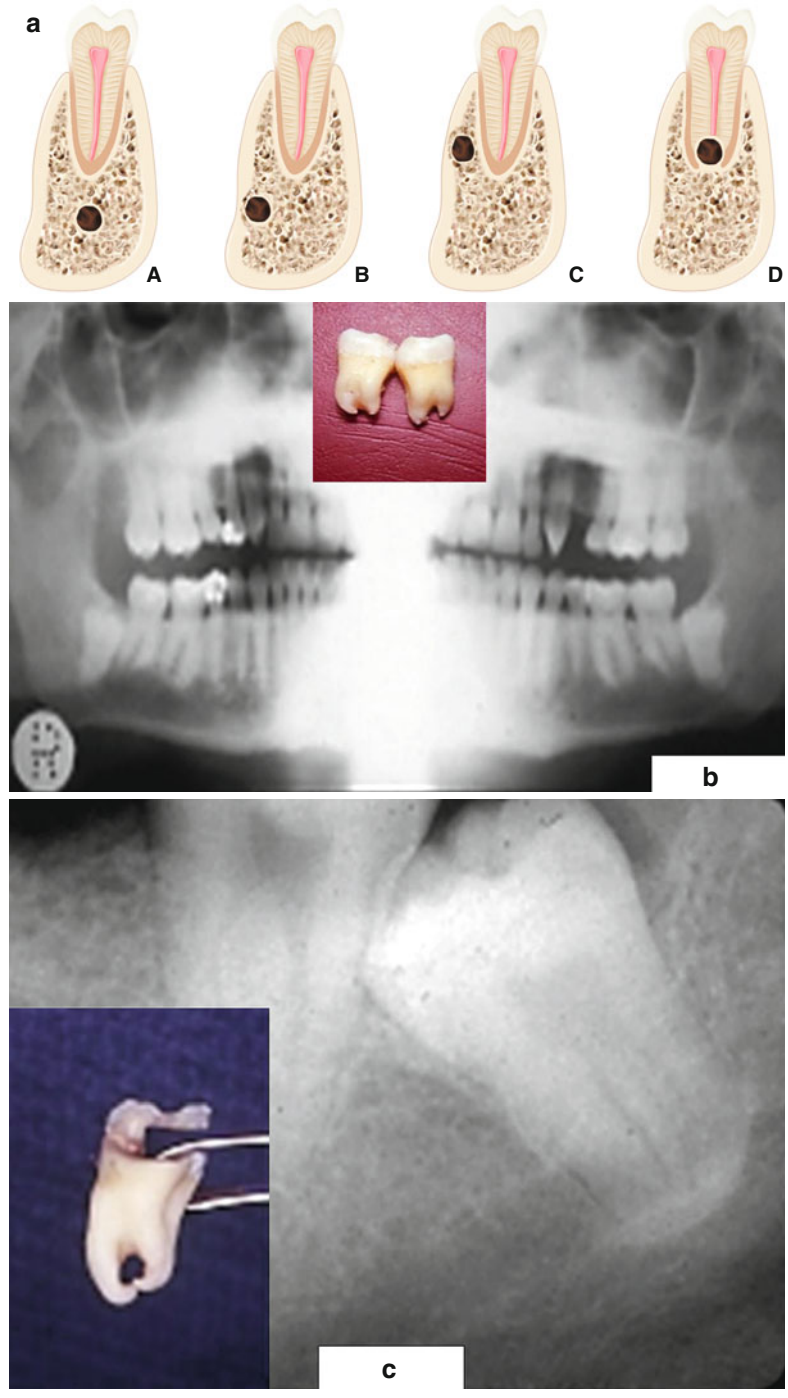


Fig. 3.3 Variable locations (*left*, **a**) of the inferior alveolar canal (IAC) in the mandible molar region (seen in cross section) which can be determined from preoperative imaging studies: (*A*) the IAC lies several millimeters inferior to the tooth root apex, a favorable position during M3 removal; (*B*) the IAC, situated inferiorly, is grooving the lateral cortical bone, placing it at risk during the mandibular sagittal split ramus osteotomy (MSSRO); (*C*) the IAC, located superiorly, again is grooving the lateral cortical bone, placing it at risk when performing the MSSRO or inserting superior border monocortical internal fixation screws; (*D*) the IAC lies within a groove in the root apex, posing a risk of injury during the removal of the tooth. Bilateral impacted mandibular third molars (M3) in which the roots are straddling the IAC (*middle*, **b**). An M3 whose roots were perforated by the IAC (*right*, **c**). The IAN was severed during M3 removal and was later successfully repaired with microsurgery

alveolar bone atrophy in the mandibular body region, the MFN and/or the IAC may be located at, or near, the alveolar crest, placing the MN or the IAN at risk from incisions or other surgical manipulations in this area [55, 77, 78] (Fig. 3.6).

3.2.5 Lingual Nerve

The lingual nerve (LN), after it leaves V3 in the pterygomandibular space, proceeds anteriorly where it assumes a variable relationship to the

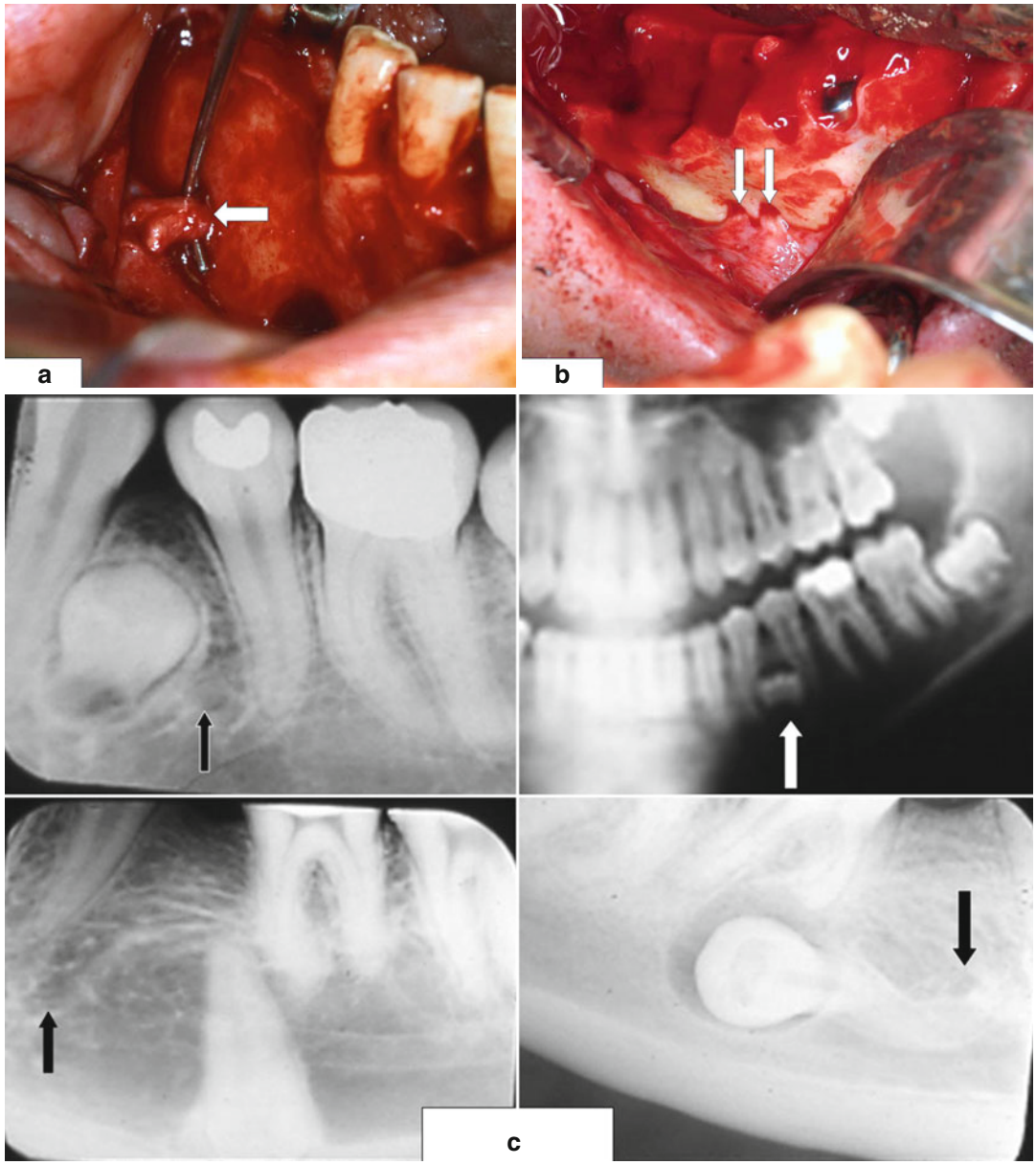


Fig. 3.4 Mental nerve (MN, indicated by *white arrow*) usually (**a**) exits the buccal surface of the mandible inferior to the root apices of the two premolar teeth; (**b**) a view of patient with two right mental foramina, each with a MN; (**c**) radiographic views of impacted mandibular premolar teeth, each of which is in close proximity to its adjacent mental foramen (*arrows*), posing a risk of MN injury during their removal

medial surface of the mandible in the third molar (M3) area. Cadaveric dissections and clinical experience have shown that the LN in the M3 area may be located in intimate contact with the medial mandibular periosteum at, or above, the level of lingual crest of bone (Fig. 3.7) or one to several millimeters below the alveolar crest at various distances (from 0 to several millimeters) medial to the lingual mandibular periosteum [20, 63, 86]. It has been noted that these nerve-bone relationships may not necessarily change in patients who subsequently lose their teeth and

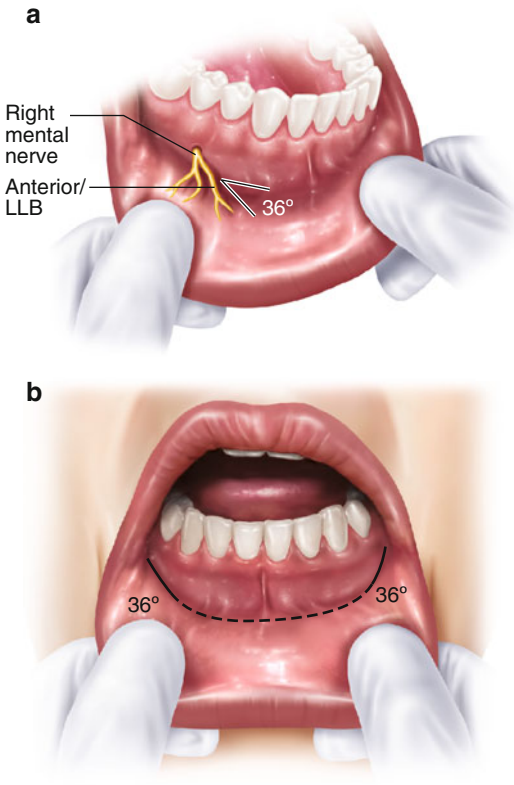


Fig. 3.5 (**a**) MN gives off its anterior/lower labial branches (LLB) which course anteriorly at an angle of about 36° with the horizontal plane of the lower lip. An incision in this area should parallel the LLB; (**b**) a labial vestibular incision for access to the mandibular symphysis has its lateral wings (*solid black lines*) parallel to the LLB. Remainder of the incision is a *dotted line*

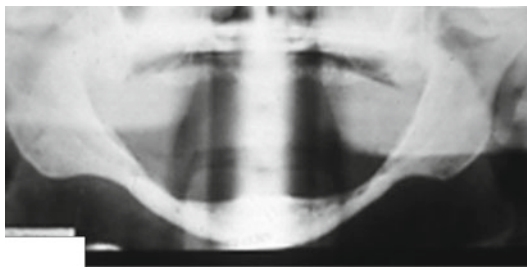


Fig. 3.6 Severe atrophy of the mandible. IAC and MF are at or near the crest of the residual alveolar ridge

undergo mandibular atrophy [55]. The position of the LN on one side of a bilateral cadaver dissection [103] and as seen in clinical experience is not a reliable predictor of its position on the contralateral side. The frequently noted intimacy of the LN and the mandible in the third molar region increases the risk of LN injury from removal of M3s or other surgical procedures in the retromolar pad area (see Sects. 3.5.2, 3.5.3, 3.5.4, and 3.5.5). As the LN courses anteriorly from the M3 region, it again may assume a variable relationship with the submandibular salivary duct and the submandibular salivary gland. In some patients, the LN runs medially inferior to the submandibular duct and then into the floor of the mouth and tongue musculature. In other patients, the LN runs through or inferior to the submandibular gland to reach the body of the tongue muscle [88, 89]. In these latter two relationships, the LN might be in jeopardy during surgical procedures of the sublingual salivary gland, submandibular gland, or Wharton's duct. As the LN proceeds anteriorly from the M3 area and into the floor of the mouth, it assumes a more tortuous course. This has implications for the surgical repair of LN injuries in that dissection and mobilization of the distal portion of a severed nerve often allows it to be advanced without tension to approximation with the proximal nerve stump. The nerve gap is eliminated and a direct neurorrhaphy, rather than an indirect reconstruction with a nerve graft or conduit, may be performed [12, 13].

3.2.6 Long Buccal Nerve

The long buccal nerve (LBN) leaves V3 in the pterygomandibular space and crosses lateroinferiorly in a supraperiosteal location over the deepest concavity of the external oblique ridge of the mandibular ramus, or up to 12 mm inferior to this point. There the LBN may separate into several smaller branches or continue as a single structure into the mandibular buccal vestibule in the molar area where it then sends multiple smaller branches medially, laterally, and anteriorly to supply the buccal molar gingiva, buccal mucosa, and mandibular vestibule, respectively [52]. While the main trunk of the LBN as it crosses the external oblique ridge is often 1 mm in diameter, it is seldom noted in surgical dissections in the retromolar pad or vestibule of the posterior mandible unless it is the subject of exploration

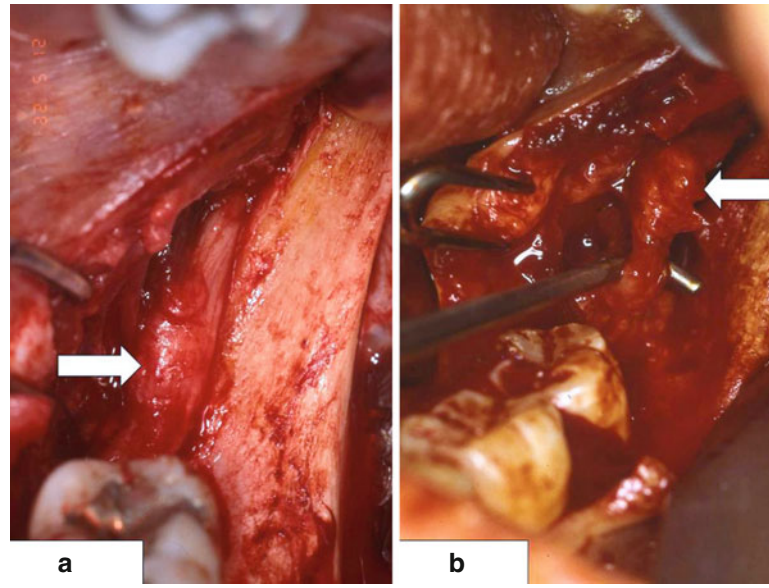


Fig. 3.7 (**a**) Intact left lingual nerve (LN, *arrow*), exposed during a mandibular ramus surgical procedure, is located at level of alveolar crest in mandibular retromolar area; (**b**) left LN injured during mandibular third molar removal several months previously has developed a neuroma-in-continuity (*arrow*)

and repair. When the LBN crosses below the greatest concavity of the external oblique ridge, it may be at risk of injury from incisions in the posterior mandibular buccal vestibule, such as those performed for M3 removal, mandibular ramus osteotomies, or open reduction of posterior mandibular body, angle, ramus, or condylar fractures. In the majority of patients, transection of the main trunk of the LBN, or one or more of its branches, is associated with little, if any, perceived sensory aberration [76], possibly due to a high mechanosensory threshold of this nerve [51]. However, in some patients, a LBN injury results in significant sensory dysfunction, especially if a painful neuroma develops on the proximal stump of a severed LBN [11] (Fig. 3.8).

3.3 Types of Nerve Injury

3.3.1 Clinical Categories of Nerve Injury

Clinically, peripheral nerve injuries are divided into two categories: *closed* and *open injuries*. The vast majority of TN5 injuries occurring during elective surgery, except those nerve resections which are planned as part of ablative surgery, are unobserved or are unsuspected by the surgeon at the time of operation [110]. Only in retrospect, when the patient returns with a complaint of sensory dysfunction, is the diagnosis established and the surgeon obliged to evaluate the situation further. Such an injury, not directly observed by the surgeon at the time of its occurrence, is termed a *closed* (*or unobserved*) *injury*. When a nerve injury is noticed at the time of surgery, whether it is produced intentionally, such as during surgical excision of a malignant tumor in which the nerve is involved, or unintentionally, such as during an elective, non-ablative operation, this is called an *open* (*or observed*) *injury*. An open injury is documented in the surgeon's notes or operative report, and if the nerve is not to be repaired at the time it occurs, the injured area of the nerve may be tagged with fine, nonabsorbable, nonreactive sutures (such as 8-0 monofilament nylon) to assist the surgeon who does the subsequent microsurgical repair in identifying the proximal and distal nerve stumps.

3.3.2 Mechanisms of Nerve Injury

There are many aspects of surgical manipulation that can lead to TN5 injury (Table 3.1). Some of these might be recognized clinically, if the nerve is exposed, and repaired at that time or within a

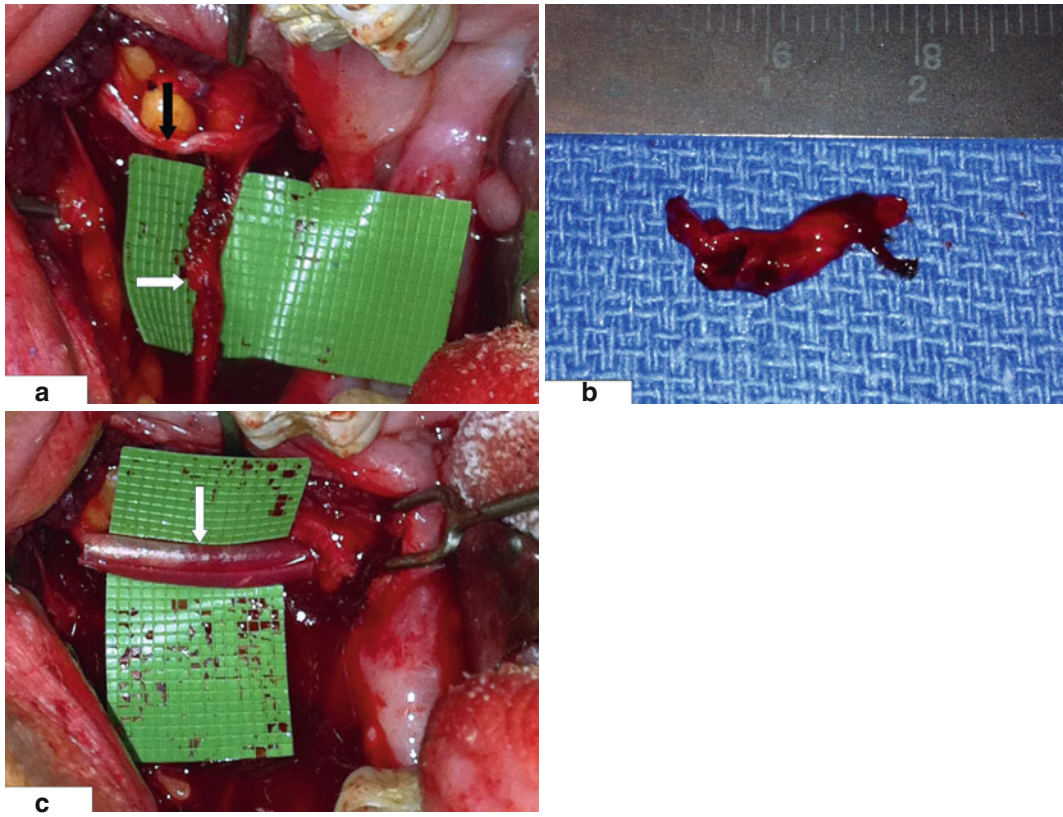


Fig. 3.8 Right long buccal nerve (LBN) is shown, (**a**) with its normal-appearing main branch (*black arrow*) traversing laterally into the cheek mucosa and an abnormal anterior branch with neuroma (indicated by *white arrow*). The patient developed stimulus-evoked pain in the right buccal vestibule following removal of the right third molar tooth. The pain resolved (**b**) after resection of the anterior branch and its neuroma; (**c**) main branch of the LBN is surrounded by membrane sheath (indicated by *white arrow*) to facilitate healing after resection of anterior branch

short time after injury (as in a delayed primary repair at 3 weeks) [99]. However, within several weeks, the healing process has begun and scar tissue has formed, and although these events may render the surgical repair technically less difficult [79], they alter the appearance of the injured nerve, and frequently make the mechanism of injury difficult to determine clinically or histologically. From direct clinical observation and other considerations [97], it has been proposed that the TN5 may be injured via the following: (1) sharp incision (as from a scalpel or an anesthetic needle) that may cause a partial (one or more fascicles) or total (all fascicles) nerve transection; (2) blunt trauma associated with maxillofacial injuries or from instrumentation such as elevation of a mucoperiosteal flap; (3) stretching, compression, or laceration from displaced bone fragments in facial bone fractures; (4) manipulations during reduction of fractured bone fragments or osteotomized bone segments that produce nerve compression or crushing; (5) a high-speed rotating bur during bone removal or a slower-speed drill preparing dental implant sites or bone holes for internal fixation screws that causes ragged and irregular nerve shredding; (6) impaling the nerve with an internal fixation screw; (7) prolonged or excessive retraction of the nerve that induces ischemia and a stretching (or neurapraxic) injury; or (8) contact with a toxic root canal medicament or sealer or other chemical medications (such as tetracycline placed into a tooth-extraction socket) that generate a chemical burn of the nerve.

Table 3.1 Etiology of TN5 injuries

Procedure	Nerves affected	Mechanism of injury
Local anesthetic injection	IAN, LN	Direct needle trauma Toxic effect of anesthetic Bleeding, hematoma
M3 removal	IAN, LN, LBN	Incision Flap retraction Rotating bur, osteotome Compression (bone, root) Suturing Socket medication
Orthognathic surgery: Lefort I, MSSRO, MIVRO	IFN, IAN, LN	Drill, osteotome, saw Internal fixation Nerve retraction Nerve compression
Maxillofacial trauma: Fracture, laceration, GSW	SON, IFN, IAN, MN	Compression Severance Avulsion Internal fixation
Preprosthetic surgery: Ridge augmentation Vestibuloplasty Dental implants	IAN, LN, MN	Chemical burn Compression, suture Compartment syndrome Rotating bur
Endodontic treatment: Root canal filling Periapical surgery	IAN, MN	Overinstrumentation Compression Chemical burn
Salivary gland surgery: Submandibular, sublingual	LN	Dissection
Ablative surgery: Benign cysts/tumors Malignant tumors	IAN, MN, LN	Unintentional injury Intentional nerve resection
Cosmetic facial surgery: Genioplasty, facelift, forehead/brow lift	SON, MN, ATN	Dissection Compression Rotating bur, saw

TN5 trigeminal nerve, *IAN* inferior alveolar nerve, *LN* lingual nerve, *M3* mandibular third molar, *LBN* long buccal nerve, *IFN* infraorbital nerve, *MSSRO* mandibular sagittal split ramus osteotomy, *LeFort I* maxillary horizontal osteotomy, *Fx* fractured facial bone, *SON* supraorbital nerve, *MN* mental nerve, *GSW* gunshot wound/missile injury, *ATN* auriculotemporal nerve

3.4 Incidence of Nerve Injuries

Reliable statistics about the frequency of TN5 injuries are hard to obtain since so much of the activity (dental treatment, intraoral surgery, and cosmetic procedures) associated with these injuries is performed in private practice offices where either thorough documentation is incomplete or the databases lack the capability for retrieval of pertinent information on nerve-injured patients. Even in hospitals, recognition of the event of a nerve injury may not take place until after the patient has been discharged, rendering a retrospective database search futile or misleading in many cases, even with the most sophisticated electronic medical record computer systems; without the data input, there can be no data retrieval. In the absence of national or international registries for the accumulation of nerve injury data, most of the current information concerning the causes and frequency of TN5 injuries has come from group

Table 3.2 Incidence of TN5 injury based on procedure

Procedure	Posttraumatic NSD[a] (%)	Postoperative NSD[b] (%)	Permanent NSD[c] (%)[d]
Local anesthetic injection	N/A[e]	0.0033–3.3	0.54
M3 removal	N/A[e]	0.10–0.40	0.001–0.040
Genioplasty	N/A[e]	100	3.33–10.0
Mandibular SSRO	N/A[e]	63.3–83.0	12.8–39.0
SSRO + genioplasty	N/A[e]	100	66.6
Mandibular IVRO	N/A[e]	18.0	0.01
Mandibular DO	N/A[e]	46.7	<5.0
Mandible fracture	46.0–58.5	76.1–91.3	38.8
ZMC fracture	52.0–100	7.7–55.0	37.0
Mandibular vestibuloplasty	N/A[e]	100	50–100
Dental implant	N/A[e]	1.7–43.5	0–15

TN5 trigeminal nerve, *M3* mandibular third molar tooth, *SSRO* sagittal split ramus osteotomy, *IVRO* intraoral vertical ramus osteotomy, *DO* distraction osteogenesis

[a]Paresthesia of TN5 branch present after injury, but before surgical intervention

[b]Postoperative sensory dysfunction = paresthesia present after operation that resolves by 3 months post-injury and/or is acceptable to the patient

[c]Permanent sensory dysfunction = sensory aberration (moderate hypoesthesia to anesthesia ± hyperesthesia) that persists beyond 3 months post-injury. This may or may not be acceptable to the patient and require surgical intervention

[d]Permanent sensory dysfunction may be better tolerated by patient when this was either an expected sequel as disclosed during the preoperative consent process or due to a traumatic injury in which the patient's expectations for sensory recovery were modest or had low priority when there were coexisting life-threatening injuries

[e]N/A = applies only to mandibular fracture and ZMC complex fracture

surveys, reports of individual experience in the performance of certain procedures, or retrospective or prospective case reports or case series of the results of microsurgical repair of TN5 injuries in the literature. There is little doubt that the incidence of TN5 injuries from all etiologies, but especially those resulting from local anesthetic injections, is underreported [100]. This information is summarized in Table 3.2, and it is discussed further below in relation to the individual causes or mechanisms of TN5 injuries.

Perhaps of equal importance to the "clinical outcome" of peripheral TN5 injuries is the patient's perception of his or her neurosensory status and their ability to carry out the usual oral and facial functions that depend upon sensory input. Some patients who achieve a level of "functional sensory recovery" based upon clinical testing may still continue to experience adverse symptoms or interference with function and activities of daily living, while others will tolerate compromised oral or facial sensation without significant difficulty. In general, however, most patients who experience greater neurosensory improvement after surgical repair of TN5 injuries report lower frequencies of related oral/facial dysfunction [119]. Assessment of the degree of recovery of sensory function and its long-term effects on quality of life (i.e., "patient-centered research") deserve more attention of clinicians and researchers, since the care of the nerve-injured patient [68, 105] and, indeed, all types of patients [16] will continue to evolve in the future.

3.5 Causes of Nerve Injury

3.5.1 Local Anesthetic Injections

Injection of local anesthetics for dental treatment or oral and maxillofacial surgery is by far the procedure most frequently performed in proximity to peripheral branches of the TN5. It is estimated that the average general dental practitioner administers between 3 and 10 mandibular nerve blocks per day, or 20–25 per week, and he/she sees some type of IAN or LN involvement (either paresthesia at the time of the injection and/or subsequent sensory dysfunction) as a result of the injection about once every 2–8 weeks. This data would imply an incidence of nerve injury of between 1:30 (3.3 %)

and 1:300 (0.003 %) [47, 100] (Table 3.2). Given that it is essentially a blind (albeit trained and practiced) maneuver within the pterygomandibular space, it seems curious that the incidence of injection-associated IAN and LN injuries is not greater. Is this a case of underreporting or a testament to the skill of the average dentist or merely luck? Of course, the goal of the injection is to deposit the anesthetic solution *in close proximity* to the nerves being anesthetized and to avoid actual contact with the nerve. If this is achieved and apparently it is in the vast majority of injections, then the happenstance of needle contact with the nerve and the possible sudden dysesthesia ("electric shock," the possibility of which is always a patient's fear) is avoided in most cases, or it may be that contact between the needle and the nerve may not result in any significant neurosensory dysfunction. The dysesthesia resulting from needle contact with the nerve is not a reliable indicator of subsequent significant, prolonged or permanent, sensory dysfunction, however. "Needle shock" does not always occur in patients who subsequently fail to regain sensation in the usual time frame, and in many patients who experience the sudden pain of needle to nerve contact, there is no subsequent sensory dysfunction [100]. In those patients who are under intravenous sedation or general anesthesia before the injection of local anesthetic is performed, there will be no recollection of needle contact with the nerve [82].

There are three proposed mechanisms of nerve injury resulting from a local anesthetic injection [101]. These include the following: (1) *direct trauma*, the needle may pierce the nerve, injuring one or more fascicles, and (2) *chemical toxicity*, the anesthetic solution may have a neurotoxic effect. All local anesthetic solutions have to meet FDA specifications and are thought to be nontoxic in the concentrations used to produce local anesthesia in human patients. Recently, however, mention has been made of the potential toxicity of a 4 % solution of articaine hydrochloride when used for local anesthetic nerve blocks for dental procedures [54] (see Chap. 5 on Injection Injuries). Also, there is the possibility that a cartridge containing any of the commonly used local anesthetics (i.e., lidocaine, mepivacaine, bupivacaine) could have a leak, and when placed into storage in a sterilizing solution (alcohol or other chemical that is neurotoxic), that cartridge might become contaminated. Upon injection of the contents of the cartridge to produce local anesthesia, the toxic sterilizing solution could be carried into contact with the nerve. The use of a disclosing agent (such as methylene blue) in the sterilizing solutions where anesthetic cartridges are stored in professional offices and clinics could eliminate this iatrogenic nerve injury; (3) *bleeding and hematoma formation*: the injection needle pierces or tears a blood vessel in the mesoneurium or epineurium of the nerve, causing localized bleeding and formation of a hematoma around or within the internal structure of the nerve thereby producing a compression effect on the nerve. In some patients, the hematoma is rapidly resorbed, and any effect on sensory function is transient. In others, the hematoma organizes and is replaced by scar tissue that exerts a continued compression on the nerve, and neurosensory dysfunction persists. Which specific one of these effects, a combination of several effects, or other mechanisms as yet unknown occurs in a given patient remains an unresolved question at this time [100].

Following a protocol for the administration and documentation of local anesthetic injections might minimize the risk of nerve injury and provide an impetus for proper follow-up evaluation, which increases the likelihood that a nerve injury is recognized and that rapport with the patient is maintained [82]. When the patient is fully conscious, the clinician proceeds to insert the local anesthetic needle into the proper location (i.e., pterygomandibular space). In the absence of the patient's complaint of sudden pain or shocking sensation (dysesthesia, which may radiate to the lower teeth, lower lip, mandible, or tongue), the syringe is aspirated. If the aspirate is free of blood, the local anesthetic is administered with the needle position unchanged. If there is a bloody aspirate, the needle is withdrawn 2–3 mm and aspiration is repeated. If the aspirate is then clear, the local anesthetic is injected with the needle in the new position. If the patient complains of sudden pain or shocking sensation, the needle is withdrawn 2–3 mm. Following a clear aspiration, the anesthetic is injected in this new position. If

there is either a bloody aspirate or a dysesthesia associated with the injection, the incident is noted in the patient's record, and a follow-up evaluation of sensory function is done at the patient's next visit (see Chap. 10). When the patient is under general anesthesia or intravenous sedation, the patient will not be able to react to a dysesthesia. Therefore, aspirate before injecting and proceed as described above.

While the IAN and the LN are the TN5 nerves most frequently injured by local anesthetic injections [101, 102], injuries to other branches including the LBN, nasopalatine, mental, and IFN have been seen by the authors.

For further discussion of this topic, the reader is referred to Chap. 4.

3.5.2 Mandibular Third Molar Removal

Removal of third molar teeth is the most frequently performed surgical procedure in oral surgery practice [95]. It has been estimated that some oral and maxillofacial surgeons (OMFS) remove as many as 25 or more M3s per week in their office practices. During the latter half of the twentieth century, a number of reports (from Europe, the United Kingdom, New Zealand, and the United States) indicated that an injury to the IAN or LN during M3 removal occurred in 1.0–6.0 % of patients, with 0.1–1.0 % of these injuries failing to resolve within a few months and becoming permanent in the absence of surgical intervention [3, 23, 24, 27, 46, 53, 64, 129].

More recently, a prospective study conducted by the American Association of Oral and Maxillofacial Surgeons (AAOMS) of a selected group of 63 American oral and maxillofacial surgeons who removed 8,333 M3s from 3,760 patients over a 1-year period (January–December 2001) found an incidence of IAN injury of 1.1 % on the left side versus 1.7 % on the right side, while the LN was involved in 0.3 % (equal on both sides). These figures were for the immediate postoperative period only, so there was no indication of whether any of these injuries failed to resolve spontaneously [49]. A retrospective survey of California OMFS showed that in 95 % of practices surveyed ($n = 535$), over a 1-year period, 94.5 % experienced one or more IAN injuries, and 53 % had one or more LN injuries. Over their practice lifetimes, 78 % of these OMFS reported one or more cases of "permanent" IAN injury, while 46 % indicated one or more instances of "permanent" LN injury. The mean rate for any IAN involvement (temporary or prolonged) was 4/1,000 (0.4 %), and the permanent IAN injury mean rate was 0.4/1,000 (0.04 %). For the LN, the mean rate for any involvement was 1/1,000 (0.1 %), while that of permanent LN injury was 0.1/10,000 (0.01 %). In most cases of IAN injury, the surgeon was aware of the cause of the injury, probably due to the surgeon's knowledge of the relationship of the M3 to the inferior alveolar canal as seen on the preoperative panoramic radiograph. However, in most LN injuries, the surgeon did not know the cause, which may be because the LN was not imaged preoperatively and not directly visualized during the procedure. Nerve injury rates varied inversely with the numbers of M3s removed per year by each surgeon and his/her total years of surgical practice, emphasizing the importance of experience in the reduction of M3 surgical complications [110].

Removal of an impacted mandibular third molar (M3) presents unique surgical requirements, especially with regard to avoidance of nerve injuries. Even in an operation that is conducted according to the existing standards of care by a well-trained and experienced OMFS, it is accepted and expected that complications may occur. Mechanisms of TN5 injury while removing M3s can occur during local anesthetic injection (see above), incision placement, soft tissue flap retraction, removal of bone, sectioning of teeth, elevation of teeth, suturing, and placement of socket medications. Delayed injury of the IAN may occur when the IAC is disrupted during M3 root elevation or removal [25]. During the osseous healing process, bone proliferation may have the effect of narrowing the diameter of the IAC and compressing the IAN, a "closed box" effect similar to the sequelae of increased intracranial pressure on the intracranial contents as a result of a closed head injury. Discussed below are

suggestions for minimizing the risk of TN5 nerve injury during the removal of M3s.

Imaging studies are indispensible in the preoperative planning for M3 removal. An acceptable radiograph displays the entire tooth, the surrounding alveolar bone, the periapical area, and the inferior alveolar canal (IAC). A plain panoramic view is most often the basic imaging study for M3 evaluation. Although the depth of the tooth within the mandible (soft tissue, partial bone, or complete bone impaction) and the angulation of the tooth (vertical, horizontal, mesioangular, distoangular) are certainly important to the surgeon, perhaps most critical to the prevention of IAN injury is the relationship of the M3 roots to the IAC [56]. Several conditions seen on a plain films may indicate the likelihood of exposure of the IAN during M3 removal including (1) darkening (decreased radiodensity) of the tooth root where it is crossed by the IAC, (2) narrowing of the IAC where it crosses the M3 root, (3) interruption of the white lines (cortical walls) of the IAC, (4) diversion of the IAC, and (5) narrowing of the M3 roots [115]. When a plain radiograph suggests a possible intimate relationship between an M3 and the IAC, this situation may be clarified with advanced radiographic technology [118]. Computed tomography (CT) provides a three-dimensional view of soft tissue and bony anatomy. Although the CT scan was available only in the hospital setting, the introduction (in the 1990s) of cone-beam computed tomography (CBCT) brought this important imaging technology to office surgical practice. In the evaluation and treatment planning for M3 removal, CBCT is invaluable in determining the relationship of M3 roots to the IAC [109]. For more information on this topic, see Chap. 5.

The *location* of the *soft tissue incision* is important in avoiding injury to the LN. The posterolateral extension of the buccal incision from the mesiobuccal corner of the mandibular second molar often encounters the LBN, but injury to this nerve is only rarely symptomatic. Far more important is that the incision is not carried directly posteriorly or even posteromedially where it may cross the path of the LN, which may be located in the soft tissues overlying the impacted M3 [63].

Soft tissue flap retraction, while allowing access and visualization of the operative site, also provides protection to important neighboring structures such as the LN. Lingual flap retraction, a mainstay of the split-bone technique for M3 removal [111], might be followed by a temporary paresthesia due to mild compression of the LN, but the incidence of permanent paresthesia is not increased [94]. The LN retracting instrument protects the nerve from more severe, possibly permanent, injury in case an errant osteotome, elevator, or high-speed rotating bur penetrates the lingual cortical bone [43].

Removing soft tissue pathology from around the crown of an M3 (e.g., granulation tissue, enlarged follicular sac, dentigerous cyst) should be performed with care. If the lingual bone has been eroded or perforated, the pathologic tissue, mandibular lingual periosteum, and LN may be adherent to one another and inadvertently removed en masse, causing an avulsion injury to the LN. Periapical pathology may be located adjacent to the IAC, and curettage of the socket should be performed gently to avoid encroachment on the IAC.

During *removal of bone* or *sectioning of the tooth*, great care is taken regarding the positions of the LN and the IAN [56, 76]. Placement of a lingual retractor (see above) protects the LN if it is necessary to remove lingual bone with the high-speed drill or osteotomes in order to expose, section, or deliver the M3 [108]. When sectioning the tooth with the high-speed drill, the rotating bur should section only three-fourths of the way through the M3, thus avoiding direct trauma to an adjacent LN or IAN. Completion of the separation of the tooth fragments is performed with an elevator. Vectors of force created when *elevating teeth* should be appreciated; for example, upward and posterior elevation of the crown of a mesioangular M3 may cause a reciprocal anteroinferior rotation of the root apex and possibly adjacent bone into the IAC, causing compression of the IAN, and this would be a situation that would undoubtedly go unnoticed during the procedure. Application of excessive force during tooth elevation, especially in a patient with extensive bone resorption, or where a large amount of

bone has been removed to expose the tooth, may cause a fracture of the mandible, and fracture displacement may cause significant IAN injury.

Partial odontectomy [40] or *coronectomy* [104] can be considered as an alternative treatment to M3 removal in certain instances, including when the roots of an M3 reside in close approximation to the IAC, when there is an atrophic mandible containing a deeply impacted M3 and there is risk of pathologic fracture of the mandible, and if cases of advanced patient age. After the crown of the tooth is removed, the roots are left in situ. Subsequent development of infection or other complications such as root migration or even IAN paresthesia may occur rarely. The root migration in an occlusal direction in some patients away from the IAC may allow their subsequent removal with less chance of IAN involvement.

In general, if either the LN or the IAC contents were directly visualized during M3 removal, it is not advisable to *medicate the socket* with antibiotics (cones, powder, etc.) at the conclusion of the operation or to place analgesic liquids or pastes into the socket afflicted with alveolar osteitis several days following the extraction. If such substances (e.g., eugenol, tetracycline, Surgicel) come into direct contact with the LN or IAN, they have the potential to cause a chemical burn with long-term paresthesia, including unpleasant dysesthesia [33].

When lingual bone in the M3 area has been eroded by pathology, fractured off during removal of an ankylosed tooth, or removed surgically with a bur or osteotome, the LN may be exposed and vulnerable during *suturing* of the lingual soft tissue flap. This may cause a compressive injury to the LN, but long-term paresthesia is unlikely via this mechanism of injury.

For further discussion, the reader is referred to Chap. 5.

3.5.3 Orthognathic Surgery

The most common surgical procedures to correct developmental facial deformities associated with dental malocclusions in the upper jaw are the LeFort osteotomies (LeFort I, or horizontal maxillary osteotomy; Lefort II, or pyramidal osteotomy; and LeFort III, or transverse facial osteotomy) and, in the lower jaw, the mandibular sagittal split ramus osteotomy (MSSRO), the mandibular intraoral vertical ramus osteotomy (MIVRO), and mandibular distraction osteogenesis (MDO). Of these, the LeFort I and MSSRO pose the greatest risk of significant TN5 injury (to the IFN in the maxilla and to the IAN in the mandible) [38].

Injury to the IAN during MSSRO has been studied extensively [32], and it is well known that sensory dysfunction of the IAN following MSSRO is nearly universal (~100 %) among patients in the immediate postoperative period, being reported in 63.3–83.0 % of patients. When patients are followed for more than 1 year, the incidence of prolonged or permanent IAN injury varies from 12.8 to 39.0 %. In both the immediate postoperative evaluation and in the longer follow-up periods, both objective and subjective methods of sensory assessment were used; however, following MSSRO many patients are satisfied with their neurological status and do not request further treatment for residual IAN sensory dysfunction [41, 133]. See Table 3.2. Factors which have been found to increase the risk of IAN injury during MRSSO include the *position of the IAC* [131], especially when it is located just medial to or within the lateral cortical plate of the mandible [130, 134]; *patient age*, especially greater than 40 years [2, 5]; *type of fixation*, whether wire osteosynthesis or mono- or bicortical screws [42, 70, 80]; *magnitude of mandibular advancement*, and whether or not there was *manipulation of the IAN* [133], whether an *additional osteotomy* (such as for genioplasty) was performed [124], and also the *duration of the operation* [120].

The MSSRO is a technically demanding surgical procedure, and although the steps, techniques, instruments, and internal fixation systems utilized may differ among surgeons and are influenced by anatomic variations among patients, the following suggestions for modifications are intended to reduce, in so far as is reasonable, the risk of injury to the IAN [13, 83]: (1) Determine the exact location of the IAN preoperatively by appropriate

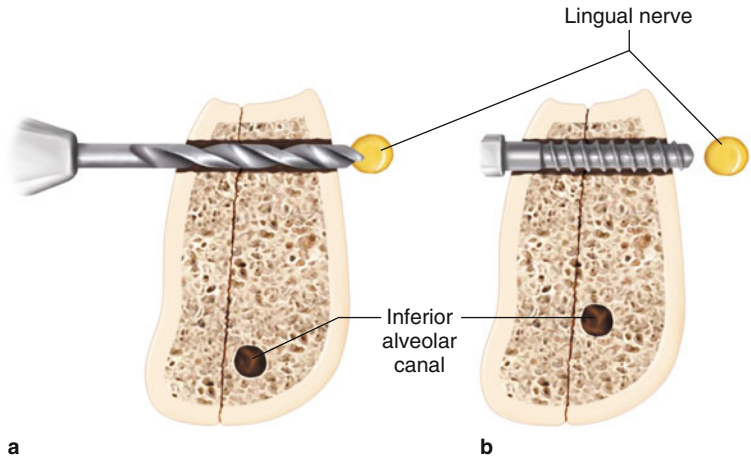


Fig. 3.9 Possible mechanisms of LN injury while placing internal fixation during a MSSRO: (**a**) allowing rotating drill bit to extend medially beyond cortical bone; (**b**) excessively long screw penetrates LN

imaging studies; (2) identify and protect the IAN with suitable retractors in the pterygomandibular space and where it enters the medial surface of the mandible at the mandibular foramen before proceeding with the horizontal osteotomy; (3) extend the vertical anterior osteotomy *just barely* through the buccal mandibular cortical bone, using a light touch with the bur or saw, as the IAN may rest just medial to the cortex; (4) begin initial separation of the osteotomy segments with anteroinferior and superior border "spreaders" until the IAN can be visualized within the separation. If necessary, when the IAN is found to be in the proximal (posterior, condyle-containing) mandibular segment, it is carefully dissected free (under magnification, as needed). After the IAN is safely contained or repositioned into the distal (anterior, tooth-bearing) mandibular segment and protected with a retractor, osteotomes can be placed to complete the osteotomy and mobilize the segments; (5) irregular bone is removed from the medial surface of the proximal segment with rasps, files, or rotating burs to provide room for the IAN and prevent its compression when the two mandibular segments are fixated together. If additional room for the IAN is needed, bone grafts (autogenous or allogeneic) are inserted between the two segments before clamping them together and placing internal fixation; (6) bicortical fixation screws are placed only through the superior aspect of the mandible, above the level of the IAC and posterior to the last tooth. Although more stable than the linear configuration of three bicortical screws at the superior border, the L configuration of bicortical screws with two at the superior border and one near the inferior border places the IAN at risk for iatrogenic injury. If preferred, monocortical screws of no longer than 5 mm are used with monocortical plates to avoid entering the IAC. A fine tactile sense is required when drilling monocortical holes so that it can be immediately appreciated when the drill has completely penetrated through the cortical bone and drilling can be terminated.

Less common is the risk of iatrogenic injury to the LN during MSSRO [60]. The LN, as it lies adjacent to the superior border of the mandible in the retromolar area (see above, Fig. 3.7), is susceptible to injury with the incision, retraction of the lingual soft tissue flap, or placement of internal fixation. This situation can be alleviated with careful attention to surgical technique [12, 77]. First, the incision is not carried to the lingual aspect of the retromolar area. Dissection and retraction of the lingual mandibular periosteum are done carefully with blunt instruments. During placement of drill holes for bicortical superior border internal fixation, either the lingual flap (containing the underlying LN) is protected with a suitable retractor (Henahan or Freer, if access allows) or the drill is not allowed to penetrate medially beyond the mandibular lingual cortical bone. A light touch and manual tactile sense are indispensable in this regard. When bicortical screws are inserted, the appropriate length is chosen to prevent "skewering" of the LN by an overly long screw (Fig. 3.9).

The MIVRO is associated with sensory dysfunction of the IAN in up to 18.0 % of patients in the early postoperative period. Long-term follow-up shows that permanent sensory aberration is extremely rare (0.01 %) [61, 135]. The key consideration in reducing risk of IAN is placement of the vertical osteotomy posterior to the location of the IAN as it enters the IAC at the mandibular foramen. This relationship can be easily determined from preoperative imaging studies [6]. At surgery, a vertical osteotomy placed 5 mm posterior to the antilingula on the lateral surface of the mandibular ramus should avoid injury to the underlying neurovascular bundle [7], although the validity of using the antilingula to determine the location of lingual has been questioned in anatomic studies.

The incidence of long-standing or permanent dysfunction of the IAN as a result of mandibular distraction osteogenesis (MDO) is small [71, 125, 127]. Distraction at a rate of no greater than 1 mm/day is tolerated well by the IAN [57]. Temporary paresthesia is expected in a majority of patients in the early postoperative period during the distraction phase that places gentle intermittent traction on the nerve. However, if direct trauma to the IAN is avoided during the corticotomy procedure, preparatory through-and-through osteotomy [74], or the sagittal split osteotomy [126] and the IAN is not injured during drilling or placement of monocortical screws for fixation of the distraction device [81], long-term recovery of sensory function is excellent with few, if any, patients experiencing bothersome residual sensory aberrations.

Altered sensory function following the LeFort I maxillary osteotomy in the upper lip, maxillary gingiva and teeth, and palatal mucosa is likely due to the severance of the terminal branches of the superior alveolar nerves with the standard maxillary circumvestibular incision and involvement of the nasopalatine nerve during the down fracture. This sensory dysfunction is common in the early postoperative period, seen in 34 of 62 (54.8 %) patients in two reported studies [61, 113], and persistent altered sensation beyond 3 months was seen in only 1 of these patients (1/62, 1.6 %). Apparently, these minor branches of V2 either heal rapidly, the lost sensation is not readily perceived by the patient, or it does not interfere with normal oral function. Permanent injury to the IFN itself during maxillary orthognathic procedures is a rare event, easily avoided by protecting the nerve as it exits the infraorbital foramen with a suitable retractor, placing LeFort II osteotomies at a safe distance medial to the infraorbital foramen or inferior orbital canal, and careful drilling and placement of internal fixation screws and plates to avoid IFN encroachment.

The anterior sliding horizontal mandibular osteotomy, employed in chin-reshaping procedures, and implants to augment chin contour place the MN at risk for injury. These genioplasty procedures are discussed under Sect. 3.5.9 below.

For further discussion of this topic, the reader is referred to Chap. 8.

3.5.4 Maxillofacial Trauma

The causes of traumatic injuries to the oral and maxillofacial region include interpersonal violence, motor vehicle accidents (MVA, or road traffic accidents, RTA), missile injuries, military combat, athletic events, and individual accidents. The prevalence of these has varied throughout history with MVA being the current most frequent cause in industrialized western societies [90]. Control of vehicle speed on highways, the installation of air bags, the wearing of seat belts by occupants in automobiles and of helmets by motorcyclists, special types of facial armor on military combatants, and the use of mouth guards and facial protection bars on football and hockey helmets have all had the effect of reducing the incidence of facial injuries in the affected populations. In the past, little attention was given to TN5 injuries that were associated with facial injuries. Emphasis was placed on anatomic reduction and stable fixation of facial bone fractures to reestablish normal facial contour, dental occlusion, and chewing function and on repair of soft tissue injuries with restoration of the integrity of the facial (seventh cranial, FN7) nerve and its control of facial movements and especially eyelid closure. In some cases, of course, in a patient in critical condition with severe multisystem injuries, definitive treatment of severe

maxillofacial injuries was deferred necessarily, while exploration and surgical treatment of primary survey life-threatening intracranial, thoracic, and abdominal injuries were performed. Many of these patients remained unconscious for long periods of time, during which they were unresponsive to sensory testing and of inadequate physical status to tolerate additional surgery on the maxillofacial region.

The mechanisms of injury to the TN5 subjected to trauma include laceration, severance or avulsion from penetrating missiles or weapons, stretching or tearing from fracture displacement, compression or crush from direct contusion of nerve branches within soft tissue (i.e., LN, MN, SON, STN, or extraosseous branches of IFN), or indirect pressure of mobile fracture fragments on nerves contained in bone (i.e., IAN or IFN) (Fig. 3.10). During the healing phase of a fracture that crosses a nerve-containing canal (i.e., IFN or IAN), exuberant bone proliferation in the area might cause a narrowing of the canal diameter producing direct compression of the nerve [25]. Such effect would be seen clinically in a delayed onset (one to several months after the injury) of neurosensory dysfunction (NSD) in the distribution of that nerve. When it occurs, this effect would impact on the incidence of long-term or permanent NSD after treatment of fractures involving the IAN or IFN (see below). The authors, in fact, have seen this effect of delayed onset of NSD in several patients with IFN injuries. In such patients, nondisplaced midfacial fractures that passed through the inferior orbital canal or infraorbital rim and were not surgically treated quickly recovered normal IFN sensation. One or more months later, onset of numbness and/or pain in the IFN distribution caused the patients to seek treatment for this paresthesia.

More recently, improved treatment protocols of trauma centers have greatly enhanced survival potential of the multiply injured patient, and facial repair and reconstruction have become an integral part of the overall treatment [8, 9, 21]. Dedicated research in TN5 injuries and patients' desires to regain control of oral and facial functions dependent on intact sensory input have stimulated OMFS interest in the evaluation and surgical repair of TN5 injuries associated with maxillofacial trauma [10]. Current information on the incidence of trauma-related injuries of the TN5 comes from a compilation of available studies, many of which are poorly documented and lack standards for nerve evaluation, grading of sensory function, or adequate follow-up periods [121]. Data summarizing TN5 injury incidence are presented in Table 3.2. Collated data from reports of maxillofacial trauma with appropriate information regarding diagnosis, neurosensory testing, and adequate length of follow-up shows that in fractures of the mandibular body and angle that involve the IAN, the incidence of posttraumatic/pretreatment NSD was 46.0–58.5 %. Risk factors for posttraumatic/pretreatment NSD included patient age (risk increased with age), gender (females have higher risk), fracture displacement, and missile trauma which frequently causes nerve severance or avulsion. Immediately after these fractures had been reduced and fixated (i.e., posttreatment), this incidence *increased* to a range of 76.1–91.3 %. That the incidence of NSD increased after surgical treatment of the fracture was most likely due to the manipulation required to expose, reduce, and fixate the fracture segments that might cause additional compression or stretching injury to the nerve. This increase was not found with injury to the IFN (see below). Combining the patients from four studies, long-term follow-up found permanent NSD of the IAN in 92 of 237 patients (38.8 %). Factors that increased the incidence of permanent NSD of the IAN after mandibular fracture repair included fracture segment manipulation, open reduction, and internal fixation.

In a 10-year retrospective review from Edinburgh of 2,067 patients with 2,160 zygomaticomaxillary complex (ZMC) fractures, the incidence of NSD in the distribution of the IFN varied from 52 to 80 %, depending upon the type of fracture (nondisplaced, 52 %; blowout of orbital floor, 60 %; orbital rim, 71 %; zygomaticoorbital [ZO] with non-distracted frontozygomatic suture [FZS], 74 %; ZO with distracted FZS, 80 %) [37]. Unfortunately, there was no long-term follow-up to provide information on recovery of IFN function in affected patients.

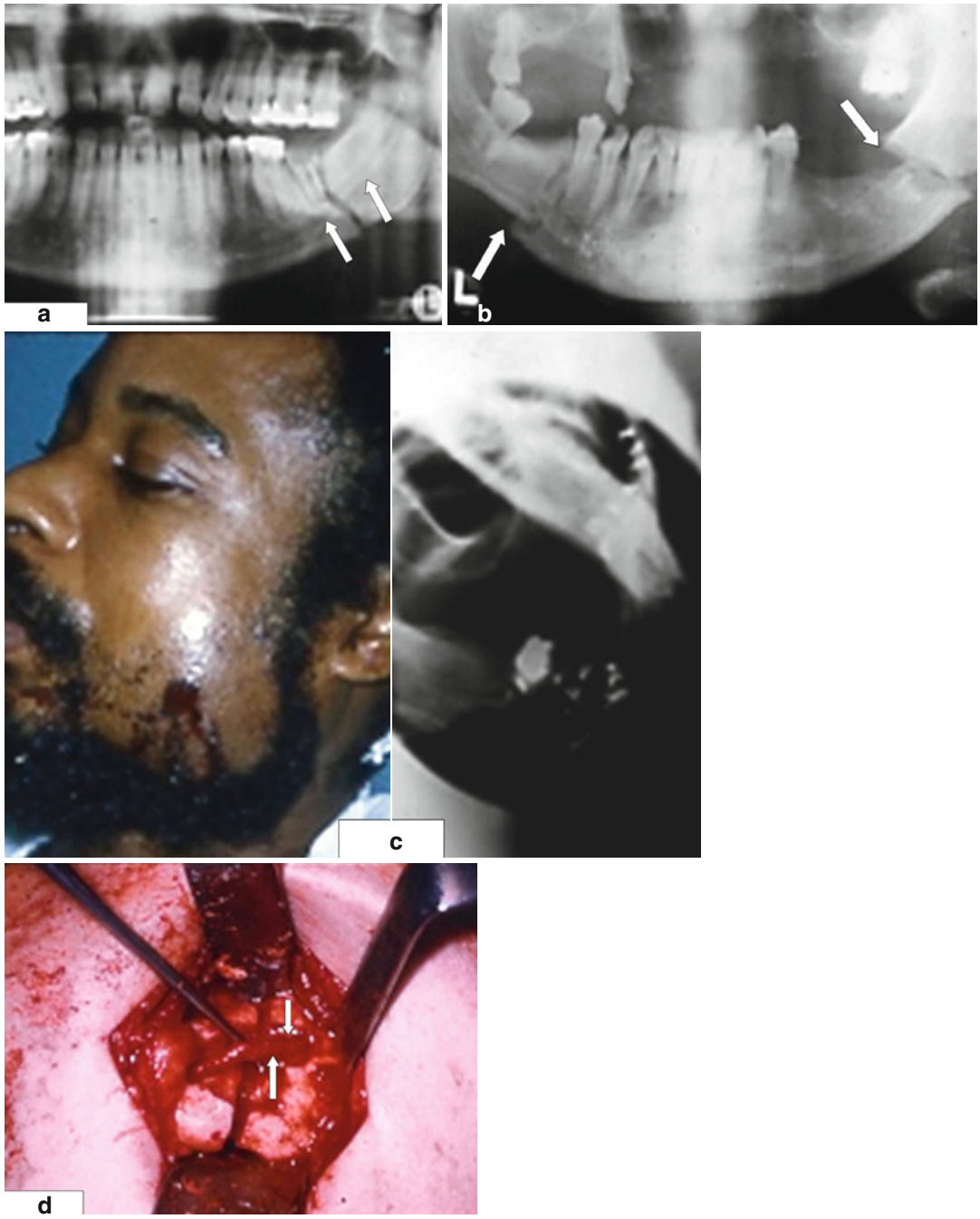
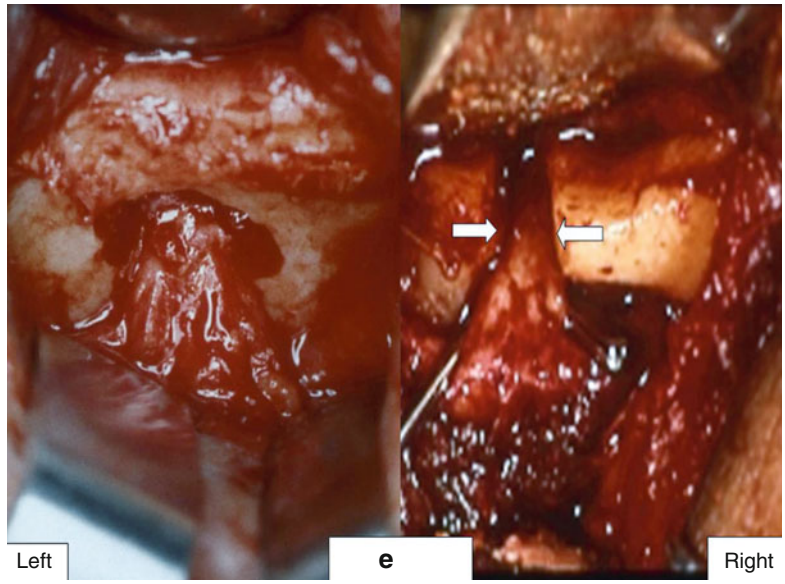


Fig. 3.10 Examples of fractures involving branches of the trigeminal nerve: (**a**) fracture of posterior body of left mandible has minimum offset of inferior alveolar canal (IAC, *arrows*) and low risk of permanent IAN injury; (**b**) grossly displaced fractures of right and left mandible through IACs (*arrows*) may cause stretching or severance injury of IAN; (**c**) gunshot wound of left mandible (*left*) with missile penetration of the IAC resulting in avulsion of a segment of IAN (*right*); (**d**) malunion of left mandibular angle fracture treated by osteotomy and decompression of IAN (*arrows*); (**e**) *left*, healed untreated orbital floor and inferior orbital rim fractures causing entrapment, compression, and scarring of infraorbital nerve (IFN); *right*, infraorbital foramen and inferior orbital canal unroofed (*arrows*) for decompression of IFN

Fig. 3.10 (continued)



Data combined from several studies included 462 patients who had ZMC fractures [121]. In these patients, 65–100 % had posttraumatic/pretreatment NSD of the IFN. After fracture treatment, these numbers had decreased to 7.7–55 %. Long-term follow-up indicated that 171 (37 %) of these 462 patients had permanent NSD of the IFN.

The final outcomes of many of these patients who in the past sustained maxillofacial trauma and were left with permanent NSD of the IAN or ION are certainly less than ideal. The reasons might include needful delay in assessing and treating the facial and associated peripheral nerve injuries because of the priorities in stabilizing the patient and treating life-threatening conditions first, lack of training of surgeons in management of peripheral nerve injuries associated with maxillofacial injuries or lack of ready availability of such expertise locally, or patients' satisfaction with their final status of recovery from what might have been life-threatening injuries. Some surgeons contend that reducing the fracture into anatomic alignment also restored a natural conduit (IAC for the IAN, inferior orbital canal for the IFN) that serves as a guide for nerve regeneration, and this was considered adequate treatment. However, restoration of neurological function has now become a specific treatment goal in the care of patients with maxillofacial fractures, according to AAOMS [48]. More patients have begun to seek treatment for the residual sensory dysfunction that is often a continuing reminder of their facial injuries, now they are aware that something can be done. A recent retrospective study reviewed 42 patients who had undergone microsurgical repair of TN5 nerves (IAN, 21; MN, 12; IFN, 7; LN and LBN, 1 each) injured as a result of maxillofacial trauma [10]. After a follow-up of at least 1 year, neurosensory testing showed that, according to the Medical Research Council Scale (MRCS) [22], 23 nerves (55 %) had regained "useful" sensory function, 13 nerves showed full sensory recovery, and 6 nerves (14 %) showed little or no sign of recovery, for an overall success rate of 86 %. These results compare favorably with those of microsurgical repair of TN5 injuries from other causes [11–14], and they establish this type of microneurosurgical intervention as an acceptable treatment modality in selected patients.

Timing is critical in successful microsurgical repair of all peripheral nerve injuries. In most clinical studies, the best results are achieved when the nerve is repaired within 6 months of injury [10, 12–14, 36, 79, 96, 112, 117, 136]. Although repair of an observed or suspected nerve injury is not routinely delayed that long, there may be valid reasons or extenuating circumstances for postponing nerve repair in a patient who has sustained multiple injuries.

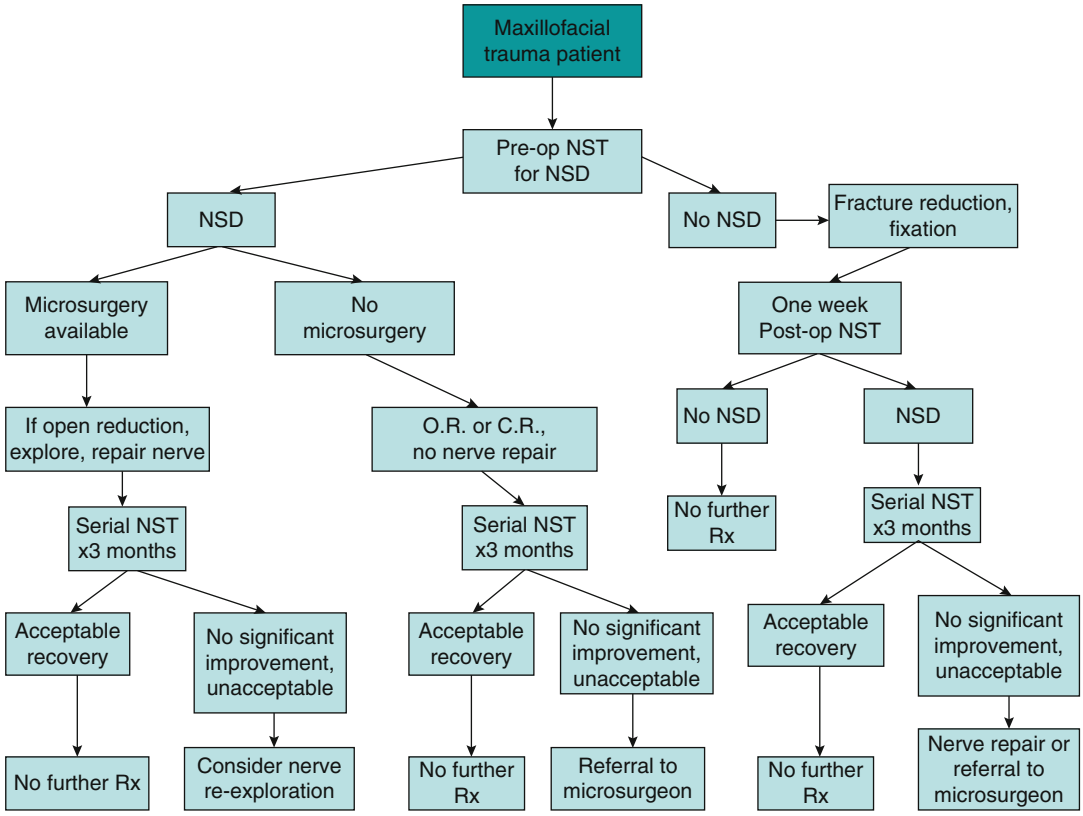


Fig. 3.11 Method of evaluation and treatment for maxillofacial trauma patients with peripheral trigeminal nerve injuries. See text for discussion. *NSD* significant neurosensory deficit (i.e., moderate hypoesthesia to anesthesia), *NST* neurosensory testing, including responses to pain, static light touch, and two-point discrimination, *O.R.* open reduction of fracture, *C.R.* closed reduction of fracture, *Rx* treatment

These include (1) gross contamination of the wound (especially prevalent in combat injuries), (2) poor patient physical status due to multiple system injuries making him/her a poor risk for additional anesthesia and surgery after life-threatening conditions are stabilized, and/or (3) the surgeon in charge of management of the maxillofacial injuries does not have microsurgical training or it is not readily available. In such cases, the microneurosurgery is delayed until the wound is free of infection, the patient's physical status has improved, and a surgeon trained in microsurgery becomes available. Such delays are acceptable and usually amount to only days or weeks [79]. Longer delay in repairing nerve injuries may occur if the injury is not suspected or recognized, the patient desires no further treatment for NSD that is judged "acceptable," or the patient is lost to follow-up.

The algorithm shown in Fig. 3.11 will assist the clinician who manages maxillofacial trauma in the evaluation and treatment of associated TN5 injuries. Patients who have sustained maxillofacial injuries and are conscious and able to cooperate should undergo a cranial nerve screening, including neurosensory testing (NST) of the TN5 (see Chap. 10). If *no* NSD of the major branches of the TN5 (IAN, IFN, SON) is found, the facial fractures are reduced and fixated as needed. Follow-up NST is done within 1 week postoperatively. If no TN5 NSD is present at 1 week after fracture repair, no additional nerve follow-up is necessary at that time. The patient is advised, however, that if a sensory aberration develops within the next several months, another evaluation is advised *at that time* (see delayed onset of NSD, discussed in this section above). If the patient has significant NSD at 1 week postoperatively,

follow-up NST is done serially for 3 months. If the patient's sensory function has recovered within that time frame, no treatment is needed. If, however, acceptable recovery of NSD has not occurred by 3 months following fracture repair, exploration and microsurgical repair of the injured nerve, or referral to a microsurgeon, should be considered.

If the patient *has* a significant TN5 sensory deficit following his facial injuries, the fractures are repaired. When *microsurgical repair is not readily available*, closed or open reductions of the fractures are performed as indicated. If the surgeon observes a nerve injury (crush, severance, or avulsion) during the fracture repair, the area of the nerve injury should be marked with one or two fine (6-0 or 8-0) nylon sutures, the nerve placed in as normal alignment as possible, and mention made in the operative report of the location and nature of the injury. The patient is followed postoperatively for 3 months with serial NST. If the NSD has resolved or is acceptable to the patient, no further treatment is necessary. If the NSD has not recovered to an acceptable level at 3 months post-injury, the patient is referred to a microsurgeon for further evaluation, and a decision is made regarding the necessity for another operation to repair the nerve. When the patient's maxillofacial fractures are being evaluated and treated by *a surgeon with microsurgical skills*, the fractures are also treated as indicated. If an open reduction is performed, the nerve is exposed, its nerve canal enlarged to compensate for post-injury osseous proliferation, and repaired as indicated. If a closed reduction is performed, the nerve will not be directly observed in most cases. In either situation, this patient is followed with serial NST for 3 months. If the patient has recovered acceptable sensory function, no further treatment is indicated. On the other hand, if the NSD is unacceptable after 3 months, re-exploration of the nerve should be considered. Adherence to these recommendations will more likely afford patients with significant TN5 NSD the greatest likelihood of regaining "useful sensory function" [10, 84].

Methods of fracture repair are modified when they encroach upon adjacent nerves, in order to minimize or avoid iatrogenic injury. Manipulation for satisfactory reduction of fracture segments should be done carefully to avoid excessive stretching or compression of involved nerves. Decompression by removing adjacent bone or enlarging the nerve canal may prevent a "closed box" phenomenon by creating additional space for temporary posttraumatic/postoperative edema of the nerve. Also, this may compensate for the delayed effect of osseous proliferation and canal narrowing that may occur during postoperative fracture healing (see above). Using monocortical, rather than bicortical, fixation screws and not placing them adjacent to, or into, bony nerve canals are always desirable considerations in fracture management. Internal fixation plates are placed so as not to encroach on nerves exiting from bone (i.e., the SON, IFN, MN). Nerve repair that is not performed at the time of fracture treatment is delayed for 6 weeks (for a ZMC fracture) to 6 weeks (for a mandibular fracture). This amount of time allows the fracture to become clinically stable and the inflammatory response to have resolved. Bleeding is less troublesome, and epineurial tissue will have thickened and lost its friability, making visualization, debridement, and suturing much easier. This time delay allows the zone of neural damage to declare itself so that adequate resection of neuromatous tissue can be performed by visual inspection under magnification. The recovery of neurosensory function will not be compromised by this prudent delay and, in fact, may be improved when compared to recovery after immediate repair [59, 79].

Although the SON and the STN must necessarily be involved when the supraorbital region is injured in military combat and other missile actions, MVA, interpersonal violence, or household accidents, this type of paresthesia has been mentioned rarely in published studies. NSD in the forehead region may be omitted on initial evaluation because of immediate concern for life-threatening injuries, it may resolve spontaneously, the patient may not be afflicted with significant symptoms, and/or the deficit does not interfere with normal facial functions, or it is simply underreported. In any case, SON injuries due to maxillofacial trauma, though seldom reported, do occur (Fig. 3.12).

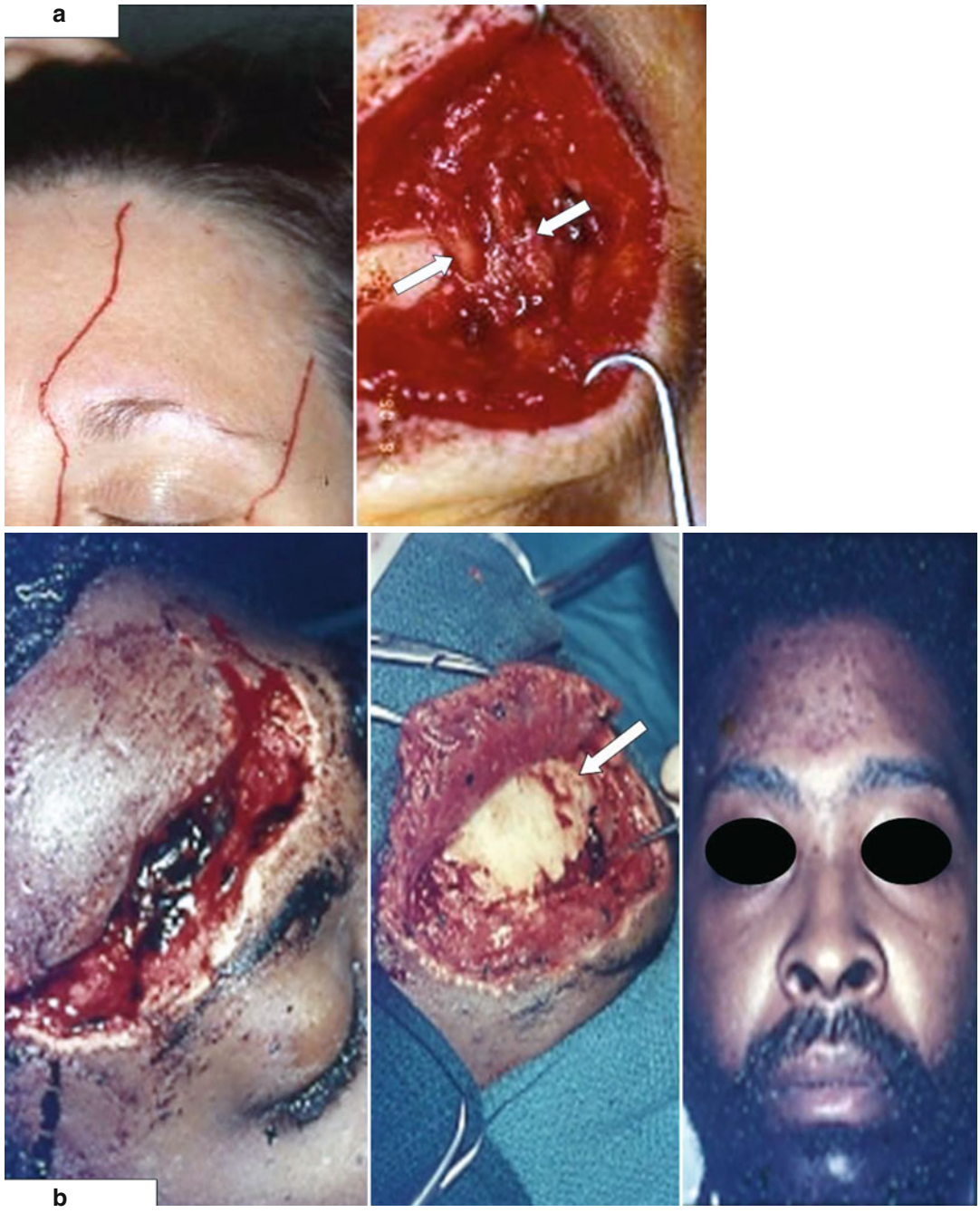


Fig. 3.12 Injuries to supraorbital nerve (SON): (**a**) patient sustained blunt trauma to left forehead (*left*). She developed pain, numbness, and hyperesthesia in the area outlined. *Right*, exploration revealed injured branches of the left supraorbital nerve, each with a neuroma-in-continuity (*arrows*). Repair was done by excision of neuromas and reconstruction of each branch with autogenous great auricular nerve graft; (**b**) unrestrained passenger in motor vehicle accident struck forehead on dashboard. Laceration transected right supraorbital (SON) and supratrochlear (STN) nerves (*left*); depressed frontal fracture (*arrow*) required surgical reduction (*middle*); patient satisfied with postoperative status and refused exploration/repair for SON/STN sensory loss (*right*)

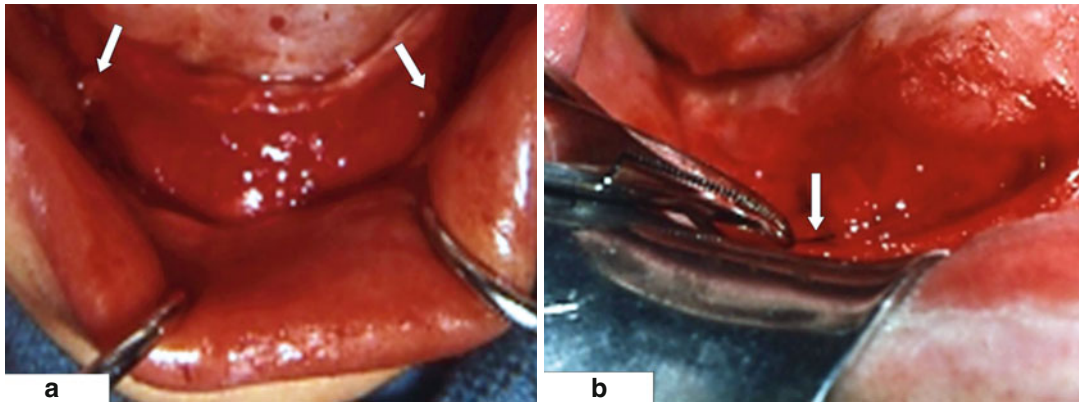


Fig. 3.13 Mandibular vestibuloplasty (MVest): (**a**) exposure of both mental nerves (MNs) (indicated by *white arrows*) during supraperiosteal dissection prior to placing split-thickness skin graft; (**b**) patient who developed persistence numbness after MVest was found on re-exploration to have a retained nylon suture (indicated by *white arrow*) around right MN. Removal of suture was followed by recovery of MN sensation

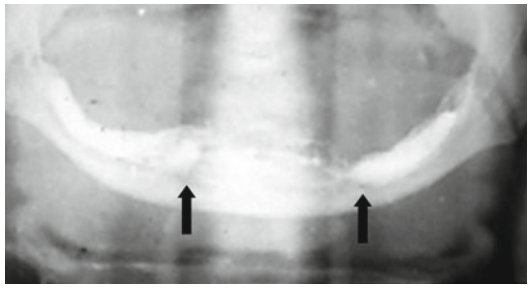


Fig. 3.14 Severely atrophic mandibular ridge was augmented with calcium hydroxyapatite (CHA). CHA was in direct contact with both mental nerves (*arrows*), producing anesthesia and constant pain in MN distribution. Both MNs were explored and found to have pathologic changes consistent with a chemical burn

3.5.5 Dental Implants and Preprosthetic Surgery

In the past, "preprosthetic" operations to alter interfering soft tissue attachments, deepen the vestibule of an edentulous ridge, or augment the residual alveolar ridge that had undergone excessive resorption following the loss of teeth were the only surgical options available to improve conditions of retention and stability for dental prostheses [45, 50, 75, 116]. In the mandible, performance of a vestibuloplasty procedure necessarily required supraperiosteal soft tissue dissection with risk of injury to the MN (Fig. 3.13). If a ridge augmentation were required utilizing an osteotomy with placement of a bone graft or alloplastic material, the IAN or MN might be at risk for injury [15]. While calcium hydroxyapatite (CHA) has been placed safely around the MN in ridge augmentation procedures [62], in the senior author's experience, CHA was found to produce a chemical burn in some patients when it was placed in contact with the MN (Fig. 3.14). This untoward outcome sometimes improved spontaneously over time, but in some unfortunate patients, it was prolonged or permanent, often associated with pain or hyperesthesia, and it served as a deterrent to many prospective patients needing preprosthetic surgery. With the current dental implant options, these procedures are seldom performed today.

The development and introduction of dental implants [26] has revolutionized the replacement of individual missing teeth and restoration of lost dentition in free-end saddles and totally edentulous dental arches. Despite the availability of improved imaging studies, careful treatment planning, modified surgical techniques, special instrumentation, and the application of surgical skills, injuries to the IAN and the MN can, and do, occur during osseous drilling and implant fixture placement [18, 66, 78]. The incidence of temporary nerve injury has been reported in several case studies varying between 1.7 and 43.5 %. Long-term or permanent (greater than 1 year) NSD was found in 0–15 % of patients. The larger the number of patients, the

lower the rate of nerve injury reported, perhaps indicating the value of surgical experience in reducing the potential for nerve injury. There are many theories regarding IAN injury from dental implant placement other than direct injury due to imprecise determination of the amount of available bone above the canal. One explanation is that intracanal bleeding from an inferior alveolar vein or artery injured during bone preparation may create a "compartment syndrome" within the inferior alveolar canal which compresses the IAN. This would explain the fact that many of these implant-related nerve injuries result in unpleasant dysesthesia rather than solely hypoesthesia or anesthesia. For a complete discussion of this topic, the reader is referred to Chap. 6.

3.5.6 Endodontic Treatment

The onset of persistent numbness or pain following completion of root canal treatment of a mandibular molar or premolar tooth is especially distressing to the patient who expected salvage of that tooth as the primary outcome. The instrumentation necessary to remove necrotic tissue from the pulp canal of a non-vital posterior mandibular tooth and smooth or enlarge its walls and the substances used to medicate and fill the canal can injure the underlying IAN [17, 67, 87, 91, 98]. The incidence of IAN injury associated with endodontic treatment has not been determined, since the only reports in the literature are single case reports or small series of fewer than ten patients. Many of these patients had their root canals filled with Sargenti (N2) paste, a substance that contains paraformaldehyde which has been shown to be toxic to nerve tissue. When N2 paste is injected into the prepared canal under pressure, it has the potential to flow beyond the root apex in the periapical area and thence, particularly if the root canal has been overinstrumented, come into contact with the IAN.

The mechanisms of nerve injury that might result in NSD of the IAN from endodontic treatment include (1) *direct trauma* from overinstrumentation, (2) *compression* from overfilling of the root canal with extrusion of inert filling material into the IAC, and (3) *chemical injury* [33]. This is particularly prone to occur, if the IAC is in close proximity to the root apex. Endodontic broaches or files passed beyond the apex might enter the IAC and pierce the IAN, causing its internal disruption and partial or complete severance. Some medicaments and materials used to irrigate, sterilize, and fill the canal might be inert (e.g., normal saline) when in contact with nerve tissue and/or only produce compression (e.g., gutta-percha, zinc oxide), if allowed to enter the IAC. On the other hand, many root canal cements contain derivatives of phenol (such as eugenol) or other substances (e.g., calcium hydroxide, paraformaldehyde), and sterilizing solutions may consist of parachlorophenols, paraldehydes, or other agents such as sodium hypochlorite and antibiotics, all of which may be toxic to nerve tissue and capable of producing a chemical burn if allowed to make contact with a nerve.

The patient who has sustained an IAN injury during endodontic treatment may experience immediate onset of pain and/or loss of sensation. In such a case, it may be concluded that direct contact with the nerve was made during the procedure by overinstrumentation and/or extrusion of root canal filling cement or filling material beyond the confines of the root canal into the periapical region and thence into the IAC. If there is pain, it is often intense, prostrating, and difficult to control with opioid analgesics. In some patients, addition of a neurotropic medication (e.g., clonazepam 0.5–2.0 mg. every 8 h) will provide adequate pain relief until surgical intervention is begun. In other instances, after the effect of local anesthesia administered for the procedure has worn off, there may be a return of normal sensation (the so-called lucid interval), and only in one to several days later does the patient experience the onset of pain and altered sensation [98]. This is thought to be due to delayed percolation of toxic materials into the IAC that have leaked out the root apex of an overinstrumented canal. In either situation, the symptomatic patient requires immediate attention (Fig. 3.15). Imaging studies (plain films or CBCT) will demonstrate whether filling material has extruded beyond the confines of the root canal and if there is involvement of the

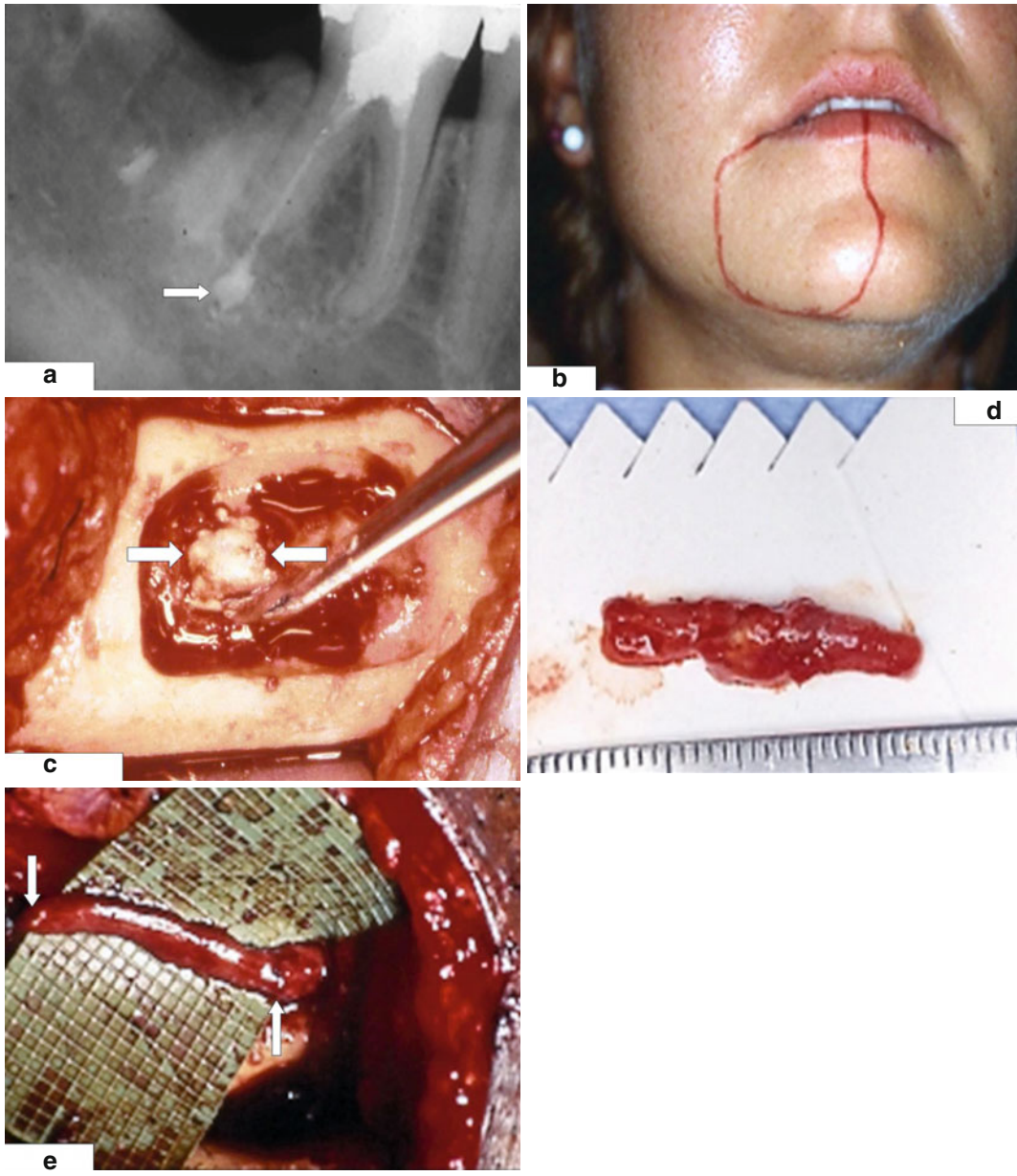


Fig. 3.15 Inferior alveolar nerve (IAN) injury associated with root canal (RC) treatment: (**a**) overfilled mandibular right first molar with radiopaque material (*arrow*) extruded into inferior alveolar canal; (**b**) the patient presents with pain and numbness in the distribution of the right IAN (outlined in *red*); (**c**) surgical exploration shows extruded filling material (*arrows*) in contact with IAN; (**d**) resected 2.5 cm segment of IAN had sustained chemical burn from contact with extruded RC material; (**e**) the IAN was reconstructed with autogenous sural nerve graft (*arrows*)

IAC. If so, the patient should be scheduled as soon as possible in the hospital operating room under general anesthesia for microsurgical exploration and debridement of the IAC and repair of the IAN as indicated by surgical findings. The IAC is approached either transorally or through a submandibular cutaneous incision, depending on the ease of exposure, as dictated by the location of involvement of the IAN. In some patients, the IAN will be found to be compressed by impinging root canal filling material. All extruded material surrounding the IAN is removed. If it appears

that material has breached the nerve itself, the epineurium is entered through an axial incision. Under magnification, the fascicles are identified, and a thorough intraneural debridement is done, often a tedious task with the potential for intraneuronal scarring limiting full neurosensory recovery, followed by copious saline irrigation. If the nerve has sustained a chemical burn, the epineurium may appear thickened and chalky white, rather than with its normal translucent sheen appearance. If a segment of the nerve appears to have sustained a chemical burn, it is important to accurately determine the line of demarcation between necrotic and viable tissue [79], and this may not be apparent for several days to weeks following the injury. Then, the damaged nerve tissue is resected so that normal fascicular tissue is present in the proximal and distal nerve stumps and the IAN is then reconstructed in the usual manner (see Chap. 14).

A nerve injury from endodontic treatment can be a serious emergency for the patient whose principal symptom is pain. Every effort should be made to avoid or minimize the occurrence of this complication. The practitioner is advised to ascertain an accurate estimate of the root length from a standardized radiograph. Instruments should be armed with stops at the determined distance to avoid overinstrumentation beyond the root apex. Root canal filling materials should not be inserted or injected under pressure. A radiograph should be taken immediately upon completion of treatment to assess the location of filling material. If there is evidence of overfilling of material with encroachment on the IAC, the patient should be referred immediately for microsurgical consultation. When performing apical surgery on a mandibular premolar or molar tooth, the location of the mental foramen and the IAC should be determined and care taken to avoid these areas or use suitable gentle retraction of any nerve branches in the area of the procedure on the root apex.

The development of the dental specialty of endodontics [58], closer scrutiny of the toxicity of root canal filling materials [4], the modification of techniques, and the introduction of magnification to endodontic treatment have greatly diminished the case load of endodontically associated TN5 injuries in microsurgical practice.

3.5.7 Salivary Gland Surgery

Surgical operations on the submandibular salivary duct and the submandibular and sublingual salivary glands are frequently required to treat tumors, ranulas, sialolithiasis, obstruction, acute and chronic infections, and end-stage salivary gland dysfunction [28, 29]. Such operations, whether they are performed through transoral or submandibular cutaneous approaches, will often involve the LN as it courses medial to or even through the submandibular gland and in close proximity to the sublingual gland and submandibular salivary duct in the floor of the mouth (see Sect. 3.2, above). Although the incidence of NSD of the LN following salivary gland surgery is not known, occasional cases are seen. Patients present with varying complaints of tongue numbness, pain or hypersensitivity, and altered taste sensation, all of which are often quite distressing and interfere with normal oral functions.

In order to minimize the risk of LN injury during surgery on salivary structures, the surgeon should be proactive. When the sublingual or submandibular gland is to be excised, a maneuver that is helpful in identifying the facial nerve during parotid gland surgery can be employed. At the beginning of the operation, the submandibular salivary duct is dilated, cannulated, and injected with 1–2 mL of an inert dye such as methylene blue. When the salivary gland (sublingual or submandibular) to be operated upon is exposed, it will be stained an intense blue color. This technique makes it easy to locate the LN, which will retain its usual translucent opalescent appearance in contrast to the stained blue color of the gland. In operations upon the submandibular duct, one is advised to maintain a cannula in the duct while opening or dissecting within it in order to maintain perspective with the rest of the floor of the mouth. Confining instruments to within the duct while removing a stone will avoid their contact with the nearby LN.

As mentioned above (see Sect. 3.2), the ATN is at risk during parotidectomy. The development of Frey's syndrome (syndrome of gustatory sweating) with preauricular flushing and sweating during mastication of food is thought to be due to abnormal reconnections of postganglionic parasympathetic fibers of the ATN which supply

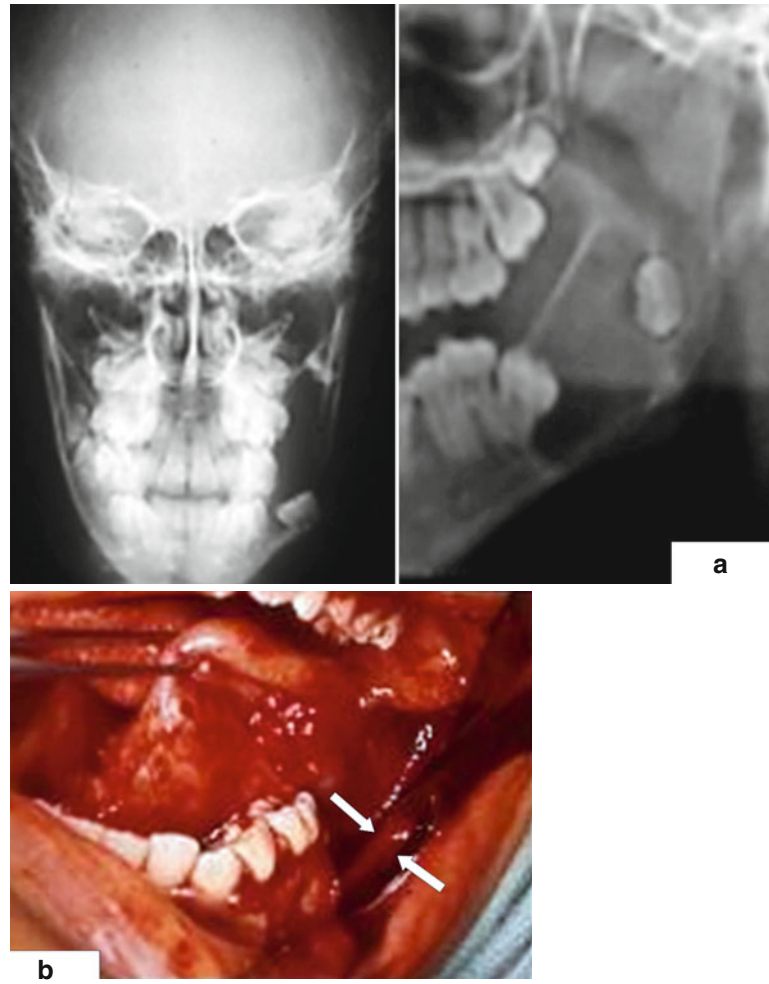


Fig. 3.16 Large dentigerous cyst of left mandible: (**a**) preoperative radiographic views; (**b**) cyst and associated teeth have been successfully dissected away from the left inferior alveolar nerve which is seen to be intact (*arrows*)

the parotid gland with severed sympathetic nerve branches that stimulate subcutaneous sweat glands. The incidence is unpredictable and is reported to range from 2.6 to 97.6 % in various studies. This complication can be prevented by interposition of various types of soft tissue (fat, temporalis fascia, fascia lata femoris, dermis, and myocutaneous flaps) beneath the skin flap, and the problem can also be treated with injections of botulinum toxin [44].

3.5.8 Ablative/Oncologic Surgery

Large cysts and benign tumors of the mandible often involve the IAN, and the surgeon is then faced with a decision regarding surgical management of the nerve as well as the lesion. In the case of dentigerous, or other types of odontogenic cysts, without malignant or invasive potential, generally the cyst can be carefully dissected away from the IAC contents, sometimes aided by magnification and microsurgical instruments (Fig. 3.16). Such careful technique may result in immediate temporary paresthesia of the IAN in the early postoperative period, but this often resolves over the course of several months. Occasionally, the nerve is *unintentionally* partially or completely transected during removal of the cyst. If this is observed by the surgeon at the time of its occurrence and the surgeon has microsurgical expertise, the nerve can be surgically repaired at that time. If not, the nerve ends are tagged with fine, nonreactive sutures (i.e., 6-0 or 8-0 nylon), the nerve ends are placed in as close approximation as possible, and a note describing the nature and location of the nerve is included in the operative report. Subsequently, the patient

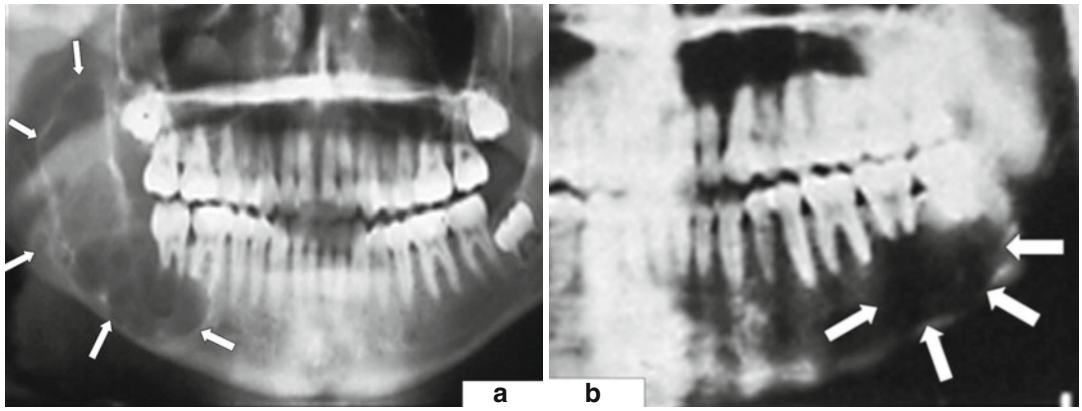


Fig. 3.17 Locally aggressive tumors can involve the inferior alveolar nerve, necessitating its resection along with the tumor: (**a**) large, multiloculated ameloblastoma of right mandible (indicated by *white arrows*); (**b**) myxoma of left mandible (indicated by *white arrows*)

is given a *timely* referral to a microsurgeon for follow-up and possible delayed primary nerve repair, typically 3 weeks following the injury.

Locally aggressive benign tumors of the mandible, such as the ameloblastoma and myxoma, have a high rate of recurrence if not excised with adequate margins [128] (Fig. 3.17). Since there is a question whether such tumors actually invade an adjacent nerve, some clinicians have advocated preservation of the IAN when excising these tumors [19, 122]. However, in an effort to maximize the likelihood of a curative result without recurrence due to inadequate removal, an *intentional* resection of the IAN is included with the surgical specimen in most surgeons' hands. Following mandibular resection (including sacrifice of the IAN) in patients younger than 16 years, spontaneous return of partial IAN sensation to which the patients adapt well has been noted [30]. However, the IAN is often reconstructed immediately with good return of sensation in many patients [93]. Similarly, excision of nerve tumors such as the neurilemmoma (schwannoma) requires resection of the involved nerve and its reconstruction with a nerve graft.

Altered sensation (pain, numbness, loss of sensation to neurosensory testing) is an important clinical symptom and sign of malignancy in a tumor that approximates a sensory nerve such as the TN5. Malignant tumors are well known for their propensity to invade nerves (neurotropism) and use them as a route for spread of malignant cells [72]. Therefore, the IAN is *always* intentionally sacrificed when the mandible is resected for treatment of malignancy.

3.5.9 Cosmetic Surgery

Operations to improve appearance of the chin (the genioplasty) are among the most frequent in facial cosmetic surgery. In the past, contour deficiency corrected by insertion of an alloplastic implant was the favored esthetic operation on the chin [85], and it still is in many situations [35]. However, the development and addition of the anterior horizontal sliding mandibular osteotomy to the orthognathic and facial cosmetic surgeon's repertoire provided a versatile operation that could be utilized in the correction of deficiency of chin contour, objectionable chin prominence, excessive or inadequate chin height, and asymmetry, especially in combination with operations to correct developmental facial bone deformities and concomitant dental malocclusion [73].

Various studies have reported on involvement of the IAN or MN and postoperative NSD in the lower lip and chin following horizontal mandibular osteotomy for genioplasty [38, 92, 107, 124]. Immediately following surgery, most patients experience decreased or absent responses to pain, static light touch, and/or two-point discrimination. When done as a solitary procedure, patients usually regain most or all of their sensory function in the lower lip and chin (Table 3.2). When the genioplasty is done in conjunction with the MSSRO (see Sect. 3.5.3, above), however, there seems to be an exponential additional effect on NSD. For instance, in one series of 115 adolescent patients who underwent surgical correction of dentofacial deformities, the incidence of long-

term NSD in the IAN and/or MN was 10 % for patients having a genioplasty only, 20 % following bilateral MSSROs, and 67 % for those having *both* MSSROs and genioplasty [107]. This has been described as the "double crush syndrome" and indicates that, at least in some instances, the patient who undergoes MSSROs *and* genioplasty at the same operation will have greater loss of sensory function in the IAN and MN distribution than in patients that have either procedure alone [38, 107, 123, 124].

The MN is at risk during the creation of a pocket for insertion of an alloplastic implant through a submental skin incision because the surgeon seldom can visualize the nerve directly or very well at all. The location of the mental foramen can be determined preoperatively from a panoramic radiograph of the mandible, and the surgeon can plan to avoid this area when creating the soft tissue pocket. An implant size is selected which does not impinge upon the mental foramen when seated into place. When raising a mucoperiosteal flap to expose the facial aspect of the mandibular symphysis for a horizontal osteotomy, the dissection is done carefully until the MN is identified on each side. The exit of the MN from the mandible is at a level several millimeters superior to that of the IAC (see Sect. 3.2, above). This vertical distance is variable and is determined from preoperative imaging studies. The horizontal osteotomy must be made sufficiently inferior to the anatomic mental foramen to avoid contact with the anterior loop, or genu, of the IAN (Fig. 3.18).

Additional cosmetic facial operations that might affect branches of the TN5 include the

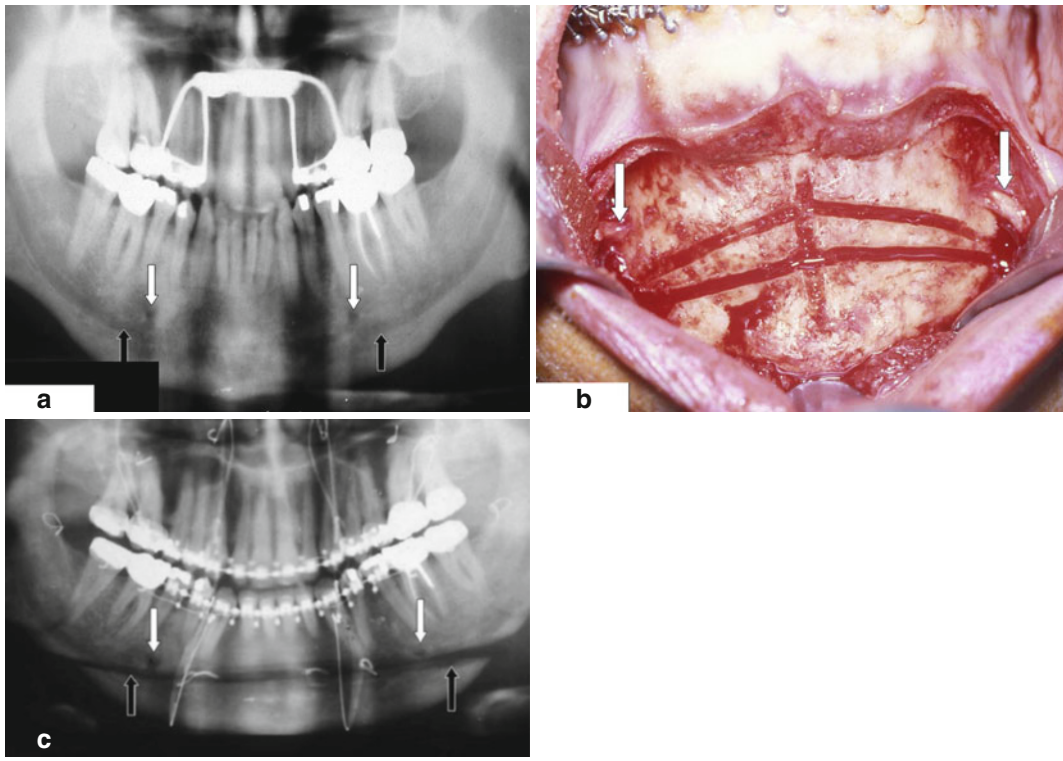


Fig. 3.18 Relationship of mental foramen (MFN) to inferior alveolar canal (IAC): (**a**) preoperative x-ray shows MFNs (*white arrows*) are several millimeters superior to IACs (*black arrows*); (**b**) osseous markings made for osteotomies for reduction of chin height and chin advancement are located inferior to mental nerves (*white arrows*); (**c**) postoperative film shows that horizontal sliding osteotomy, although located inferior to both MFNs (*white arrows*), has traversed through both IACs. Both inferior alveolar nerves (*black arrows*) were transected. They were successfully repaired microscopically 3 months after injury

facelift (rhytidectomy) and the brow/forehead lift. The facelift procedure commonly involves the auriculotemporal nerve (ATN), a branch of the V3 of TN5. Seldom is sensory loss in the preauricular or temporal areas permanent or mentioned as a problem by the patient [35]. Of more concern is the risk of injury to the FN7 with weakness or paralysis of facial and/or eyelid musculature, a subject not within the scope of this discussion on trigeminal nerve injuries. When planning an open forehead and brow lift, the surgeon should place skin incisions well into the hair-bearing scalp to avoid the superficial branches of the SON and thereby maintain forehead sensation and to preserve the deep division of this nerve and scalp sensation by not carrying the incision through the galea aponeurotica [65]. The introduction of endoscopic procedures has undoubtedly lessened the incidence of permanent forehead or scalp numbness or other undesirable sensory aberrations in patients undergoing eyebrow and forehead lifts [34].

3.6 Summary

Surgical procedures, routine dental treatments, and traumatic injuries in the face and oral cavity occur in close proximity to peripheral branches of the trigeminal (fifth) cranial nerve, the major sensory supply to this important area. Despite the best of care, trigeminal nerve injuries are recognized and accepted risks of surgical operations, dental treatment, and injuries in the oral and maxillofacial regions. However, lost or altered sensation resulting in numbness, pain, or hypersensitivity seriously interferes with common orofacial functions and, if persistent, is often distressing and unacceptable to patients so afflicted. The incidents associated with peripheral trigeminal nerve injuries, the likelihood (incidence) of their occurrence, and the potential mechanisms causing them have been presented, and suggestions have been proposed for reducing the risk of injury associated with specific situations. In the chapters to follow, the treatment of trigeminal nerve injuries will be thoroughly presented.

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