Atlas of PAIN MANAGEMENT INJECTION TECHNIQUES

FOURTH EDITION

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To David Mayo, Corey William, Jenifer Elyse, and Reid Alexander…
You are amazing!

Dad

PREFACE

Well over 130 years have passed since Carl Koller first used cocaine to perform an eye operation without pain. Although Koller's landmark discovery forever changed how surgery is performed and unwittingly created a cottage industry in regional anesthesia drugs, needles, and, of course, regional anesthesia textbooks, a careful analysis of the ensuing years reveals that most of the advances in regional anesthesia drugs have centered around the development of safer local anesthetics and improved needles. The landmark texts by Pitkin, DeJong, Moore, and others helped make the use of regional anesthesia drugs accessible to the general practitioner. These early books standardized techniques, needle sizes and lengths, and, perhaps most importantly, the dosages of local anesthetics for the common techniques. Lofgren's discovery of lidocaine in 1943 moved regional anesthesia drugs into the operating room and obstetric suite, as well as into doctors' and dentists' offices. Much safer than local ester anesthetics such as procaine, the most widely used local anesthetic up to that time, lidocaine's larger therapeutic window was much more forgiving of clinical missteps when nerve blocks were performed, and lidocaine has been the mainstay of regional anesthesia drugs ever since.

In the 1980s, pain medicine came into its own as practitioners tested and applied the simple hypothesis that the cause of pain must be diagnosed first to ensure a successful treatment. Along with the birth of this new subspecialty came a new set of "bibles" by Raj and by Cousins and Bridenbaugh and the first edition of *Atlas of Pain Management Injection Techniques,* which was first published in 2000.

I think that the field of pain management is entering a new and exciting era now that ultrasound guidance has become increasingly utilized when regional anesthesia drugs are administered. Only time will tell whether ultrasound guidance is a true "moment" or merely a passing fancy that will go the way of nesacaine and succinylcholine drips, but my clinical impression is that ultrasound guidance represents an important advance in regional anesthesia and pain management that I think will stand the test of time. In addition to presenting the fundamentals of ultrasound guidance in the how-to-do-it format that has made previous editions of this book so popular, I have included many new color figures and ultrasound, fluoroscopic, and magnetic resonance images, along with new full-color, clinically relevant anatomic drawings to make the techniques described even more accessible to my readers. With the able assistance of the editorial team at Elsevier, we have added clear, concise captions to every figure and strived to improve the layout of the book to make it even more readable than previous editions.

I sincerely hope that you will enjoy using this book as much as I enjoyed writing it.

Steven D. Waldman, MD, JD
2016

ACKNOWLEDGMENTS

I would like to thank my editors at Elsevier for their able assistance in the editing and layout of this new edition as well as my friend Dr. Michael Meng for all of his invaluable help with the original ultrasound images included in this new edition.

SDW

1

Temporomandibular Joint Injection

INDICATIONS AND CLINICAL CONSIDERATIONS

Injection of the temporomandibular joint is indicated as an important component in the management of temporomandibular joint dysfunction, in the palliation of pain secondary to internal derangement of the joint, and in the treatment of pain secondary to arthritis of the joint. Temporomandibular joint dysfunction (also known as *myofascial pain dysfunction of the muscles of mastication*) is characterized by pain in the joint itself that radiates into the mandible, ear, neck, and tonsillar pillars. Headache often accompanies the pain of temporomandibular joint dysfunction and is clinically indistinguishable from tension-type headache. Stress is often the precipitating or exacerbating factor in the development of temporomandibular joint dysfunction. Dental malocclusion may also play a role in the evolution of temporomandibular joint dysfunction. Internal derangement and arthritis of the temporomandibular joint may manifest as clicking or grating when the joint is opened and closed and may be easily heard on auscultation of the opening and closing joint (Figures 1-1 and 1-2). Plain radiographs and computerized tomography may help identify arthritic changes, with magnetic resonance imaging useful in identifying articular disk abnormalities (Figure 1-3). If the condition is not promptly treated, the patient may experience increasing pain in the just-mentioned areas and limitation of jaw movement and opening. Recently, the injection of autologous blood and platelet-rich plasma into the temporomandibular joint has gained popularity in the treatment of recurrent temporomandibular joint hypermobility dislocation (Figure 1-4). This injection technique is also useful in the injection of other substances into the temporomandibular joint, such as hyaluronic acid derivatives and tenoxicam.

CLINICALLY RELEVANT ANATOMY

The temporomandibular joint is a true joint divided into an upper and a lower synovial cavity by a fibrous articular disk. In health, the disk and muscles allow the joint, muscles, and articular disk to move in concert (Figure 1-5). The internal derangement of this disk may result in pain and temporomandibular joint dysfunction, but extracapsular causes of temporomandibular joint pain are much more common. The joint space between the mandibular condyle and the glenoid fossa of the zygoma may be injected with small amounts of local anesthetic and corticosteroid. The temporomandibular joint is innervated by branches of the mandibular nerve. The muscles involved in

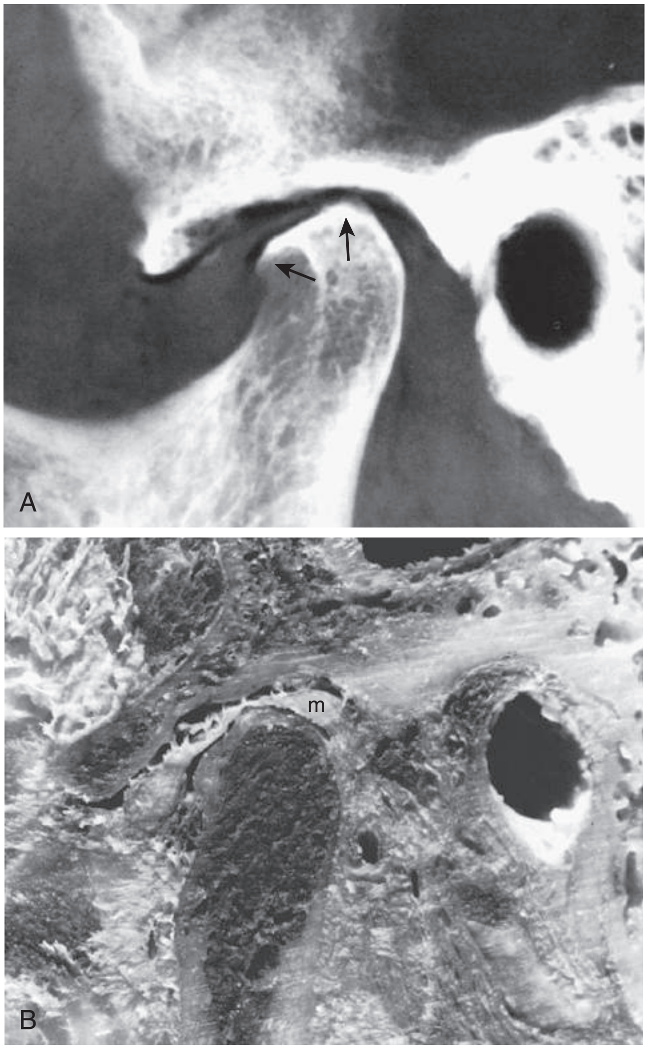

FIGURE 1-1 Osteoarthritis compared in a specimen radiograph (A) and photograph (B) of a sagittally sectioned specimen. The joint space is narrow and the disk is dislocated anteriorly, with thinning and fraying of the meniscal *(m)* posterior attachment or bilaminar zone. The condylar head cortex is thickened, with small osteophytes *(arrows)*. The mandibular fossa is sclerotic and remodeled, and only a shallow concavity is seen where the articular eminence once was. (From Resnick D: *Diagnosis of bone and joint disorders*, ed 4, Philadelphia, 2002, Saunders.)

FIGURE 1-2 Internal derangement and arthritis of the temporomandibular joint may manifest as clicking or grating when the joint is opened and closed and may be easily heard on auscultation of the opening and closing joint. (From Olson KA, editor: Examination and treatment of temporomandibular disorders. In *Manual physical therapy of the spine*, ed 2, St. Louis, 2016, Saunders; Fig. 7-16.)

temporomandibular joint dysfunction often include the temporalis, masseter, and external and internal pterygoid and may include the trapezius and sternocleidomastoid. Trigger points may be identified when these muscles are palpated.

TECHNIQUE

Landmark Technique

The patient is placed in the supine position with the cervical spine in the neutral position. The temporomandibular joint is identified by asking the patient to open and close his or her mouth several times and by palpating the area just anterior and slightly inferior to the acoustic auditory meatus. After the joint has been identified, the patient is asked to hold his or her mouth in a neutral position.

A total of 0.5 mL of local anesthetic is drawn up in a 3-mL sterile syringe. When temporomandibular joint dysfunction, internal derangement of the temporomandibular joint, arthritis pain of the temporomandibular joint, or other painful conditions involving the temporomandibular joint are treated, a total of 20 mg of depot corticosteroid is added to the local anesthetic with the first block, and 10 mg of depot corticosteroid is added to the local anesthetic with subsequent blocks.

After the skin overlying the temporomandibular joint has been prepared with antiseptic solution, a 25-gauge, 1-inch styletted needle is inserted just below the zygomatic arch directly in the middle of the joint space. The needle is advanced ½ to ¾ inch in a plane perpendicular to the skull until a "pop" is felt that indicates the joint space has been entered (Figure 1-6). After careful aspiration, 1 mL of solution is slowly injected. Injection of the joint may be repeated at 5- to 7-day intervals if the symptoms persist.

Ultrasound-Guided Technique

To perform ultrasound-guided injection of the temporomandibular joint, the patient is placed in the supine position with the cervical spine in the neutral position. An imaginary line between the tragus of the ear and the ala of the nose is then

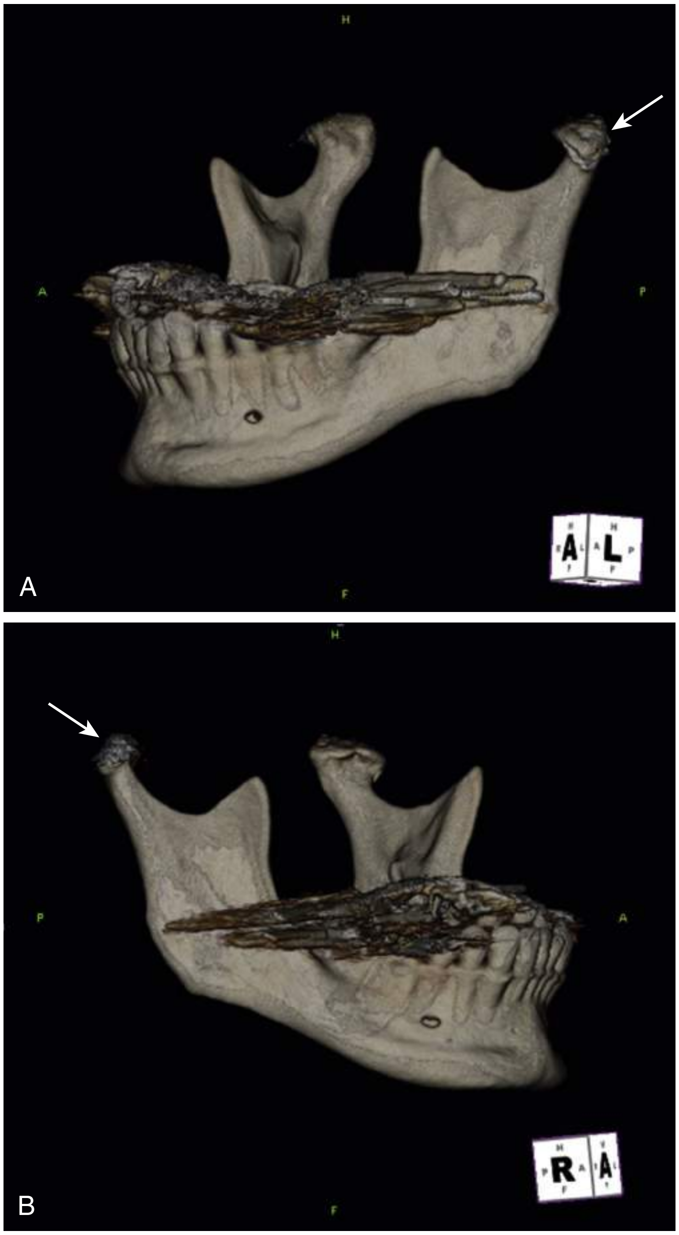

FIGURE 1-3 Computed tomography images acquired on a GE high-density 64-slice scanner (GE Healthcare, Cleveland, OH) in spiral acquisition and reformatted in multiplanar reconstructions. **A,** Left mandible and condyle. **B,** Right mandible and condyle. The bilateral temporomandibular joints show flattening, lipping, and erosion of the condyle, suggestive of degenerative changes. (From Brazelton J, Louis P, Sullivan J, et al.: Temporomandibular joint arthritis as an initial presentation of acute myeloid leukemia with myelodysplasia-related changes: a report of an unusual case. *J Oral Maxillofac Surg* 72[9]:1677–1683, 2014; Fig. 1.)

drawn (Camper's line) (Figure 1-7). The temporomandibular joint is identified by asking the patient to open and close his or her mouth several times and by palpating the area just anterior and slightly inferior to the acoustic auditory meatus. After the joint has been identified, the patient is asked to hold his or her mouth in a neutral position. The skin overlying the mandibular notch is prepped with antiseptic solution and a high-frequency linear transducer is placed directly over the joint in a transverse position. The transducer is then tilted in a cephalad or caudad

FIGURE 1-4 Injection of autologous blood into the temporomandibular joint. (From Daif ET: Autologous blood injection as a new treatment modality for chronic recurrent temporomandibular joint dislocation. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 109:31–36, 2010.)

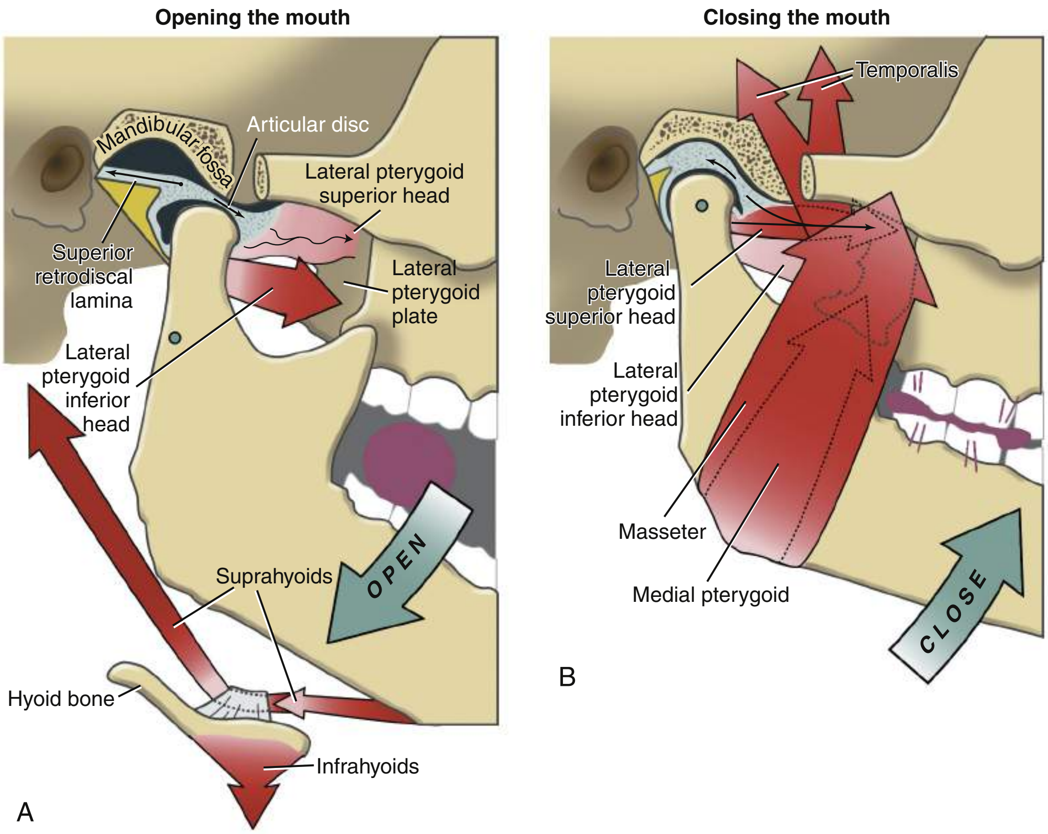

FIGURE 1-5 The muscle and joint interaction during opening (A) and closing (B) of the mouth. The relative degree of muscle activation is indicated by the different intensities of red. In B, the superior head of the lateral pterygoid muscle is shown eccentrically active. The locations of the axes of rotation (shown as small green circles in A and B) are estimates only. (From Neumann DA: Kinesiology of the musculoskeletal system. In *Foundations for physical rehabilitation*, ed 2, St. Louis, 2010, Mosby.)

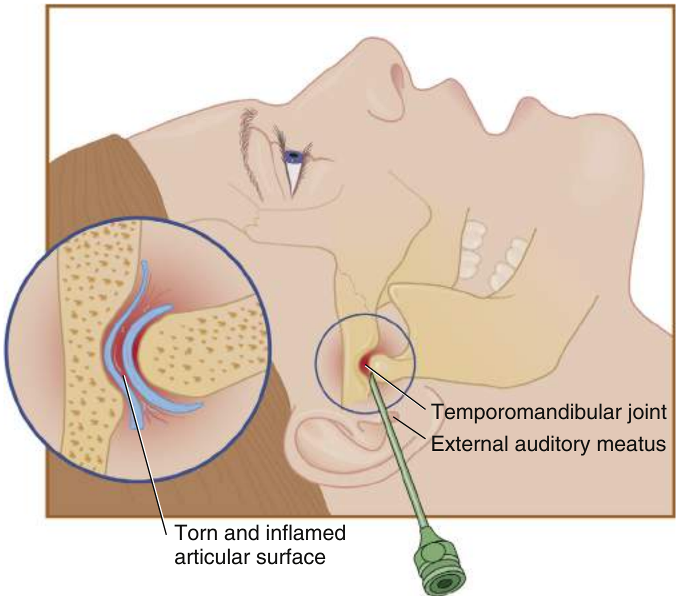

FIGURE 1-6 Needle placement into the temporomandibular joint is simplified by having the patient open and close the mouth to facilitate identification of the joint.

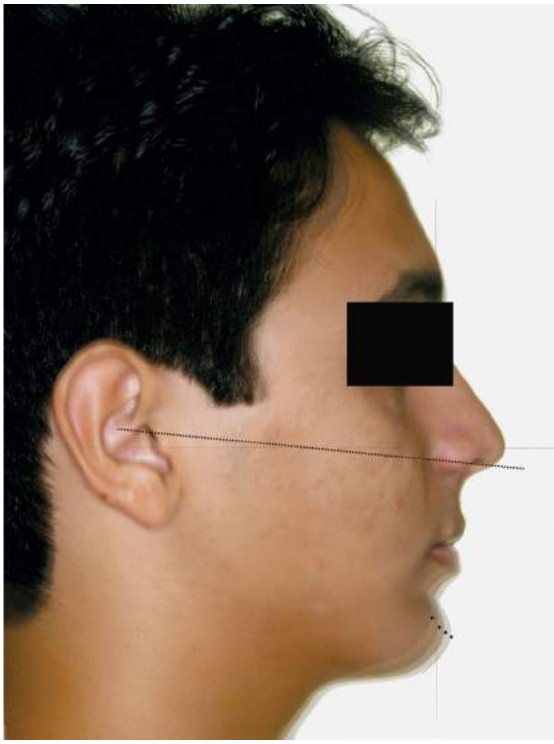

FIGURE 1-7 To identify the temporomandibular joint, an imaginary line between the tragus of the ear and the ala of the nose is drawn (Camper's line). (From Barroso MCF, Silva NCF, Quintão CCA, et al.: The ability of orthodontists and laypeople to discriminate mandibular stepwise advancements in a Class II retrognathic mandible. *Prog Orthod* 13[2]:141–147, 2012; Fig. 3.)

direction to reveal the temporomandibular joint and the curvilinear acoustic shadow of the mandibular condyle and neck just below the joint (Figure 1-8). The articular surfaces are then evaluated for narrowing and arthritic erosions. Then, using dynamic ultrasound imaging, the patient is asked to slowly open and close his or her mouth and the disk is then evaluated for position, reducible disk displacement, and irreducible disk displacement.

SIDE EFFECTS AND COMPLICATIONS

This anatomic region is highly vascular. This vascularity and proximity to major blood vessels also give rise to an increased incidence of postblock ecchymosis and hematoma formation, and the patient should be warned of such. In spite of the vascularity of this anatomic region, this technique can be performed safely in the presence of anticoagulation by using a 25- or 27-gauge needle, albeit at increased risk of hematoma, if the clinical situation dictates a favorable risk-to-benefit ratio. These complications can be decreased if manual pressure is applied to the area of the block immediately after injection. Application of cold packs for 20-minute periods after the block will also decrease the amount of postprocedure pain and bleeding the patient may experience.

Additional side effects that occur with sufficient frequency include inadvertent block of the facial nerve with associated facial weakness. When this occurs, protection of the cornea with sterile ophthalmic lubricant and patching is mandatory.

CLINICAL PEARLS

Pain from temporomandibular joint dysfunction requires careful evaluation to design an appropriate treatment plan. Infection and inflammatory causes including collagen vascular diseases first must be ruled out. When temporomandibular joint pain occurs in older patients, the pain must be distinguished from the jaw claudication associated with temporal arteritis. Stress and anxiety often accompany temporomandibular joint dysfunction; these factors must be addressed and treated. The myofascial pain component of temporomandibular joint dysfunction is best treated with tricyclic antidepressant compounds such as amitriptyline. Dental malocclusion and nighttime bruxism should be treated with an acrylic bite appliance. Narcotic analgesics and benzodiazepines should be avoided in patients with temporomandibular joint dysfunction.

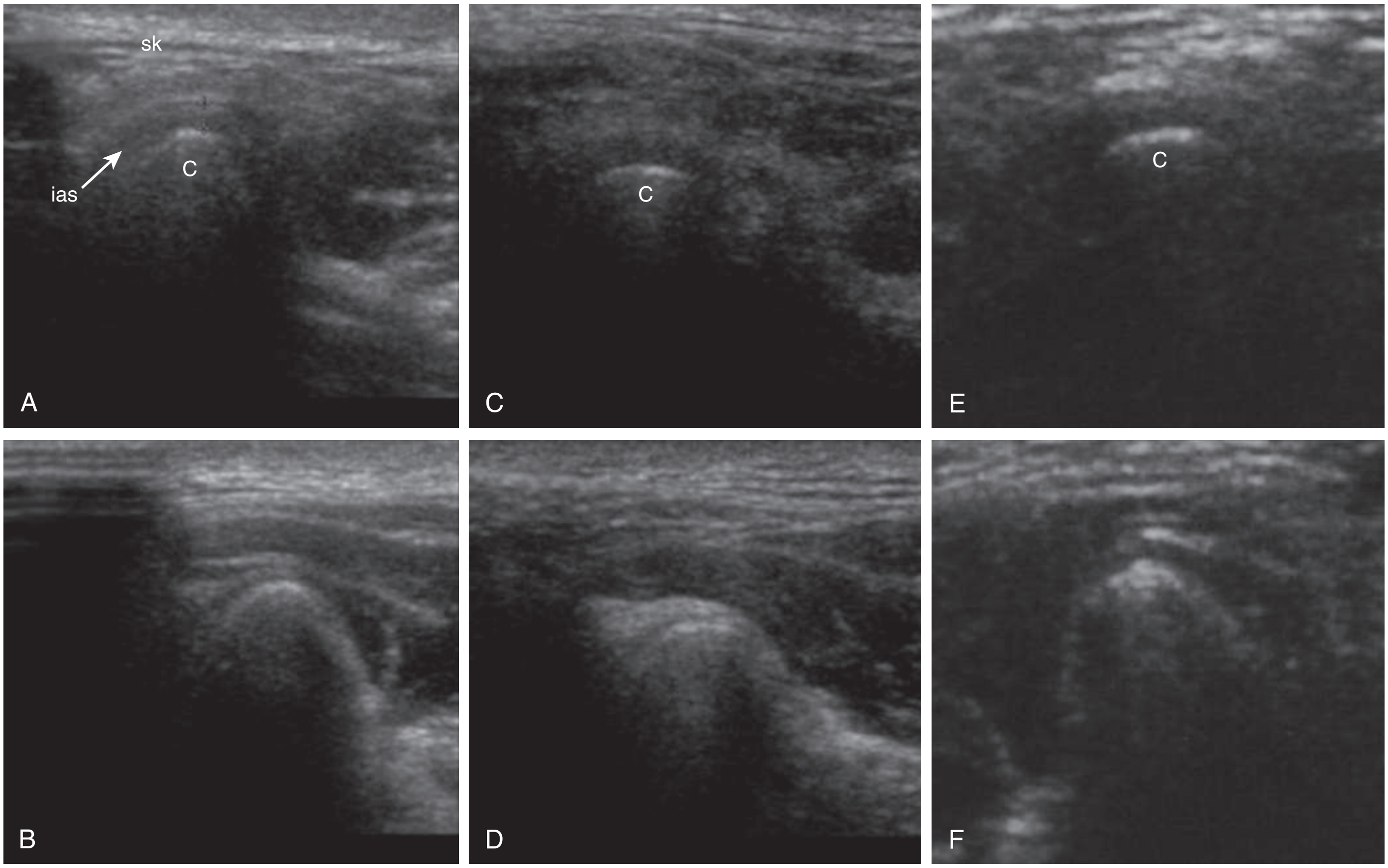

FIGURE 1-8 Examples of ultrasound examinations of individuals presenting with normal disk position **(A, B)**, reducible disk displacement **(C, D)**, and irreducible disk displacement **(E, F)**. **A, C,** and **E,** Closed-mouth. **B, D,** and **F,** Opened mouth. **A–D,** Right TMJ. **E** and **F,** Left TMJ. *C*, condyle; *ias*, intra-articular space; *sk*, skin. (From Dupuy-Bonafé I, Picot M-C, Maldonado IL, et al.: Internal derangement of the temporomandibular joint: is there still a place for ultrasound? *Oral Surg Oral Med Oral Pathol Oral Radiol* 113[6]:832–840, 2012; Fig. 2.)

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Supraorbital Nerve Block

INDICATIONS AND CLINICAL CONSIDERATIONS

Supraorbital nerve block is useful in the diagnosis and treatment of swimmer's headache and supraorbital neuralgia. Supraorbital neuralgia and swimmer's headache is often the result of compression of the supraorbital nerves by swimming goggles, welding masks, respirators, etc., that fit poorly or are worn too tightly, exerting pressure on the supraorbital nerves as they exit the supraorbital foramen (Figure 2-1). Repetitive microtrauma from wearing swim goggles may also cause these painful conditions. The pain of supraorbital neuralgia and swimmer's headache is characterized as persistent pain in the supraorbital region and forehead with occasional, sudden shocklike paresthesias in the distribution of the supraorbital nerves. Occasionally, a patient with supraorbital neuralgia or swimmer's headache will complain that the hair on the front of the head "hurts." Sinus headache involving the frontal sinuses, which is much more common than swimmer's headache, occasionally mimics the pain of swimmer's headache.

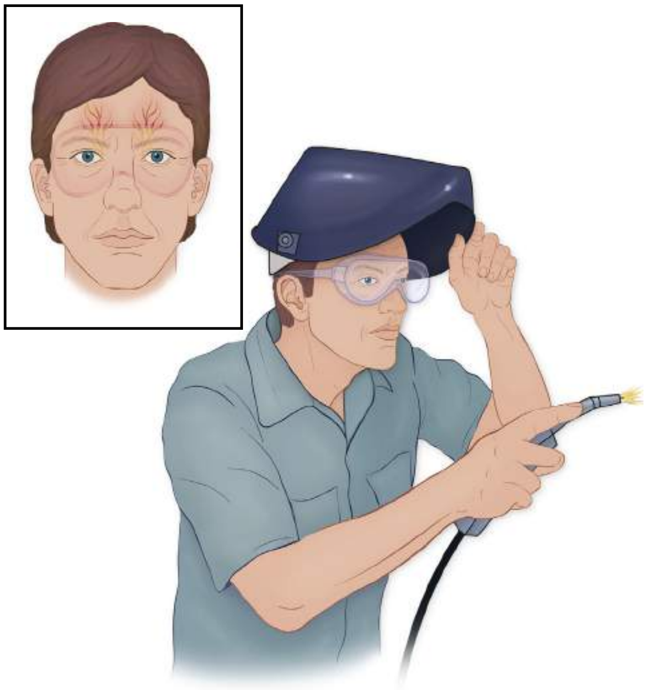

FIGURE 2-1 Occasionally, a patient with supraorbital neuralgia or swimmer's headache complains that the hair on the front of the head hurts. The supraorbital nerve sends fibers all the way to the vertex of the scalp and provides sensory innervation to the forehead, upper eyelid, and anterior scalp. (From Waldman SD, editor: Supraorbital neuralgia. *In Atlas of uncommon pain syndromes*, ed 3, Philadelphia, 2014, Saunders; Fig. 2-2.)

CLINICALLY RELEVANT ANATOMY

The supraorbital nerve arises from fibers of the frontal nerve, which is the largest branch of the ophthalmic nerve. The frontal nerve enters the orbit via the superior orbital fissure and passes anteriorly beneath the periosteum of the roof of the orbit. The frontal nerve gives off a larger lateral branch, the supraorbital nerve, and a smaller medial branch, the supratrochlear nerve, and both exit the orbit anteriorly. After exiting the supraorbital foramen, the supraorbital nerve sends fibers all the way to the vertex of the scalp and provides sensory innervation to the forehead, upper eyelid, and anterior scalp (Figure 2-2).

TECHNIQUE

Landmark Technique

The patient is placed in a supine position. A total of 3 mL of local anesthetic is drawn up in a 10-mL sterile syringe. When supraorbital neuralgia or swimmer's headache is treated with a supraorbital nerve block, a total of 80 mg of depot corticosteroid is added to the local anesthetic with the first block, and 40 mg of depot corticosteroid is added with subsequent blocks.

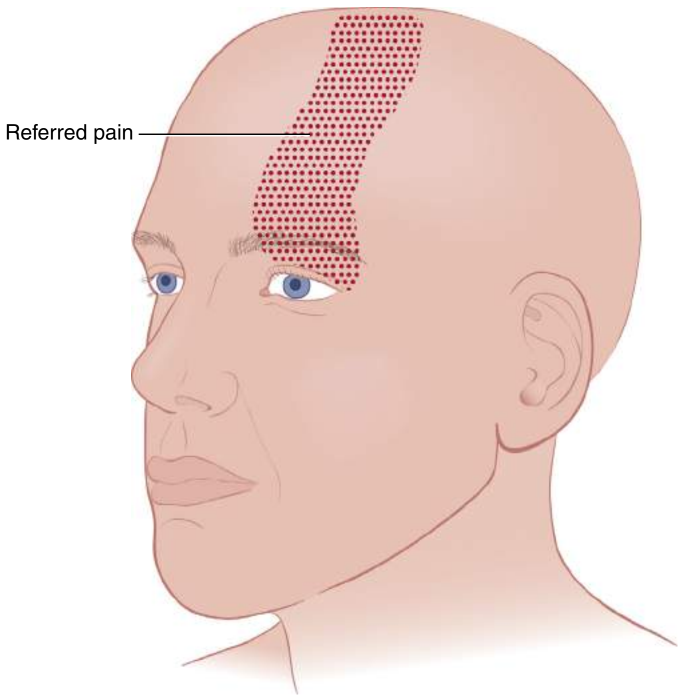

FIGURE 2-2 Supraorbital neuralgia and swimmer's headache is characterized by persistent pain in the supraorbital region with associated intermittent shocklike paresthesias.

The supraorbital notch on the affected side is then identified by palpation. The skin overlying the notch is prepared with antiseptic solution, with care taken to avoid spillage into the eye. A 25-gauge, 1½-inch needle is inserted at the level of the supraorbital notch and is advanced medially approximately 15 degrees off the perpendicular to avoid entering the foramen. The needle is advanced until it approaches the periosteum of the underlying bone (Figure 2-3). A paresthesia may be elicited, and the patient should be warned of such. The needle should not enter the supraorbital foramen; should this occur, the needle should be withdrawn and redirected slightly more medially.

Because of the loose alveolar tissue of the eyelid, a gauze sponge should be used to apply gentle pressure on the upper eyelid and supraorbital tissues before injection of solution to prevent the injectate from dissecting inferiorly into these tissues. This pressure should be maintained after the procedure to avoid periorbital hematoma and ecchymosis.

After gentle aspiration, 3 mL of solution is injected in a fanlike distribution. If blockade of the supratrochlear nerve is also desired, the needle is then redirected medially; after careful aspiration, an additional 3 mL of solution is injected in a fanlike manner.

Ultrasound-Guided Technique

To perform an ultrasound-guided supraorbital nerve block, the patient is placed in a supine position and the supraorbital foramen is then identified by palpation. A high-frequency linear transducer or transducer is then placed in a transverse plane over the supraorbital notch and slowly moved from a cephalad to caudad direction until the discontinuity of the supraorbital ridge indicating the supraorbital foramen is identified (Figure 2-4). If there is difficulty in identifying the supraorbital foramen, color Doppler can be used to identify the supraorbital artery, which exits the supraorbital foramen in proximity to the supraorbital nerve (Figure 2-5). A 25-gauge, 1½-inch needle is inserted at the level of the supraorbital notch and is advanced medially approximately 15 degrees off the perpendicular to avoid entering the foramen. Because of the loose alveolar tissue of the eyelid, a gauze sponge should be used to apply gentle pressure on the upper eyelid and supraorbital tissues before injection of solution to prevent the injectate from dissecting inferiorly into these tissues. This pressure should be maintained after the procedure to avoid periorbital hematoma and ecchymosis.

After the needle is in proximity to the supraorbital nerve, gentle aspiration is performed and 3 mL of solution is injected in a fanlike distribution. If blockade of the supratrochlear nerve is also desired, the needle is then redirected medially; after careful aspiration, an additional 3 mL of solution is injected in a fanlike manner (see Chapter 3).

SIDE EFFECTS AND COMPLICATIONS

The forehead and scalp are highly vascular, and the pain specialist should carefully calculate the total milligram dose of local anesthetic that may be safely given, especially if bilateral nerve blocks are being performed. This vascularity gives rise to an increased incidence of postblock ecchymosis and hematoma formation. In spite of the vascularity of this anatomic region,

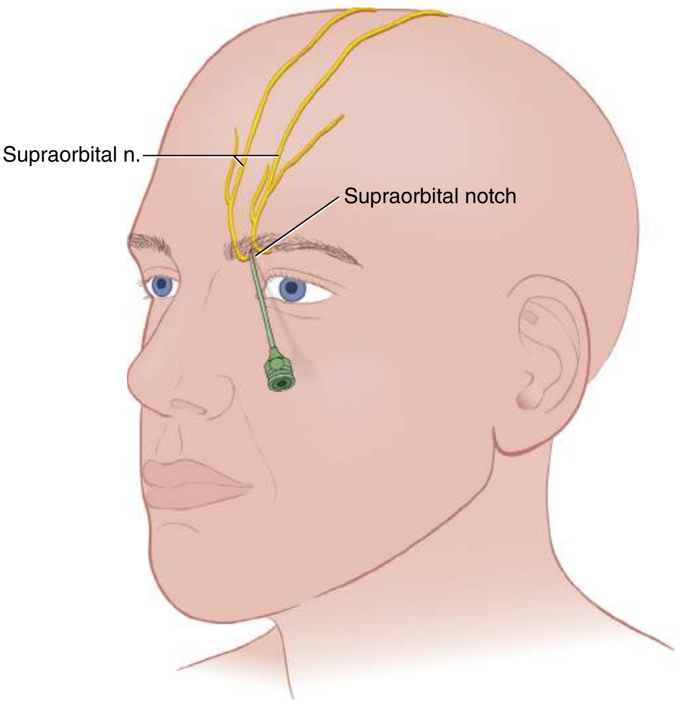

FIGURE 2-3 When the needle is placed for supraorbital nerve block, care should be taken to avoid advancing the needle into the supraorbital foramen.

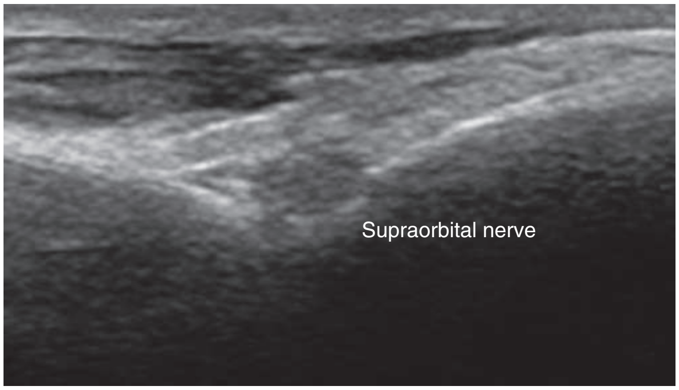

FIGURE 2-4 Transverse ultrasound image of the supraorbital nerve as it exits the supraorbital foramen.

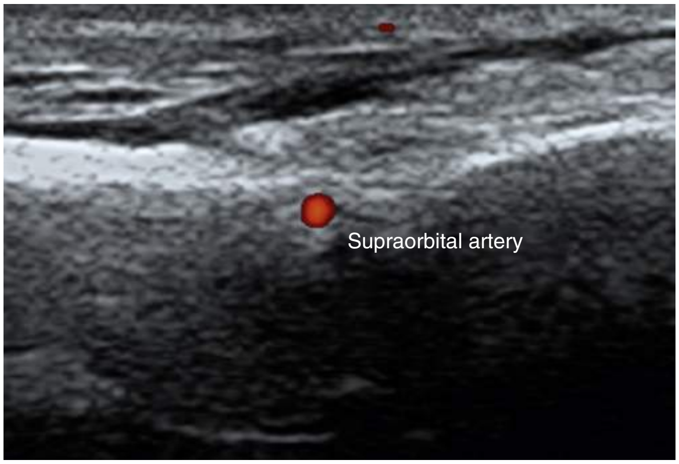

FIGURE 2-5 Color Doppler imaging may assist in identifying the supraorbital artery, which lies in proximity to the supraorbital nerve as they exit the supraorbital foramen.

this technique can be safely performed in the presence of anticoagulation by using a 25- or 27-gauge needle, albeit at increased risk of hematoma, if the clinical situation dictates a favorable risk-to-benefit ratio. These complications can be decreased if manual pressure is applied to the area of the block immediately after injection. Application of cold packs for 20-minute periods after the block also decreases the amount of postprocedure pain and bleeding the patient may experience.

CLINICAL PEARLS

Supraorbital nerve block is especially useful in the diagnosis and palliation of pain secondary to swimmer's headache and supraorbital neuralgia. The first step in the management of this unusual type of headache is the correct fitting of swim goggles, welding masks, respirators, etc., so they do no compress the supraorbital nerves. Coexistent frontal sinusitis should be ruled out in patients who do not respond rapidly to a change in swim goggles and a series of the previously described nerve blocks.

Any patient with headaches severe enough to require neural blockade as part of the treatment plan should undergo computed tomography or magnetic resonance imaging of the head to rule out unsuspected intracranial pathology.

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3

Trochlear Nerve Block

INDICATIONS AND CLINICAL CONSIDERATIONS

Trochlear injection is useful in the diagnosis and treatment of primary trochlear headache. As with most headache syndromes, the exact cause of the pain of primary trochlear headache is unknown, and whether the trochlear nerve plays a role in pathogenesis of this uncommon source of head and face pain is the subject of ongoing debate.

In patients with primary trochlear headache the presenting symptom is unilateral periorbital pain radiating from the trochlear area with associated headache. The pain of primary trochlear headache is exacerbated by supraduction of the affected eye, although no limitation of range of motion of the superior oblique should be noted. The pain of this uncommon headache syndrome is often worse at night; initially the pain is characterized by remissions and exacerbations, but without treatment it can become chronic. As the name implies, primary trochlear headache is a diagnosis of exclusion, because it occurs in the absence of primary orbital, retro-orbital, or ocular pathology.

Often confused with acute ocular diseases such as glaucoma or herpes zoster of the first division of the trigeminal nerve or Charlin syndrome, pathology of the orbit and the retro-orbital region must be ruled out before the diagnosis of primary trochlear headache can be made. Inflammatory and autoimmune conditions involving the trochlear nerve anywhere along its path, such as multiple sclerosis, cranial neuritis, and Tolosa-Hunt syndrome, as well as compromise of the trochlear nerve by tumor, abscess, or vascular abnormality must be carefully ruled out before the diagnosis of primary trochlear headache can be considered (Figures 3-1 and 3-2). The diagnosis of primary trochlear headache is then confirmed by injection of the trochlear region with local anesthetic and antiinflammatory steroid. Primary trochlear headache will uniformly respond to this injection.

CLINICALLY RELEVANT ANATOMY

The trochlear nerve (cranial nerve IV) is composed of somatic general efferent motor fibers. It innervates the superior oblique extraocular muscle of the contralateral orbit (Figure 3-3). Contraction of the superior oblique extraocular muscle intorts (rotates inward), depresses, and abducts the globe. The superior oblique extraocular muscle works in concert with the five other extraocular muscles to allow the eye to perform its essential functions of tracking and fixation on objects.

The fibers of the trochlear nerve originate from the trochlear nucleus, which is just ventral to the cerebral aqueduct in the tegmentum of the midbrain at the level of the inferior colliculus. As the trochlear nerve leaves the trochlear nucleus, it travels dorsally, wrapping itself around the cerebral aqueduct to then decussate in the superior medullary velum. The decussated fibers of the trochlear nerve then exit the dorsal surface of the brainstem just below the contralateral inferior colliculus, on which they then curve around the brainstem, leaving the subarachnoid space along with the oculomotor nerve (cranial nerve III) between the superior cerebellar and posterior cerebral arteries. The trochlear nerve then enters the cavernous sinus and runs anteriorly along the lateral wall of the sinus with the oculomotor nerve (cranial nerve III), trigeminal nerve (cranial nerve V), and abducens nerve (cranial nerve VI).

After exiting the cavernous sinus, the trochlear nerve enters the orbit via the superior orbital fissure. Unlike the oculomotor nerve, the trochlear nerve does not pass through the tendinous ring of the extraocular muscles but passes just above the ring. The trochlear nerve then crosses medially along the roof of the

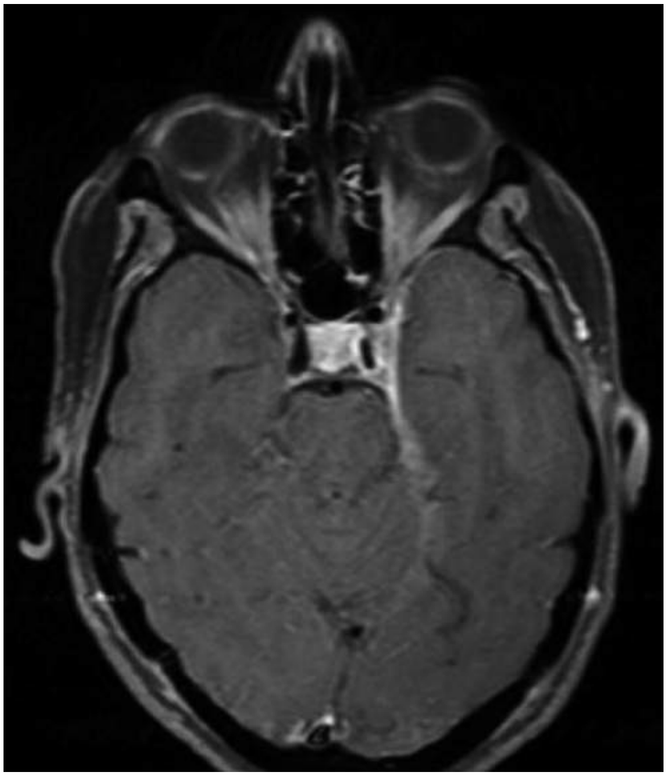

FIGURE 3-1 Axial, T1-weighted, contrast-enhanced image demonstrates soft tissue in the left cavernous sinus, which has enhanced markedly. The enhancement extends along the free edge of the tentorium cerebelli. Imaging is nonspecific, but after exclusion of other conditions this patient was diagnosed with Tolosa-Hunt syndrome. (From Tang Y, Booth T, Steward M, et al: The imaging of conditions affecting the cavernous sinus. *Clin Radiol* 65:937–945, 2010.)

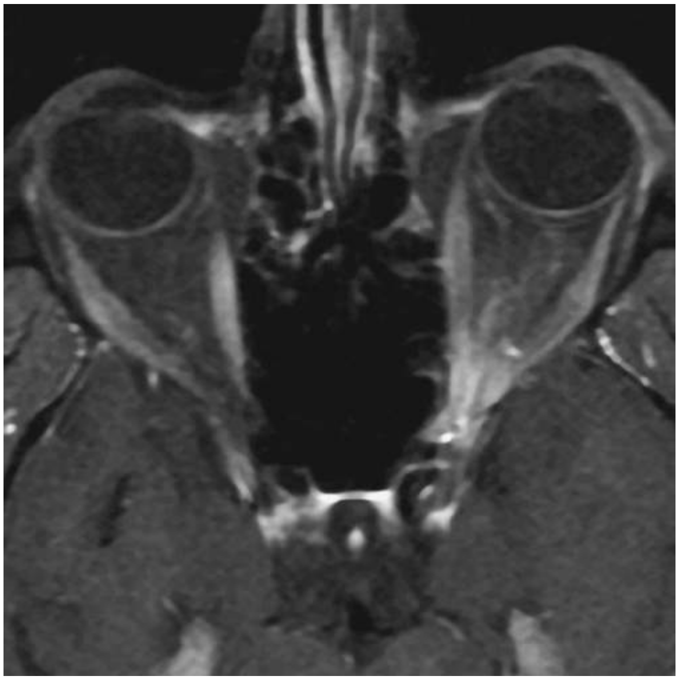

FIGURE 3-2 Axial T1-weighted image with gadolinium and fat saturation. This 55-year-old man had painful ophthalmoplegia (cranial nerves III, IV, V, and VI) and slight proptosis of the right eye. Magnetic resonance imaging shows an enhancing ill-defined process in the right orbital apex. Cavernous sinus not involved (Tolosa-Hunt syndrome). (From Ferreira T, Verbist B, van Buchem M, et al: Imaging the ocular motor nerves. *Eur J Radiol* 74:314–322, 2010.)

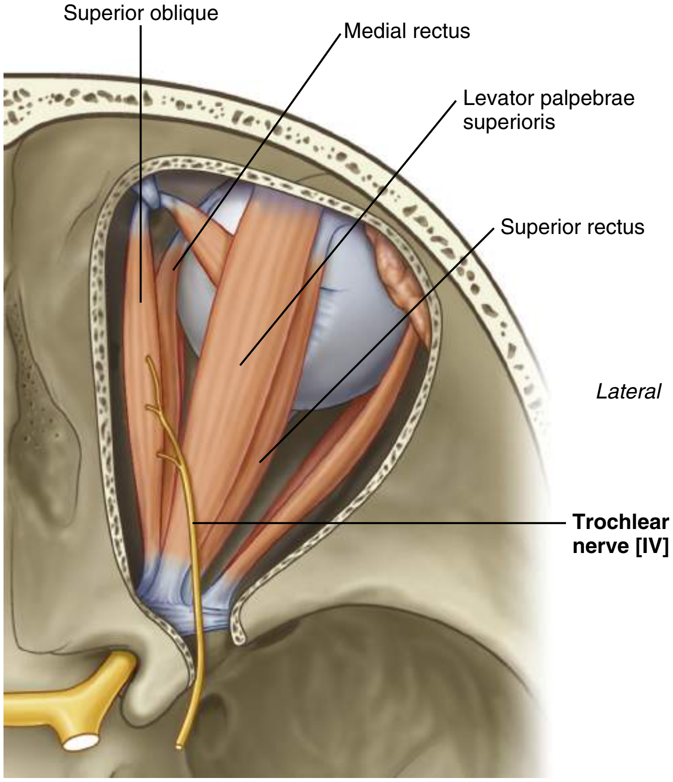

FIGURE 3-3 Trochlear nerve (IV) in the orbit. (From Drake RL, Vogl W, Mitchell AWM: *Gray's anatomy for students*, ed 2, Philadelphia, 2010, Churchill Livingstone.)

orbit above the levator palpebrae and superior rectus muscles to innervate the superior oblique muscle (see Figure 3-3).

Disorders of the trochlear nerve can be caused by central lesions that affect the trochlear nucleus, such as stroke or space-occupying lesions such as tumor, abscess, or aneurysm. Increased intracranial pressure from subdural hematoma, sagittal sinus thrombosis, or abscess can compromise the nucleus and/or the efferent fibers of the trochlear nerve as they exit the brainstem and travel toward the orbit, with resultant abnormal nerve function. Traction on the trochlear nerve from loss of cerebrospinal fluid has also been implicated in cranial nerve IV palsy. Small vessel disease from diabetes or vasculitis associated with temporal arteritis may cause ischemia and even infarction of the trochlear nerve with resultant pathologic symptoms.

In almost all disorders of the trochlear nerve, symptoms will take the form of a palsy of the superior oblique muscle, most commonly manifesting as an inability to look inward and downward. Often the patient will report difficulty in walking down stairs because of the inability to depress the affected eye or eyes. On physical examination the clinician may note extorsion (outward rotation) of the affected eye as a result of the unopposed action of the inferior oblique muscle (Figure 3-4, *A*). In an effort to compensate, the patient may deviate his or her face forward and downward with the chin rotated toward the affected side to look downward (Figure 3-4, *B*). However, it should be remembered that isolated trochlear nerve palsy is the least common of the ocular motor palsies, and its presence should be viewed as an ominous warning sign.

TECHNIQUE

The patient is placed in a supine position. A total of 2 mL of local anesthetic is drawn up in a 10-mL sterile syringe. When primary trochlear headache is treated with trochlear region block, a total of 80 mg of depot corticosteroid is added to the local anesthetic with the first block, and 40 mg of depot corticosteroid is added with subsequent blocks.

For a trochlear region block to be performed, the medial canthus is identified and a line is drawn superiorly to a point just below the eyebrow. The skin at the midpoint of this line is prepared with antiseptic solution, with care taken to avoid spillage into the eye. A 25-gauge, 1½-inch needle is inserted at this point and advanced until the tip of the needle makes contact with the bony surface of the orbit. After careful gentle aspiration, the contents of the syringe are slowly injected as the needle is slowly directed superiorly and inferiorly (Figure 3-5). Because of the loose alveolar tissue of the eyelid, a gauze sponge should be used to apply gentle pressure on the upper eyelid and supraorbital tissues before injection of solution to prevent the injectate from dissecting inferiorly into these tissues. This pressure should be maintained after the procedure to avoid periorbital hematoma and ecchymosis. When trochlear region block is used to treat the pain and symptoms associated with primary trochlear headache, 8 to 10 daily nerve blocks with local anesthetic may be required. If daily blocks are performed, as a general rule the total dose of depot corticosteroids included in these blocks should not exceed 360 to 400 mg.

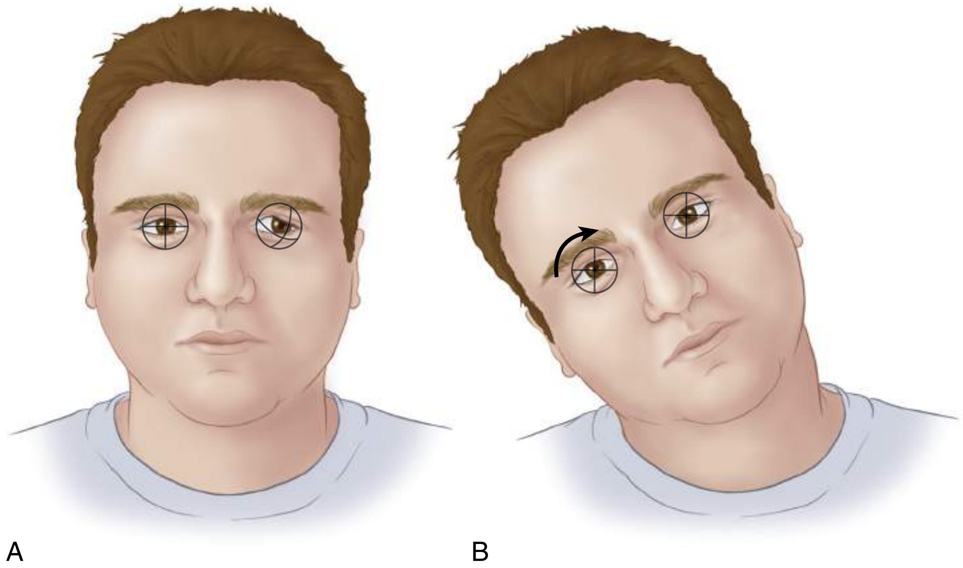

FIGURE 3-4 **A,** The unopposed action of the inferior oblique muscle in the presence of trochlear nerve palsy results in extorsion of the globe and associated weak downward gaze. **B,** To compensate for the unopposed action of the inferior oblique muscle in the presence of trochlear palsy, the patient deviates his face forward and downward with the chin rotated toward the affected side.

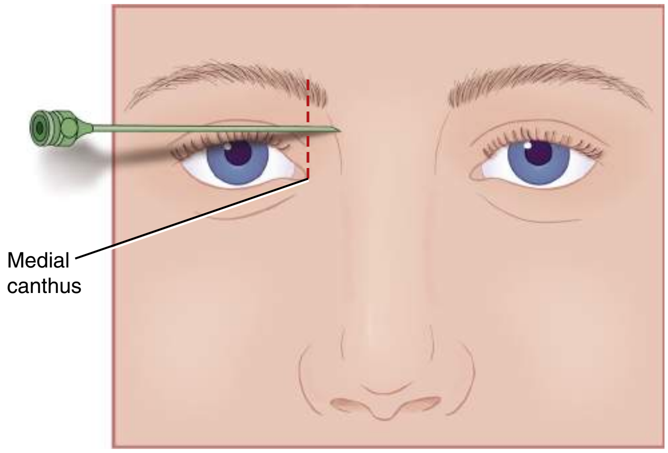

FIGURE 3-5 For trochlear block to be performed, the medial canthus is identified, and a line is drawn superiorly to a point just below the eyebrow.

SIDE EFFECTS AND COMPLICATIONS

The major complication of this procedure is inadvertent injury to the eye. Failure to maintain bony contact while advancing the needle will greatly increase the risk of the devastating complication. The practitioner should also remember that this area is highly vascular and the potential for intravascular injection of local anesthetic with its attendant risks remains an ever-present possibility. This vascularity gives rise to an increased incidence of postblock ecchymosis and hematoma formation. These complications can be decreased if manual pressure is applied to the area of the block immediately after injection. Application of cold packs for 20-minute periods after the block also decreases the amount of postprocedure pain and bleeding.

CLINICAL PEARLS

Trochlear nerve block is especially useful in the diagnosis and palliation of pain secondary to primary trochlear headache. The first step in the care of patients thought to have this unusual type of headache is ruling out more common types of headache that may mimic primary trochlear headache. Any patient with headaches bad enough to require neural blockade as part of the treatment plan should undergo computed tomography or magnetic resonance imaging of the head to rule out unsuspected intracranial pathologic conditions.

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4

Buccal Fold Injection Technique for Incisors and Canine Teeth

INDICATIONS AND CLINICAL CONSIDERATIONS

The buccal fold injection technique is useful in the diagnosis and treatment of pain involving the incisors or canine teeth of the upper jaw. This technique can provide much-needed emergency relief of dental pain while the patient is waiting for definitive dental treatment. It can also serve as a useful diagnostic maneuver when the clinician is trying to localize the nidus of pain that the patient perceives as dental in origin.

Dental pain is the result of irritation or inflammation of the nerves of the pulp and/or root of the tooth. Common causes of irritation or inflammation responsible for dental pain include infection, decay with resultant nerve exposure, gingival disease, plaque at or below the gum line, bruxism, injury, tumor, and tooth extractions (Figure 4-1). Less common causes include chemotherapy-induced odontalgia and barodontalgia. Pain involving the incisors or canine teeth may also be referred from other anatomic areas. Such referred pain may be indicative of temporomandibular joint dysfunction, sinus disease, abnormalities of the trigeminal nerve and its branches, and coronary artery stenosis.

Dental pain may range from a dull ache to severe unremitting pain. Its onset may be insidious or acute. Dental pain is often worse when the affected tooth or teeth are exposed to hot or cold temperatures and when direct pressure is applied to the tooth or teeth when chewing. Tapping on the affected tooth or teeth may elicit an acute exacerbation of the pain. If significant inflammation or infection is present, rubor and color may be present as well as swelling. Gingival bleeding or purulent drainage may also be present. It should be remembered that on occasion a severely compromised tooth that is causing a patient significant pain may appear completely normal.

CLINICALLY RELEVANT ANATOMY

The incisor and canine and surrounding periosteum and buccal and gingival tissue are innervated by the superior alveolar nerve, which is a branch of the inferior alveolar nerve just before it exits from the infraorbital canal below the orbit (Figure 4-2). Fibers of the ipsilateral superior alveolar nerve may cross the midline and may anastomose with fibers of the

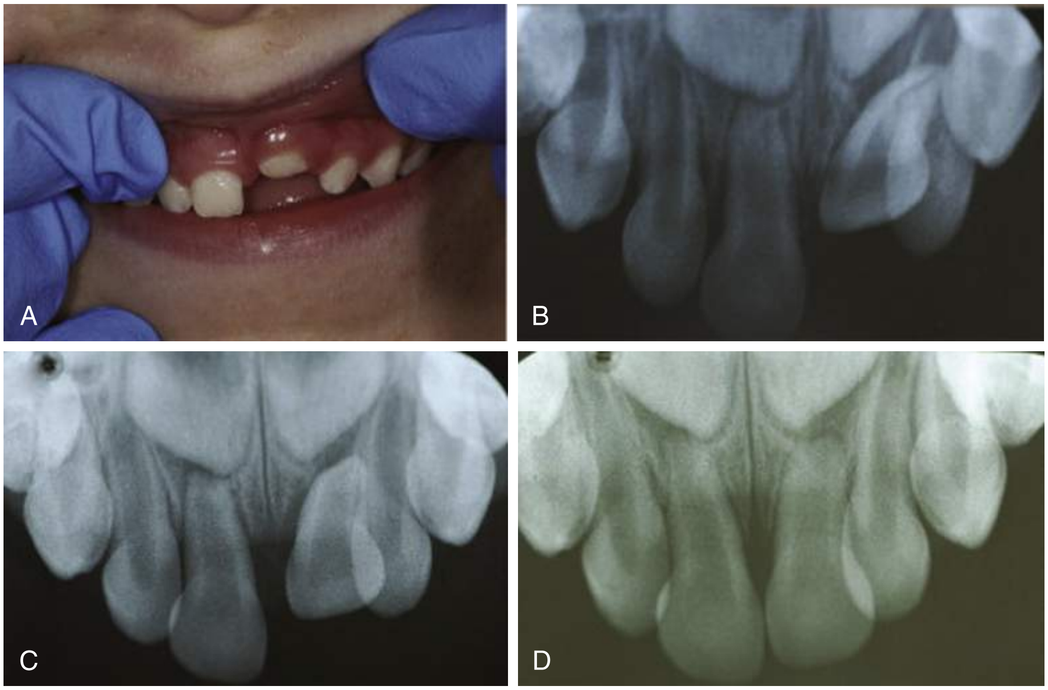

FIGURE 4-1 Intruded primary incisor. **A,** Day of injury. **B,** Radiograph on day of injury. **C,** Three weeks postinjury. **D,** Five months postinjury. (From McTigue DJ: Managing injuries to the primary dentition. *Dent Clin North Am* 53:627–638, 2009.)

contralateral nerve, although medial spread of injected local anesthetics may be limited by the attachments of the labial frenulum at the midline. The periosteum and bone that surround and support the root of the tooth are relatively thin and readily allow diffusion of local anesthetics injected in this region (Figure 4-3).

TECHNIQUE

The patient is placed in a supine position. A total of 1 to 2 mL of local anesthetic is drawn up in a 3-mL sterile syringe. The lip overlying the affected tooth is retracted, and a small amount of topical anesthetic, such as viscous lidocaine or EMLA cream, is applied to the alveolar sulcus with a cotton-tipped applicator. After topical anesthesia has been achieved, a 25-gauge, 1½-inch needle is inserted through the previously anesthetized area and advanced axially and slightly medially toward the apex of the affected tooth. When the needle tip impinges on bone, it is withdrawn slightly out of the periosteum, and after gentle aspiration the local anesthetic is slowly injected around the apex target area. The anesthetic will rapidly diffuse and anesthetize the pulp of the affected tooth (Figure 4-4). It should be remembered that the root of the canine tooth is longer than the root of the incisor, and the apical portion of the root is often slightly more distally oriented.

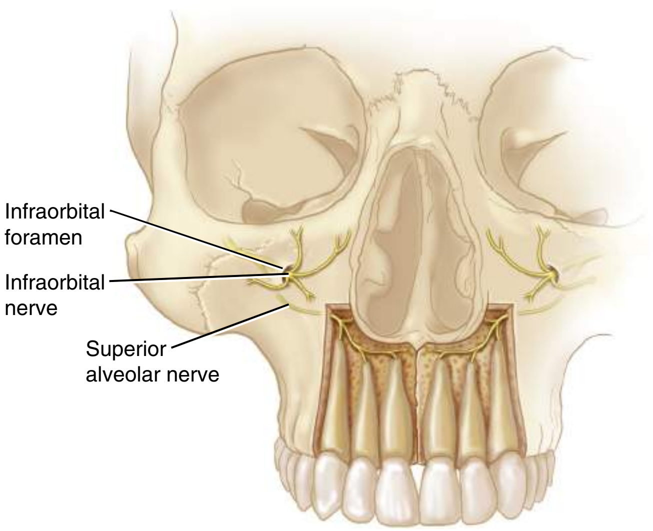

FIGURE 4-2 The relationship of the superior alveolar nerve to the incisor and canine.

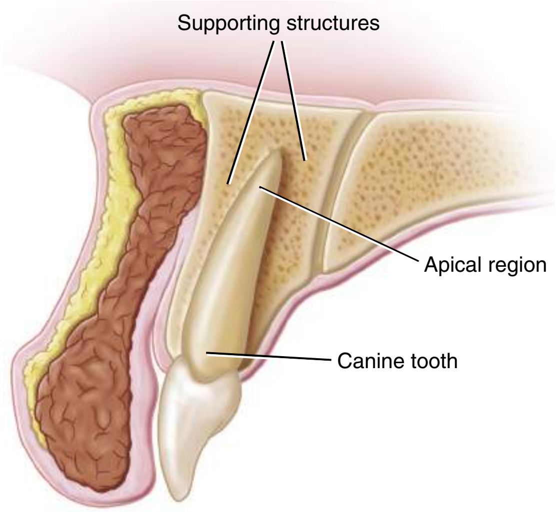

FIGURE 4-3 Lateral view of the canine demonstrating the relationship of the supporting structures and apical region.

SIDE EFFECTS AND COMPLICATIONS

Generally, the use of nerve blocks in dentistry has enjoyed an amazing track record of utility and safety. Most side effects and complications are related to inadvertent intravascular injection, the use of local anesthetics containing epinephrine, and vasovagal syncope. Occasional hematoma or ecchymosis formation is encountered after dental nerve block, especially in patients taking anticoagulants or antiplatelet drugs. Pitfalls in needle placement include decreased diffusion of local anesthetic resulting from placement that is too superficial or positioning of the needle tip between the relatively impermeable fascia and labial muscle. Excessive pain from overly rapid injection of local anesthetic should also be avoided. The clinician is reminded that severe dental abscess can be life threatening, and emergency incision and drainage combined with aggressive antibiotic therapy may be required to avoid disaster. Referred pain as well as pain from tumor should always be considered when evaluating a patient with dental pain.

CLINICAL PEARLS

Traumatic or nontraumatic dental pain is an increasingly frequent reason for adult and pediatric patients to visit urgent care and emergency room facilities. Often, urgent care or emergency room physicians have little or no training in the treatment of painful dental conditions. The use of dental nerve blocks with long-acting local anesthetics can provide excellent palliation of pain while the patient waits to obtain emergent dental care.

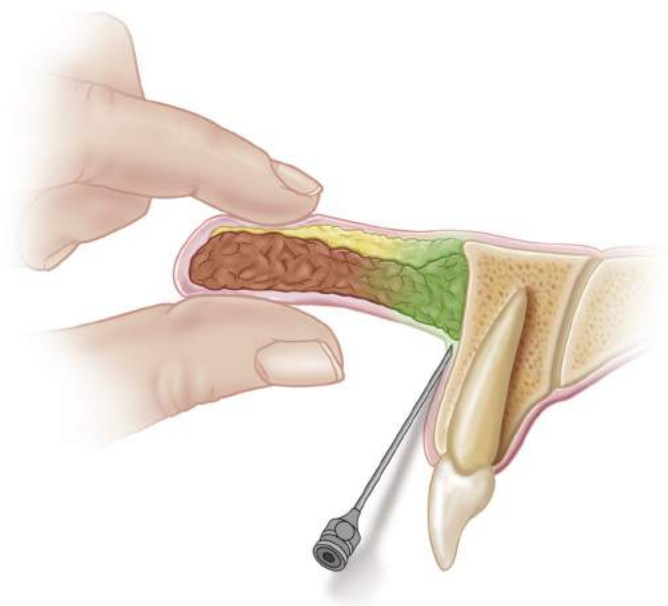

FIGURE 4-4 Proper needle placement with needle tip in good position. Green area indicates the initial flow of local anesthetic.

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Buccal Fold Injection Technique for Upper Premolar Teeth

INDICATIONS AND CLINICAL CONSIDERATIONS

The buccal fold injection technique is useful in the diagnosis and treatment of pain involving the premolar teeth of the upper jaw. This technique can provide much-needed emergency relief of dental pain while the patient is waiting for definitive dental treatment. It can also serve as a useful diagnostic maneuver when the clinician is trying to localize the nidus of pain that the patient perceives as dental in origin.

Dental pain is the result of irritation or inflammation of the nerves of the pulp and/or root of the tooth. Common causes of irritation or inflammation responsible for dental pain include infection, decay with resultant nerve exposure, gingival disease, plaque at or below the gum line, bruxism, injury, tumor, and tooth extraction. Less common causes include chemotherapy-induced odontalgia and barodontalgia. Pain involving the incisors or canine teeth may also be referred from other anatomic areas. Such referred pain may be indicative of temporomandibular joint dysfunction, sinus disease, abnormalities of the trigeminal nerve and its branches, and coronary artery stenosis.

Dental pain may range from a dull ache to severe, unremitting pain. Its onset may be insidious or acute. Dental pain is often worse when the affected tooth or teeth are exposed to hot or cold temperatures and when direct pressure is applied to the tooth or teeth when chewing. Tapping on the affected tooth or teeth may elicit an acute exacerbation of the pain. If significant inflammation or infection is present, rubor and color as well as swelling may be seen. Gingival bleeding or purulent drainage may also be present. It should be remembered that on occasion a severely compromised tooth causing a patient significant pain may appear completely normal.

CLINICALLY RELEVANT ANATOMY

The upper premolar is innervated by the superior dental plexus, which is composed of convergent fibers from the superior, posterior, and anterior alveolar nerves (Figure 5-1). In some patients the premolars are innervated from fibers from the middle superior alveolar nerve. The periosteum and bone that surround and support the root of the tooth are relatively thin and readily allow diffusion of local anesthetics injected in this region (Figure 5-2). The adjacent palate is innervated by the greater palatine nerve and occasionally fibers of the nasopalatine nerve (Figure 5-3). Supplemental blockade of these nerves will often be required to completely relieve upper premolar pain.

TECHNIQUE

The patient is placed in a supine position. A total of 1 to 2 mL of local anesthetic is drawn up in a 3-mL sterile syringe. The lip overlying the affected tooth is retracted, and a small amount of topical anesthetic such as viscous lidocaine or EMLA cream is applied to the alveolar sulcus with a cotton-tipped applicator. After topical anesthesia has been achieved, a 25-gauge, 1½-inch needle is inserted through the previously anesthetized area and advanced axially toward the apex of the affected tooth (Figure 5-4). If the needle tip impinges on bone, it is withdrawn slightly out of the periosteum, and after gentle aspiration the local anesthetic is slowly injected around the apex target area. The anesthetic will rapidly diffuse and anesthetize the pulp of the affected tooth. Supplemental block of the greater palatine nerve is performed by placing the needle at right angles to the mucosa at a point approximately half the height of the affected premolar (Figure 5-5).

SIDE EFFECTS AND COMPLICATIONS

Generally, the use of nerve blocks in dentistry has enjoyed an amazing track record of utility and safety. Most side effects and complications are related to inadvertent intravascular injection, the use of local anesthetics containing epinephrine, and vasovagal syncope. Occasional hematoma and ecchymosis formation after dental nerve block is encountered, especially in patients taking anticoagulants or antiplatelet drugs. Pitfalls in needle placement include decreased diffusion of local anesthetic as a result of placement that is too superficial or positioning of the needle tip between the relatively

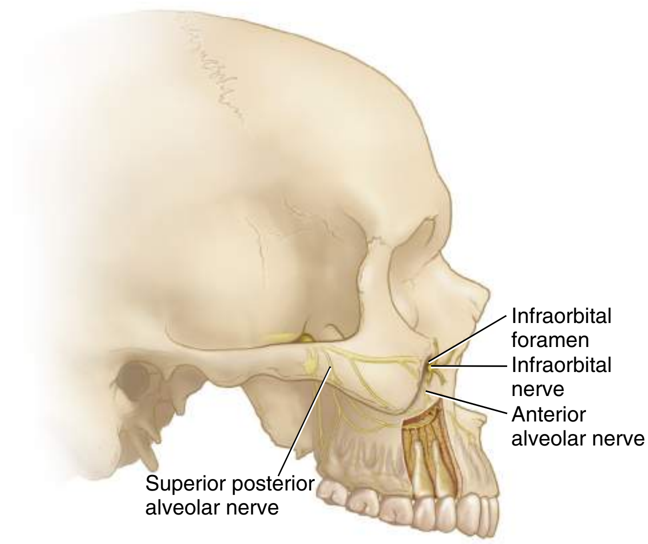

FIGURE 5-1 Innervation of the upper premolars.

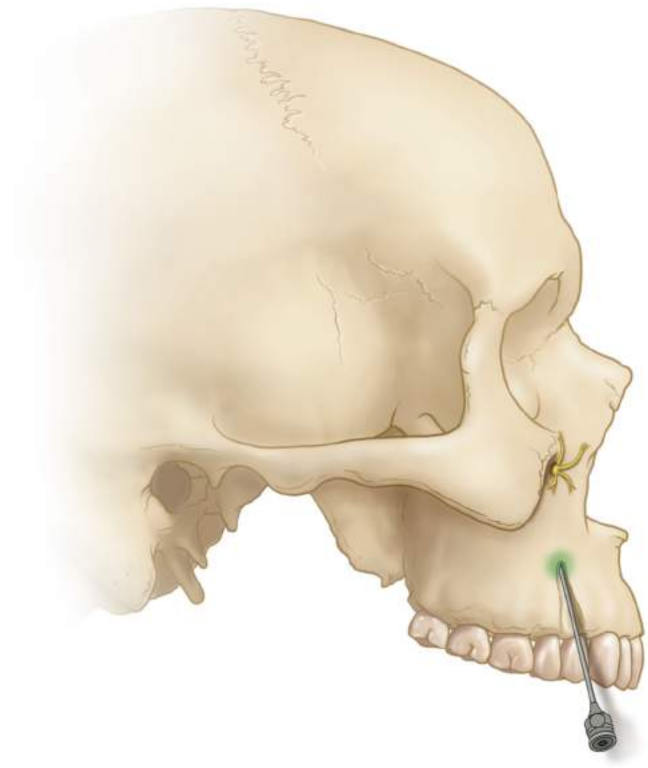

FIGURE 5-2 Spread of local anesthetic with blockade of upper premolar. Green indicates spread of local anesthetic.

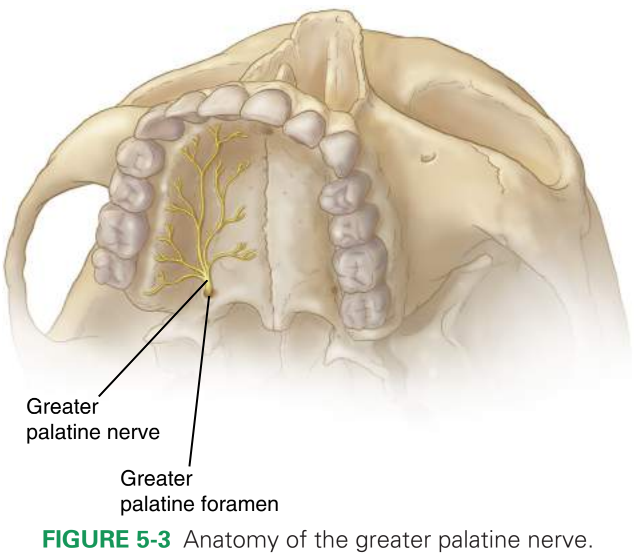

FIGURE 5-3 Anatomy of the greater palatine nerve.

impermeable fascia and labial muscle. Excessive pain caused by overly rapid injection of local anesthetic should also be avoided. The clinician is reminded that severe dental abscess can be life threatening, and emergency incision and drainage combined with aggressive antibiotic therapy may be required to avoid disaster. Referred pain as well as pain from tumor should always be considered when evaluating a patient with dental pain.

FIGURE 5-4 Needle placement for block of the upper premolars.

FIGURE 5-5 Needle placement for greater palatine nerve block.

CLINICAL PEARLS

Traumatic or nontraumatic dental pain is an increasingly frequent reason for adult and pediatric patients to visit urgent care and emergency room facilities. Often, urgent care or emergency room physicians have little or no training in the treatment of painful dental conditions. The use of dental nerve blocks with long-acting local anesthetics can provide excellent palliation of pain while the patient waits to obtain emergent dental care.

SUGGESTED READINGS

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6

Buccal Fold Injection for Upper Molar Teeth

INDICATIONS AND CLINICAL CONSIDERATIONS

The buccal fold injection technique is useful in the diagnosis and treatment of pain involving the molars of the upper jaw. This technique can provide much-needed emergency relief of dental pain while the patient is waiting for definitive dental treatment. It can also serve as a useful diagnostic maneuver when the clinician is trying to localize the nidus of pain that the patient perceives as dental in origin.

Dental pain is the result of irritation or inflammation of the nerves of the pulp and/or root of the tooth. Common causes of irritation or inflammation responsible for dental pain include infection, decay with resultant nerve exposure, gingival disease, plaque at or below the gum line, bruxism, injury, tumor, and tooth extraction. Less common causes include chemotherapy-induced odontalgia and barodontalgia. Pain involving the incisors or canine teeth may also be referred from other anatomic areas. Such referred pain may be indicative of temporomandibular joint dysfunction, sinus disease, abnormalities of the trigeminal nerve and its branches, and coronary artery stenosis.

Dental pain may range from a dull ache to severe, unremitting pain. Its onset may be insidious or acute. Dental pain is often worse when the affected tooth or teeth are exposed to hot or cold temperatures and when direct pressure is applied to the tooth or teeth when chewing. Tapping on the affected tooth or teeth may elicit an acute exacerbation of the pain. If significant inflammation or infection is present, rubor and color as well as swelling may be seen. Gingival bleeding or purulent drainage may also be present. It should be remembered that on occasion a severely compromised tooth causing a patient significant pain may appear completely normal.

CLINICALLY RELEVANT ANATOMY

The upper molars and surrounding periosteum and buccal and gingival tissue are innervated by the superior alveolar nerve, which branches from the infraorbital nerve before it enters the orbital cavity. These branches travel downward along the maxillary tuberosity to provide innervation for the upper molars and the buccal gingiva and associated periosteum. The gingiva, mucosa, and periosteum of the adjacent palate are innervated by the greater palatine nerve (Figure 6-1). The greater palatine nerve passes from the pterygopalatine fossa via the pterygopalatine canal through the pterygopalatine foramen (see Figure 5-3). In some patients an anatomic variation occurs, and the upper molars are innervated primarily by the middle alveolar nerves. This variation has little clinical import as far as the success of this block is concerned. The palate adjacent to the molars is innervated by the greater palatine nerve and in some patients by small anastomosing branches of the nasopalatine nerve (see Figure 5-3). The periosteum and bone that surround and support the roots of the molars are relatively thin and readily allow diffusion of local anesthetics injected in this region. For satisfactory anesthesia to be provided to the upper molars, three separate injections are usually required: (1) the buccal fold injection, (2) the tuberosity injection, and (3) the supplemental greater palatine nerve injection. Each injection is described in the following sections.

TECHNIQUE

Buccal Fold Injection

The patient is placed in a supine position. If the more distal molars are to be blocked, it is important not to have the patient open the mouth too widely or the coronoid process of the mandible will move ventrally and block the injection site. A total of 4 mL of local anesthetic is drawn up in a 5-mL sterile syringe. The lip overlying the affected tooth is retracted, and a small amount of topical anesthetic such as viscous lidocaine or EMLA cream is applied to the alveolar sulcus with a cotton-tipped applicator. After topical anesthesia has been achieved, a 25-gauge, 1½-inch needle is inserted through the previously anesthetized area and advanced axially and slightly posteriorly toward the apex of the affected tooth. When the needle tip impinges on bone, it is withdrawn slightly out of the periosteum, and after gentle aspiration 1 to 2 mL of

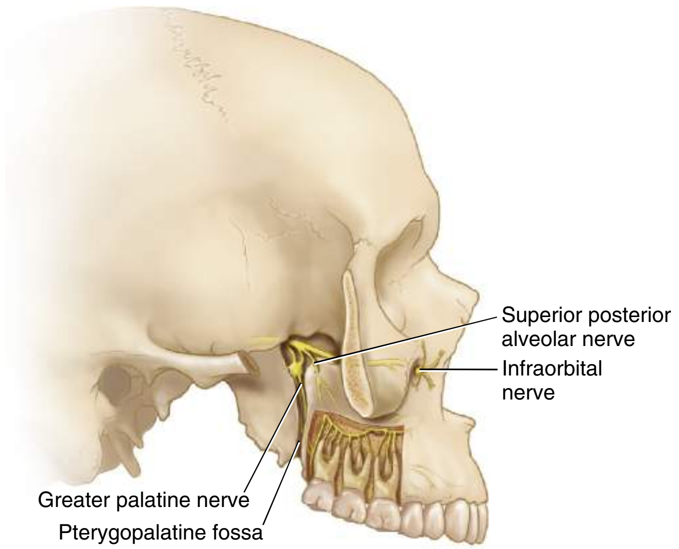

FIGURE 6-1 Innervation of the upper molars.

local anesthetic is slowly injected around the apex target area; the anesthetic will rapidly diffuse and anesthetize the pulp of the affected tooth (Figures 6-2 and 6-3). It should be noted that unlike the previously described buccal fold injection techniques for the incisors, canines, and premolars, this technique yields relatively little anesthesia of the adjacent lip (Figure 6-4).

Tuberosity Injection

The lip overlying the affected tooth is retracted, and the infrazygomatic crest is palpated with the gloved index finger, which also gently retracts the corner of the mouth posteriorly. A 25-gauge, 1½-inch needle is inserted slightly distal to the second molar. While it is kept close to the maxillary tuberosity, it is advanced in a superior and posterior trajectory (Figure 6-5). After gentle aspiration, an additional 2 mL of local anesthetic is slowly injected around the apex target area. The anesthetic will rapidly diffuse and anesthetize the pulp of the affected tooth (Figure 6-6). This injection will augment the anesthesia of the second and third molars (Figure 6-7).

Supplemental Greater Palatine Nerve Injection

Most patients with significant molar pain or those undergoing major procedures on the molars (e.g., root canals, extractions) will require supplementation of the anesthesia, which is obtained with upper molar dental block with injection of the fibers of the greater palatine nerve. This technique is performed by injection of 0.1 to 0.2 mL of local anesthetic at right angles to the affected tooth at a point approximately one half of the tooth height (Figures 6-8 and 6-9).

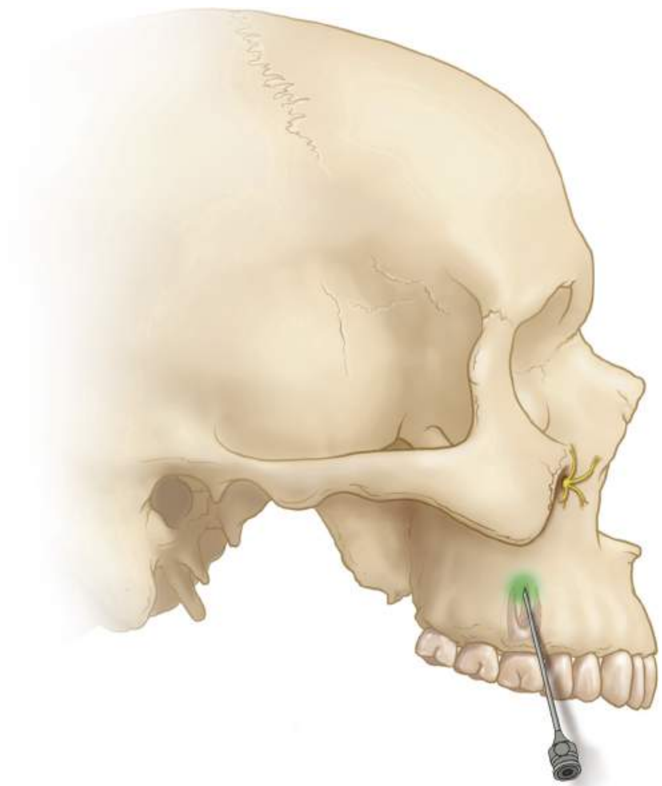

FIGURE 6-3 Needle trajectory for buccal fold injection for the upper molars.

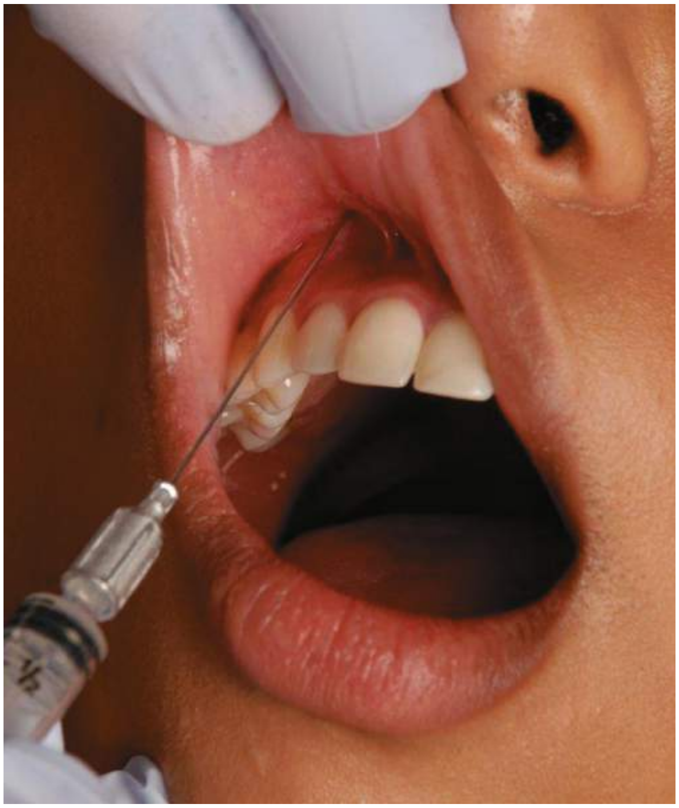

FIGURE 6-2 Buccal fold injection for the upper molars.

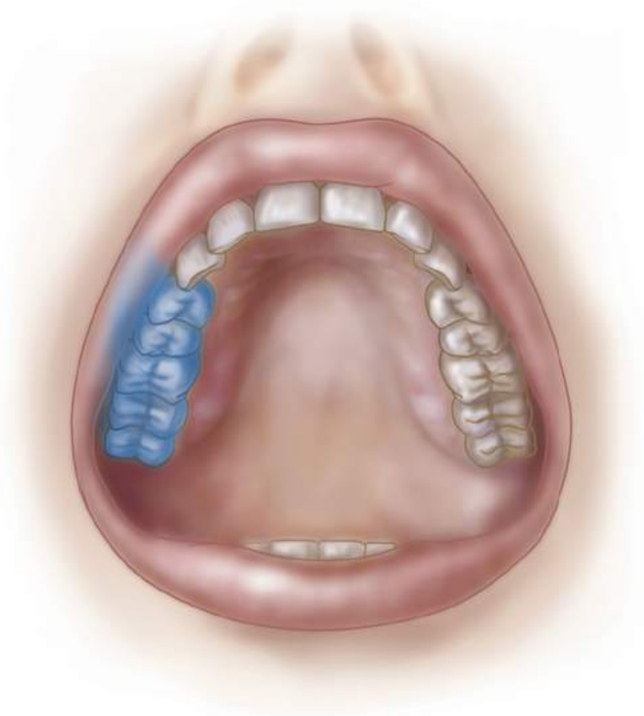

FIGURE 6-4 Spread of anesthesia after buccal fold injection for the upper molars. Note the relative lack of lip anesthesia.