

THERAPEUTIC INJECTIONS FOR PAIN MANAGEMENT

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Types of Therapeutic Injections

This article focuses on the use of therapeutic injections to treat acute and chronic pain syndromes. Discussion of this topic begins with an overview of regional anesthesia, which includes the pharmacology of frequently administered medications and basic information regarding equipment and safety. The spectrum of injection procedures and their indications for specific pain disorders and pathoanatomic regions is addressed to include therapeutic options for the various tissues or structures characteristic of each area or syndrome.

The following anatomical divisions are somewhat arbitrary and overlap in some cases; however, this mode of presentation should prove relevant and accessible by using a format to address pain complaints by region and target tissues located in the spine, extremities, head and face, autonomic nervous system, and some viscera. A discussion of the clinical use of botulinum toxin is incorporated at the end of the article.

Neural blockade and similar injection procedures often are prescribed for therapeutic benefits; however, they also can be useful for diagnostic, prognostic, or prophylactic indications, or for a combination of these purposes.

Therapeutic blocks are appropriate for alleviating acute pain, especially in a self-limiting disorder (eg, postoperative, posttraumatic, or acute visceral pain syndromes). In general, they have been advocated to alleviate acute pain or an exacerbation of chronic pain and to provide direct and localized therapeutic action, especially in patients in whom pain is accompanied by swelling and inflammation. They help the patient (1) maintain an ambulatory or outpatient treatment status; (2) maintain participation in a physical therapy or rehabilitation program; (3) decrease the need for analgesics; and (4) in some cases, avoid or delay surgical intervention.

Sympathetic blocks in causalgia and reflex sympathetic dystrophy (ie, complex regional pain syndromes) permit more effective application of adjunctive treatment techniques including physical therapy and medication. In some cases, therapeutic injections help the practitioner gain patient cooperation, which may have been compromised not only by pain but also by fear, poor nutrition, and deconditioning.

Diagnostic blocks often help the treating practitioner determine the anatomic origin(s) of the patient's pain. These procedures also may facilitate differentiation of a local from a referred somatic pain source, a visceral from a somatic pain source, or a peripheral from a central etiology. Selective blocks can help determine which peripheral tissues are primary pain generators. In cases of presumed complex regional pain syndromes, neural blockade can be used to establish relative contributions of somatic and sympathetic nervous systems.

Prognostic blocks are intended to provide information regarding the efficacy of a planned neurolytic or neurosurgical ablative procedure or potential surgical outcomes. These blocks also may help the practitioner and patient decide whether to proceed with surgery or ablative procedures.

Prophylactic blocks are used to delay and reduce postoperative pain, to prevent complications caused by posttraumatic or visceral pain, to decrease the duration of hospitalization and convalescence, and to prevent development of certain chronic pain syndromes such as autonomic dystrophy and phantom limb pain.

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Guidelines for Therapeutic Application

Numerous technical and medical factors are pertinent to avoid potential pitfalls or complications when considering application of injections for the many indications outlined in the introduction. Historically, these procedures have been used empirically, often resulting in variable or temporary benefit, despite risk and potential complications. For these reasons, some of the basic clinical principles for utilization and safety are reviewed.

Practitioner criteria

A practitioner who intends to perform therapeutic injections should be qualified by education, training, and experience to diagnose and manage the specific disorder(s) to be treated, including the capacity to determine whether diagnostic evaluation has been complete and that verification of the disorder to be treated has been conclusive. Knowledge of the natural history and expected clinical course of these disorders should influence the practitioner's judgment as to what procedure should be performed, necessity of the procedure, and likelihood of success, and lead to true informed consent.

The treating practitioner should be aware of alternative or accessory therapies that can be applied before or following

procedural intervention, and which may enhance the efficacy of treatment. Knowledge of the advantages, disadvantages, and limitations of each procedure, and the ability to manage complications, should be considered requisite. Knowledge of the anatomy and pharmacology of injected substances coupled with adequate experience and technical skill for performing prospective procedures are also requisite. The practitioner should be licensed with privileges to perform therapeutic procedures in the appropriate medical care settings.

Procedural methodology

Prior to performing or even scheduling injection procedures, the practitioner is obliged to assess the patient thoroughly, including the history of the present illness, past medical history, medications, and drug allergies, and the extent to which operant and psychological factors are salient with regard to the illness at hand. Of course, all such information should be documented thoroughly.

Prior to any medical treatment, especially neural blockade or therapeutic injection, the practitioner should inform the patient fully regarding technique, indications for the procedure, operative complications, typical time for convalescence, and cost.

The patient's pretreatment status should be documented carefully. A flow sheet and medical chart to record the procedure and document any complications or side effects from pretreatment to posttreatment is standard. On-call practitioner advice and care should also be available. Any and all adverse effects, whether related to the injection or not; objective observations such as changes in temperature, color, and/or edema affecting an extremity or other pertinent body region; and assessment of therapeutic efficacy should be documented.

Digital video or still photographic documentation of the physiological appearance of the involved extremity or anatomical region and, in some cases, the procedure, provide the practitioner with a visual record of the injection locale, including any preoperative cosmetic problems such as skin lesions, scars, or deformities. Postoperative photographic recording also can be obtained for comparison.

Further objective and meaningful information can be obtained using preoperative and postoperative visual analogue scales (VAS), pain and disability scales, quality-of-life measures, and injection-specific questionnaires. The purpose and medical necessity for therapeutic injection also should be well documented. Appropriate subspecialty consultation may be necessary in some cases to support the preoperative diagnosis and the medical necessity for application of specific procedures.

Furthermore, the use of adjunctive guidance such as electromyography (EMG), ultrasound, and radiologic studies is recommended in some cases. Injections are rarely the only suggested treatment; therefore, expectations regarding the extent to which the procedure may provide pain or symptom relief should be explained to the patient preoperatively. Most therapeutic injections are not curative; therefore, any assumption that a procedure is a panacea should be dismissed.

OVERVIEW: TECHNICAL APPLICATION

Needles and basic manipulation techniques

The application of therapeutic injections and regional anesthesia requires knowledge of equipment that includes needles, syringes, and catheters. The Luer lock is a conical tip that allows easy exchange of needle to syringe and is named after the person who developed it. Today, most needles are disposable, with the bevel cut in 3 planes to minimize tissue laceration and discomfort. Needles that are used for deep injection during regional block should incorporate a security bead on their shafts so that the needle can be retrieved if the needle hub separates from the shaft.

Generally, disposable straight needles with a beveled or pencil-point shaped tip are used for spinal interventional procedures. Spinal and deep injections are best accomplished with a stylet needle, which has an outer cannula through which a smaller needle or catheter can be inserted. The inner stylet seals the cannula and prevents tissue from entering the cannula as the needle is advanced. The stylet should always remain entirely within the cannula when there is forward movement of the needle. Many advocate the use of a short needle bevel to reduce neural and vascular trauma.^[1] Rounded needle tips have been advocated for puncturing the dura to access the subarachnoid space. In theory, rounded tips gently spread the dural fibers and may reduce the incidence of dural cuts that cause post spinal tap headaches. Caution must be exercised with long bevel needles because the soft metal tip is more likely to develop a hook or barb at the tip after striking a bony surface or with prolonged use during a procedure. Furthermore, needles with a smaller caliber (less than 20 gauge) or with a length greater than 3.5 inches are more difficult to steer through tissues of low resistance.

Knowing how to manage the bevel of the outer cannula and inner stylet are key to successful needle navigation. The hub of the needle usually has a notch that corresponds to the face of the bevel needle tip. After puncturing the skin, as the needle is advanced through the deeper soft tissues, the needle tip tends to veer slightly in the direction opposite to the hub notch; therefore, enter the skin as close to the target as feasible. The tendency for a needle to

travel in a curved trajectory can be useful at times and can be enhanced by placing a small 5-10° bend in the tip. When traveling a significant distance with a bent needle tip, the needle must be continually rotated to prevent it from straying off course, which may cause significant tissue disruption. To counter this potential problem, a larger coaxial needle can be placed just proximal to the target, and then if a curved trajectory facilitates steering just beyond the needle tip, a bent needle can be inserted through the larger needle, which allows it to swerve or turn in the direction necessary to reach the anatomical objective.

The needle hub is held with the thumb on top pointing toward the notch. The index and middle fingers are placed opposite the thumb at the junction of the hub and needle. The needle is pushed by the thumb and can be steered by turning the notch in a direction that is 180° opposite to the target. This maneuver places the sharp edge of the needle tip toward the direction that the needle is intended to travel. The stylet should always be contained entirely within the cannula while the needle is moving forward.

Before skin entry, the angle of the needle tip and its trajectory define its course. However, after the needle passes into deeper soft tissues, it cannot be steered by redirecting or pushing it sideways. Bowing of the needle is a technique, whereby, pressure is established both at the skin surface and at the proximal end of the needle. The needle bows toward the surface pressure. The hub is moved in a direction opposite to the notch, causing the needle to arc and the needle tip to travel in the same trajectory as the bow, opposite to the notch. Turning the hub changes the course of the needle, but always in a direction that is opposite to the bowed posture of the needle.

The needle should always be advanced slowly over short distances with frequent monitoring by fluoroscopy. The operating practitioner needs to be aware to move his hands out of the path of the x-ray beam when using intermittent fluoroscopy. The needle tip position can be determined by tissue feel (soft tissue vs bone), fluoroscopic visualization [lateral, oblique and AP planes] and using radiopaque contrast. Fluoroscopic localization requires an AP and lateral of the needle or one fluoroscopy view and contact with an identifiable bony landmark. Contacting bone during the procedure offers a unique opportunity to know needle tip position. Also, when the needle tip is resting on bone, it is unlikely to be in a dangerous venue, such as a blood vessel, neural tissues, or the intrathecal space. Injection of radiopaque dye can be used to further establish certainty of the needle location. Water-soluble contrasts are benign, even when injected into the intravascular or intrathecal space; however, the presence of contrast may obscure view of the needle's tip for continued placement. The operator must know the needle tip's location before injecting any active medication. If injected radiocontrast dye washes away rapidly from the needle tip during the injection be wary, because the contrast may be entering a blood vessel. The dye should stay at the injection site. [2]

Pharmacology: Local anesthetics

The application of any injectable substance can lead to allergic, idiosyncratic, or adverse side effects. Previous suspicious or unfavorable responses may be verified through prior hospital or office records. In some cases, a small amount of the substance in question may be injected subcutaneously to test the patient's reaction to exposure.

Safe and effective use of local or regional anesthesia requires thorough knowledge of the pharmacology of local anesthetics (LAs). Local infiltration for neural blockade can be accomplished by using dilute concentrations of LAs, as they rapidly penetrate the various tissues around targeted nerve endings. When large-diameter nerves are targeted, the quantity of drug reaching the central axonal core is reduced because of incomplete penetration of surrounding epineurium, perineurium, endoneurium, fat, blood vessels, and lymphatics, which can constitute as much as 40% of the peripheral nerve diameter.

Some of the injected substance is absorbed by local blood during its diffusion, which acts as another important mechanism for reducing the amount of drug that actually reaches the nerve axon. Higher concentration of LAs may cause local vasomotor paralysis, which increases local blood flow and enhances systemic absorption. Blood flow through injected tissues can be reduced by using an LA solution mixed with epinephrine, which decreases systemic absorption and improves penetration of the anesthetic to its target. Therefore, the vascularity of various tissues should be considered when deciding on the LA concentration and amount of injectate. Absorption into the bloodstream not only reduces potency of the injected material at the target site, but also increases systemic side effects. Low concentrations of LAs typically are used to block smaller, lightly myelinated and unmyelinated nerve fibers, such as C, A-delta, and B-preganglionic sympathetic fibers.

Several clinical characteristics should be considered when choosing an LA. The latency of onset of anesthetic action is an important clinical property; however, concentration, total dose, distance between the injection site and target, and relative penetrance of the compound also should be considered. Penetrance depends on target-tissue characteristics, including the thickness of superimposed, fibrous, and other intervening tissues. Tissue penetrance of specific LAs determines latency of onset and intensity of induced anesthesia. Duration of LA action depends on pharmacodynamic properties of the anesthetic, concentration, total dose, and vascularity of the region under scrutiny. LA toxicity relates to all of these factors and also to biotransformation.

All LAs have the same basic chemical structure with an aromatic and amino end joined by an intermediate chain. The amino esters use an ester link between the aromatic and intermediate chain. These drugs include cocaine, procaine, 2-chloroprocaine, and tetracaine. Cocaine was the first anesthetic used clinically and continues to be used for topical

airway anesthesia because it is unique among LAs in also being a vasoconstrictor. The amino amides contain an amide link between the aromatic and intermediate chain. These medications include lidocaine, mepivacaine, prilocaine, oral pivalacaine, bupivacaine, and etidocaine.

Lidocaine is a widely used LA because of its rapid onset, potency, and tissue penetration. Within this group bupivacaine is also a popular and frequently used LA for peripheral nerve block and epidural or spinal anesthesia. Commercially available concentrations of this drug range from 0.125-0.75%. Altering the concentration of bupivacaine can elicit a separate sensory or motor neural blockade, ie, lower concentrations primarily induce a sensory block, whereas higher concentrations cause motor blockade. Bupivacaine alters myocardial conduction more dramatically than lidocaine; therefore, the need for cardiorespiratory monitoring during the use of LAs should be emphasized.

Several agents are used to prolong or modify the action of LAs. As already discussed, epinephrine causes vasoconstriction, which reduces vascular and systemic absorption of the drug from the intended site of action, lowers the risk of systemic toxicity, and enhances LA efficacy on the target tissue. Epinephrine is the agent most often combined with LAs, which have a short to moderate duration of action. Epinephrine is contraindicated in some patients because of side effects or drug sensitivity or when a compromise of blood flow should be avoided (ie, when used in distal portions of the extremities, especially with coexisting peripheral vascular disease). Phenylephrine and norepinephrine (NE) also have been used as vasoconstrictors for spinal anesthesia; however, they do not appear to provide any significant advantage over the more commonly used epinephrine.

Alkalinizing agents are thought to facilitate the onset of action and prolong neural blockade when combined with LAs; however, recent double-blind studies in humans have failed to substantiate that this actually occurs. Nevertheless, addition of sodium bicarbonate to bupivacaine still is advocated to produce faster onset of epidural blockade with longer duration.

Corticosteroids

Injectable corticosteroids have been traditionally advocated to treat pain and inflammation associated with a myriad of musculoskeletal conditions, except when infection or skin breakdown is present at the target site, or in patients with poorly controlled diabetes.[3]Several therapeutic actions have been proposed for their beneficial effects.[4] They reduce inflammation by inhibiting the synthesis or release of a number of proinflammatory substances, including arachidonic acid and its metabolites (eg, prostaglandins, leukotrienes), some cytokines (eg, interleukins 1 and 6, tumor necrosis factor- α), and other acute phase reactants.[5]Other proposed mechanisms of action include a direct membrane-stabilization effect, reversible inhibition of nociceptive C-fiber transmission, and modulation of nociceptive input within the dorsal horn substantia gelatinosa neurons.

Continuous large doses of a corticosteroid adversely affect collagen synthesis, and, therefore, connective tissue strength.[6,7]Frequency of injections and dosages must be monitored by the practitioner to prevent generalized or focal immune suppression such as infection or impaired soft tissue healing.[3]Therefore, the amount of corticosteroids that can be applied over time to a specific tissue area can be detrimental, although the exact dose/time curve remains unknown.[3]Concomitant use of medications that alter corticosteroid effects or clearance is usually not salient when injections are provided intermittently.

Practitioner preference among commonly used injectable corticosteroids is often arbitrary. Corticosteroid esters have long been preferred because of their relative safety and efficacy. The relative solubility of these solutions is considered a factor when determining the appropriate injectate.[3]Highly soluble steroids such as betamethasone sodium phosphate-acetate are rapidly absorbed and pose a lower risk for connective tissue injury, such as tendon rupture, fat atrophy, and muscle wasting. Relatively insoluble steroid esters have a longer duration of action.[3]

Corticosteroids are among the most commonly used active substances for spinal intervention. Particulate steroids should not be placed into the cervical foramina, because foraminal arteries, specifically the radiculomedullary artery, can be occluded by the injection. Foraminal artery occlusion is also a consideration between spinal levels T10 to L4. Particulate steroids, when injected into a foraminal spinal artery, can cause paralysis, even death.[8]

Commonly experienced adverse reactions from corticosteroid injections include dizziness, nervousness, facial flushing, insomnia, and transient increased appetite.[9]Flare-up of pain intensity at the injection site may occur, lasting for 24-48 hours in 10% of patients[9]and presumed related to a local inflammatory response to corticosteroid crystals.[3]The likelihood of a flare-up reaction is reduced by using a soluble, rapidly absorbed steroid. Rest and physical therapy are sometimes necessary in these cases. In addition, adverse reactions may occur in persons who have active peptic ulcer disease, ulcerative colitis, active infection, hypertension, congestive heart failure, renal disease, and psychiatric illness.[9]Hyperglycemia in known diabetics warrants careful postprocedural monitoring. Other less serious side effects of corticosteroids include injection site hyperpigmentation, subcutaneous fat atrophy, peripheral edema, dyspepsia, and malaise. Systemic responses frequently occur even in local injections of corticosteroids. Allergic reactions to systemic glucocorticoids in slow-release formulations have been reported to occur up to 1 week after injection.[9]

Spinal interventional procedures

Pain sensitive spinal structures within the 3 joint complex (composed of the disk and 2 posteriorly situated facet joints) include the nerve roots, dura, posterior longitudinal ligaments, outer annular fibers of the disk, facet joints, joint capsules, and cancellous bone. Intraspinous structures without proven pain innervation include the ligament flavum, inner annulus and nucleus pulposus. Spinal interventional techniques can isolate potential pain generators, and also provide therapeutic relief of pain and associated neurologic symptoms. Identification of a pain-producing structure can be inferred when the patient's characteristic pattern is provoked by radiocontrast agents or saline. Furthermore, diagnostic value can be derived from the patient's response to an injected local anesthetic, and sometimes the use of corticosteroids or neurolysis can provide durable therapeutic value. Safety and accuracy are enhanced when the practitioner performing these procedures is knowledgeable of spinal anatomy, experienced with the use of fluoroscopy, and skilled at steering needles within the soft tissues of the back.

The decision to perform a spinal interventional procedure should be based on sound medical evidence. Evidence-based medicine is a strategic approach to managing cost by managing care. It is the judicious use of the current best evidence for making decisions about the care of individual patients. Therefore, when clinical and research evidence support the benefit of a specific procedure for an individual patient problem, it should be advocated. If medical evidence suggests that no clear benefit is derived from a procedure for a specific indication, or if the procedure may harm the patient, either directly through adverse events or indirectly by wasting medical resources, then it should be avoided. When insufficient evidence exists to determine whether the procedure is beneficial, then the operating practitioner must practice the most conservative approach. Manchikanti and colleagues have defined guidelines that classify the strength of experimental evidence that supports decisions as to whether specific interventional pain procedures should be performed. This analysis includes the prevalence of specific spinal pain generators and the efficacy of performing specific procedures for therapeutic or diagnostic purposes.[10]

Although, fluoroscopy has revolutionized pain management by increasing the precision, safety, comfort, and outcomes of interventional techniques, the number of procedures and a variety of providers has increased. All practitioner interventionalists must be adequately trained and experienced to prevent adverse events from harming patients and coworkers. All somatic and spinal injection practices carry finite plausible risks that range from medication allergies or side effects, unwanted violation of body structures with neural or vascular content, to the ultimate possibility of death as a treatment outcome. Complications that are common or unique to each procedure will be discussed. However, the thesis by which this chapter exists is only its capacity to provide information. It does not provide the skill and knowledge that are necessary to perform the outlined interventional methods. University and other ABMS-accredited fellowship programs are now commonly offered. Pain societies and certification agencies such as the American Board of Anesthesia and the American Society of Interventional Pain Physicians provide learned guidelines, assistance through teaching and coursework, and board certification examinations for physician interventionalists. Expertise in performing the outlined procedures is a matter of forethought, not afterthought.

Fluoroscopy and radiation safety

Fluoroscopy has transformed interventional pain management, not only for more precise needle placement, but also for venturing into new treatment venues, especially within the spinal canal. Precise needle placement allows practitioners to address multiple spinal pain generators with injections that include placement of radiographic contrast, local anesthetics, and corticosteroids into the epidural space, intra-articular facet joints, sacroiliac joints, and intervertebral discs. Symptomatic facet joints can be identified by median branch nerve blocks and then ameliorated with radio-frequency neurotomy or chemical neurolysis. New technologies have evolved, such as the use of spinal cord stimulators and a host of intradiscal procedures, including electrothermal coagulation, percutaneous mechanical disk decompression, laser disc decompression and radiofrequency intradiscal/annular neurolysis. Other new treatment methods include vertebroplasty and kyphoplasty for vertebral fractures. Fluoroscopy allows more precise localization of both stellate and lumbar paravertebral sympathetic blocks, visceral sympathetic blocks, celiac plexus and superior hypogastric plexus blocks, and neurolysis of the Impar ganglion.

Several studies have demonstrated the comparative accuracy of experienced injectors and anesthesiologists using fluoroscopy compared with previous blind injection techniques and have shown a superior success rate with imaged needle guidance.[11]

Manchikanti et al advocate fluoroscopy as medically necessary for the performance of epidural corticosteroid injections.[12] Dye injection may reveal incorrect needle placement or inadequate penetration of the injectate to the level of pathology. Fluoroscopy eliminates the question of incorrect or suboptimal needle placement as compared with blind injections and can provide evidence of accurate needle positioning. Documentation of dye spread often mimics the probable flow of corticosteroids and other active medications, and therefore may correlate with the patient's response to treatment. Unintentional intravascular injection may occur during procedures despite negative aspiration through the needle. Vascular locations can be suspected when the contrast dye seems to wash away from the site of the needle tip after it is injected. Limited reasons for not using fluoroscopy include the avoidance of radiation, the cost of fluoroscopy, or allergy to contrast agents.

The fluoroscopy machine

The fluoroscopy machine is primarily composed of an x-ray tube, image intensifier, C-arm, and control panel. The electron flow, called tube current, is generated through an electrically heated negatively charged filament (cathode) and is expressed in milliamperes (mA). The x-ray tube fires a beam of electrons through a high voltage vacuum tube forming x-rays that are emitted through a small opening. X-rays are generated by engaging a high-voltage switch with the output expressed as the kilovolt peak (kVp). These x-rays pass into and through human tissue creating electrically charged ions. The image intensifier collects electromagnetic particles that pass through the patient and transforms them into a usable image that can be visualized on a television monitor. X-ray production ceases immediately when the switch is released. For this reason, radiation management in fluoroscopy is best accomplished by keeping the amount of beam-on time as short as possible.[13]

The C-arm facilitates optimal positioning of the fluoroscope for the practitioner to get the most favorable view, (eg, posterior-anterior, oblique, and lateral views of the patient). The control panel allows the technician to manually adjust the quality of the image or leave it to the automatic brightness control (ABC). The quality of the image contrast depends on the balance between the tube voltage and current. A higher kVp setting increases the penetrability of the x-ray beam, but reduces the contrast of the x-ray image, whereas the tube current increases both intensity and penetration. Balance of the tube current and tube voltage (kVp) creates the optimal contrast and image resolution. This is usually accomplished by the ABC system, whereby the computer automatically analyzes the image contrast and makes the appropriate adjustments to the kVp and mA to achieve the best balance between contrast and brightness of the image with the lowest dose-rate to the patient. Dose-rates are greater depending on the thickness or size of the patient. As patient size increases, image quality decreases, patient dose increases, and exposure rates to personnel increase. The control panel also allows for magnification and collimation of the image.[14]

Radiation concepts and safety

X-rays are a form of electromagnetic energy. When transferred through matter, x-rays ionize human tissue and produce electrically charged ions that can induce molecular changes, potentially leading to somatic and genetic damage. Radiologic nomenclature describes radiation quantities using terminology such as the absorbed dose, effective dose, equivalent dose, and Dose-area-product. A roentgen (R) measures exposure to ionizing radiation equivalent to the electrical charge per unit mass of air ($1R=2.58 \times 10^{-4}$ coulombs/kg of air). The concentration of energy that is deposited locally into a tissue is called the absorbed dose. This is measured in units of gray (Gy) or milligray (mGy). One gray of absorbed dose is equivalent to the energy deposition of 1 joule in 1 kg of tissue mass. Doses lower than 1 Gy generally do not cause notable acute effects other than slight cellular changes. The absorbed dose rate describes the rate of dose accumulation in mGy/min. A typical skin entrance dose rate from fluoroscopy is about 30 mGy/min. Effective dose is the quantity of radiation exposure affecting people who are not in a stationary or typically uniform space. It is the hypothetical dose received by the entire unprotected human body and poses the same health risk as the nonuniform dose received by an individual not wearing a protective apron. For the purposes of radiation protection, regulatory limits of whole-body exposures to personnel are given in terms of effective dose. This information is extracted from the data generated by film badges or other types of personal radiation monitors.

Therefore, the radiation-absorbed dose is the amount of energy deposited into human tissue from ionizing radiation sources and is measured in units of gray (Gy). Biologic effects of radiation are caused by the ionization of water molecules within the cells, producing highly reactive free radicals that damage macromolecules of DNA. The acute effects occur at relatively high-dose levels, such as those given during radiotherapy treatments or from accidents. Chronic effects are more often the result of long-term low-dose exposure. The most common radiation-induced injuries affect the skin. Unlike a thermal burn, x-ray injuries develop slowly and may not become apparent until days or weeks later. Potential effects vary in severity from erythema to dermal necrosis and skin cancer. Additionally, the probability of induced cancer or leukemia is increased in the exposed individual. The latent period between radiation overexposure and cancer may be as short as 2 years.

To measure the effective dose (whole body dose) from occupational radiation exposure, the measure termed rad is converted to the unit of occupational exposure, which is designated as the radiation-equivalent-man (REM). The unit of dose equivalent to REM is measured by using the sievert (Sv); one REM is equivalent to 1 rad, and 100 REM is equivalent to 1 Sv. Radiation dose equivalents of 0.25 Sv (25 REM) may lead to measurable hematologic depression. Whole body total radiation doses exceeding 100 REM may lead to nausea, fatigue, radiation dermatitis, alopecia, testicular disturbance, and hematologic disorders. A maximal permissible dose (MPD) is the upper limit of the allowed radiation dose that an individual may receive without the risk of significant side effects. The annual whole body MPD limit for physicians is 50 mSv. The annual MPD for the lens of the eye is 150 mSv, and for the thyroid, gonads and extremities it is 500 mSv. The fluoroscopy's x-ray tube should be kept as far away from the patient as possible. Federal regulations limit the maximum output for C-arm fluoroscopy to 10 R/min at 12 in (30 cm) from the image intensifier. Beam collimation reduces the area being irradiated, thereby reducing the amount of x-rays received by the patient. The use of live fluoroscopy should be minimized as much as possible. Furthermore, magnification should be limited since it increases the amount of radiation to human tissue. Image magnification by a factor of 2 increases the amount of radiation by 4 times.

Radiation exposure to ionizing radiation is unavoidable when performing fluoroscopic procedures. Only necessary personnel should be present in the fluoroscopy room. The primary source of radiation to the practitioner during such procedures is from scatter that is reflected back from the patient. Less prominent is the role of radiation leakage from the equipment. The cardinal principals of radiation protection are (1) maximize the distance from the radiation source, (2) use shielding materials, and (3) minimize exposure time. Radiation scatter can also be reduced by using the lowest tube current (mA) that is compatible with a good x-ray image. In conventional fluoroscopy, the x-ray tube is located beneath the table and the image intensifier is above the table. With a horizontal table, in this arrangement, most of the radiation scatter is in a downward direction and is absorbed into the floor or side panels of the table. In the opposite arrangement, it is often difficult to get adequate shielding to medical personnel. As mentioned, the beam-on time is the most important variable for controlling radiation exposure and should be kept to a minimum; most fluoroscopy machines are armed with a 5-minute alarm.

X-ray shielding can be fixed or mobile, including the commercially available protective apparel. Fixed shielding includes the thickness of walls, doors, and protective cubicles, which should have a lead equivalent of 1-3 mm. Mobile shielding is appropriate during fluoroscopy when a member of the staff needs to remain near the patient. Specific items of apparel that are used for personal shielding include lead aprons, gloves, thyroid shields, and glass spectacles.

Typically, practitioners and assisting personnel are supplied with monitoring equipment in the form of a radiation or film badge that is packed with photographic film. These clips are typically light and slim for convenient placement on conventional clothing and apparel. Usually a "color badge" is worn outside the apron on the upper portion of the body, usually on the upper edge of the thyroid shield. This badge approximates radiation exposure to the lens of the eye. A second "behind the apron" badge is worn underneath lead apparel and clipped onto the waist of the practitioner. X-ray readings from this badge represent the actual dose to the gonads and major blood-forming organs. Also, a finger or ring badge can be worn with the film facing the underside part of the hand nearest the radiation source. Badges may also be placed on protective eyewear. These badges are usually processed monthly to monitor the type and amount of radiation exposure received by each clinical participant. Results are reported as monthly and 12-month accumulated dosages. Prompt exchange of badges on a monthly basis is required in most medical facilities.

Radiocontrast agents

Radiographic contrast agents aid in the localization of anatomical structures. Iodine atoms within these agents provide greater x-ray attenuation compared with human tissues, including bone. Osmolality describes a measure of the numbers of particles in a specific solution. The hyperosmolality of contrast agents relates directly to their toxicity. Second-generation radiocontrast agents have more physiologic properties, are labeled nonionic, and are more commonly used for spinal injections. The 2 most commonly used radiocontrast agents are iopamidol (Isovue-M) and iohexol (Omnipaque). Both are absorbed rapidly into the bloodstream from intrathecal, epidural, and paraspinal tissue injections. Plasma levels are measurable within an hour after injection. The mean half-life is 12 hours and 80-90% is excreted via the kidneys within 24 hours with minimal excretion via fecal route. Adverse reactions may be chemotoxic, osmolar-related, or allergic. Also, 90% of adverse effects occur within 15 minutes of exposure.

If an allergic reaction is suspected, patients should be observed for up to 60 minutes. The primary concern when using contrast media is unintentional intrathecal injection. For this reason, the above-mentioned water-soluble contrast media are recommended: iohexol (Omnipaque) or iopamidol (Isovue). Radiologic contrast media are not licensed for intrathecal use, but these 2 specific radiocontrast agents have not been reported to cause adhesive arachnoiditis and exhibit a low risk of seizures and neurotoxicity.

Patients at greater risk for an adverse reaction to radiocontrast media include those with a history of a previous adverse reaction, especially allergy. Any question regarding an allergic reaction can be avoided by giving oral prednisone 20-50 mg, ranitidine 50 mg, and diphenhydramine 25-50 mg orally 12-24 hours prior to exposure by injection. An additional 25 mg of diphenhydramine can be given by IV immediately before contrast injection. Further caution is required when administering radiocontrast to patients with asthma; allergy/atopy; cardiac disease with decompensation, unstable arrhythmia, or recent MI; renal failure/nephropathy; feeble individuals with general debility (especially infants or the elderly); and patients with dehydration, metabolic disorders, or hematologic disorders. Adverse reactions vary from chemotoxic reactions (such as thyrotoxicosis or nephrotoxicity) hyperosmolar responses, or more typical allergic responses characterized by vasomotor responses, cutaneous reactions, bronchospasm, cardiovascular effects (hypotension), or anaphylactoid reactions.

Although fluoroscopy has revolutionized the precise and accurate practice of interventional pain management, radiation safety training is required for any practitioner who uses fluoroscopy in his practice. Furthermore, injectable radiocontrast media and active therapeutic agents require additional knowledge. Practice in this area of subspecialty requires additional training through recognized medical certification agencies or societies.[15]

Adverse Effects and Complications of Neural Blockade

Systemic toxic reactions to LAs can result from high blood levels of the drug due to accidental intravenous (IV) infusion of all or part of the therapeutic dose, injection of an excessive amount of drug, or abnormal rates of absorption and biotransformation of the drug. Typically, these reactions demonstrate a combination of cardiovascular,

respiratory, and central nervous system side effects that range from mild to severe.

Mild reactions occur when systemic blood levels of LA rise above the usual physiologic levels. Patients may experience dizziness, vertigo, tinnitus, headache, anxiety, tachycardia, hypertension, tachypnea, dysarthria, metallic taste, and nausea.

Moderately severe reactions are manifested by abnormal mental status including somnolence, confusion, and sometimes loss of consciousness. Muscular twitching may progress to generalized motor seizures and usually is accompanied by hypertension and tachycardia that require immediate practitioner action with particular attention to proper ventilation.

Severe toxic reactions from marked overdoses of LA usually are evinced by rapid loss of consciousness with hypotension and bradycardia. Respiratory depression and arrest may accompany other signs of severe central nervous system and cardiovascular depression. If prompt treatment is not instituted, progression to complete respiratory and cardiovascular failure with death may result.

Whenever a systemic toxic reaction is suspected, oxygen administration is justified to reduce the risk of hypoxia. With recurrent seizures, a patent airway must be maintained, including tracheal intubation and artificial ventilation when necessary. Small doses of fast-acting anticonvulsant agents, such as diazepam or lorazepam, can be considered when seizures are recurrent without interictal recovery of consciousness or for continuous seizure activity lasting more than 20 minutes. Cardiovascular monitoring is essential, coupled with appropriate fluids and medication support. Other undesirable systemic reactions to local and regional analgesia include psychogenic reactions, which often are highlighted by fear and anxiety prior to the procedure.

During or after the procedure, patients may experience light-headedness, tinnitus, hyperhidrosis, tachycardia, skin pallor, hypotension, and even syncope. Any adverse reactions should be observed carefully to ensure that symptoms are not due to toxicity or allergy. Management consists of placing the patient into a recumbent position, administering oxygen, and monitoring blood pressure. In some cases, judicious IV infusion of ephedrine may be necessary to alleviate hypotension. Not infrequently, epinephrine in an LA solution can contribute to uncomfortable or adverse side effects, including apprehension, palpitations and tachycardia, dizziness, diaphoresis, and skin pallor. If severe hypertension develops, then treatment with vasodilators or other hypotensive agents is appropriate.

Allergic reactions can occur following repeated exposure to specific LAs and are characterized by urticaria, arthralgia, and edema of eyelids, hands, joints, and larynx. Severe laryngeal edema requires prompt attention to maintain airway patency and may necessitate emergency tracheostomy. Although rare, idiosyncratic reactions may result in sudden and rapid cardiovascular and respiratory collapse leading to death. Treatment includes prompt establishment of an airway, artificial ventilation, oxygen administration, cardiac monitoring, and medication support with vasopressors.

Neurological complications may result from systemic reactions or be due to specific procedures. For example, injuries to peripheral nerves may result from direct trauma including localized hematoma, compression by tourniquet, unintentional neural traction, compression due to positioning, or injection of an excessively high concentration of LA. Complications following subarachnoid or epidural injections can result from direct spinal cord or nerve root trauma, spinal cord compression by hematoma, or spinal cord ischemia.

Direct neural damage is most often reported with brachial plexus blocks. Direct intraneural injection often is attributed to the practitioner's negligence or lack of skill but can occur with highly skilled and experienced interventionists. Needles with a low bevel angle ($<45^\circ$) may contribute to a lower incidence of such complications. Postblock neuropathy can occur immediately after the block or within the first 7 days; however, recovery is common over the ensuing 2-3 months.

Accidental injection of LA into the subarachnoid space sometimes complicates paravertebral blocks aimed at addressing somatic or sympathetic neural structures, such as the stellate ganglion. If the level of anesthetic-induced spinal cord dysfunction is as high as C4, respiratory support, including artificial ventilation, may be necessary. Occasionally, withdrawal of 10-15 mL of cerebrospinal fluid (CSF) reduces CSF concentration of the misplaced LA. Hypotension also can result from unintentional extensive subarachnoid or epidural blockade, or in some cases, from paravertebral sympathetic or celiac plexus blockade.

Pneumothorax is a potential complication from thoracic paravertebral, supraclavicular brachial plexus, intracostal, and celiac plexus blocks. Occasionally, trapezius and other apically directed intramuscular injections also might lead to pneumothorax. Symptoms can develop within minutes but more often develop over several hours. Frequently, patients who experience injections that violate the respiratory space complain of tasting the anesthetic followed by hoarseness. Radiographic evaluation is obligatory in cases in which this complication is suspected.

Injection site hematomas are usually minor complications associated with the use of large needles having a dull bevel or hook, except in patients with a bleeding disorder or taking anticoagulant medications. Diagnosis is usually evident by subcutaneous extravasation of blood, and in some cases, neural deficit, which may be slow to resolve. In cases of localized hematoma, initial use of ice and pressure is warranted to slow or stop the bleeding. Occasionally, this

complication may require ultrasound or other imaging studies to document the size and location of the hematoma.

Therapeutic injections for extra-axial soft tissue structures

Therapeutic injections frequently are used as a mode of treatment in general or subspecialty practices, especially orthopedics, physiatry, and rheumatology. Many musculoskeletal disorders respond amenable to injections, including intra-articular and extra-articular tissues of many synovial joints, bursae, muscles, and tendons. Pain from extra-axial articular structures often is managed best by the aforementioned subspecialists.

Understanding a few key principles can help the neurologist determine the structural anatomy of an articular pain syndrome and respond efficiently by specialty referral, especially when certain symptoms indicate a potentially serious etiology. In most cases, patients with generalized arthralgia and arthropathy should be referred to a rheumatologist; therefore, this article concentrates primarily on localized pain disorders. In fact, the neurologist often is asked to differentiate whether pain is localized to a joint or periarticular structures or is referred from diseased neural structures.

Pain referral from joints or other soft tissue structures typically does not assume a myotomal or dermatomal pattern. Pain arising from superficial soft tissue structures that can be identified by palpation often permits more precise localization of the causative tissue or structure. However, pain that is referred from extra-axial joint capsules and other periarticular structures, such as ligaments, tendons, bursae, and muscles, may be more difficult to differentiate. Pain from bone and periosteum is usually well localized and rarely radiates; however, this discrepancy between "soft" and "hard" structures remains unexplained.

The manner in which the pain from symptomatic joints responds to biomechanical stressors is often the key to localization and causation. Pain that is worse when the joint is used suggests a mechanical etiology, especially if improved with rest. Pain in bed at night should bring about concern for a serious underlying etiology and almost always requires investigation. Persistent pain that does not fluctuate despite activity or rest is also worthy of diagnostic inquiry. Psychogenic or operant pain frequently is described as continuous and often more intense and disabling with certain activities, eg, worse at work and better with recreation. Pain and stiffness that are present in the early morning or after inactivity may be a harbinger of inflammatory arthropathy in extra-axial and axial joints. Patients with monoarticular deformity, swelling, stiffness, and warmth should be referred to the appropriate musculoskeletal specialist for evaluation.

Many common afflictions of extra-axial soft tissue structures are amenable to management by a neurologist who is skilled in the evaluation and treatment of musculoskeletal disorders. Bursae are fluid-filled sacs that facilitate smooth movement between articulating structures. Subcutaneous bursae, such as the olecranon and prepatellar bursae, form in response to normal external friction. Deep bursae, such as the subacromial bursa, form in response to movement between muscles and bones and may or may not communicate with adjacent joint cavities. "Adventitious" bursae form in response to abnormal shearing stresses (eg, over first metatarsal head) and are not uniformly present.

Acute or subacute bursitis (most often affecting subacromial, subscapular, prepatellar, and trochanteric bursae) frequently presents with severe disabling pain that can be relieved promptly by injection of LA. Depending on the size of the targeted bursa, a dilute solution of bupivacaine (0.25-0.5%) with epinephrine 5 mg/mL, with 40 mg of methylprednisolone (Depo Medrol) or an equivalent corticosteroid (ie, Celestone), is often dramatic in its effect. If the bursa is swollen and contains fluid, aspiration should be performed prior to injection for laboratory studies including cultures for a possible infectious agent.

Tendons act as functional anatomical bridges between muscle and bone. Tendinitis is also a common cause of outpatient evaluation for moderately severe to severe, often disabling, pain. Among the most frequent syndromes are bicipital tendinitis, lateral epicondylitis (tennis elbow), medial epicondylitis (golfer's elbow), and supraspinatus (rotator cuff) tendinitis. Long-acting LAs, such as bupivacaine, coupled with a long-acting corticosteroid are often effective. Repeated use of corticosteroids may risk toxicity to the soft tissues, and long-term use can result in adverse systemic effects that are associated with Cushing syndrome. Occasionally, patients experience a "steroid flare" and develop increased pain in the injection site over 24-48 hours; however, local beneficial effects usually follow after the flare resolves. Exercise and physical modalities, including ice and heat, are fitting adjuncts. LA infiltration alone without corticosteroids can be repeated until permanent benefit is achieved.

Muscle spasm and myofascial pain (ie, trigger points) and treatment of syndromes considered controversial by some, such as that caused by the piriformis and scalene muscles (thoracic outlet syndrome), are other commonly considered indications for injection treatment. The tenets of managing these syndromes must be emphasized, however; therapeutic injections are considered adjunctive to an overall treatment plan that includes postural correction, ergonomic modification of contributory occupational factors, appropriate strengthening and flexibility exercises, and concomitant use of physical modalities.

Painful scars following injury or surgery also may be associated with pain and hyperesthesia. Infiltration of LA, sometimes accompanied by corticosteroids, has been reported to be beneficial in many cases. Concomitant topical or oral agents may be useful, as well as application of transcutaneous electrical stimulation (TENS).

Neuromata can develop in nerves that are entrapped subsequent to traumatic neurosection or following surgery for amputation. Infiltration with LA is useful not only from a therapeutic standpoint but also diagnostically. LA without epinephrine mixed with a depot corticosteroid can suppress spontaneous ectopic discharges suspected of producing pain and paresthesia. Supplemental treatment with anticonvulsants may improve outcome if relief is incomplete.

Intra-articular injections of a dilute solution of LA, usually in combination with corticosteroids or articular lubricating agents, frequently are advocated for severe pain associated with chronic degenerative arthritis, especially in weight-bearing joints. Intra-articular injection of LAs into spinal facet joints is discussed in a later section of this article; however, injection of extra-axial joints is considered beyond the scope of the primary theme and the audience addressed in this article.

Types of Neural Blockade

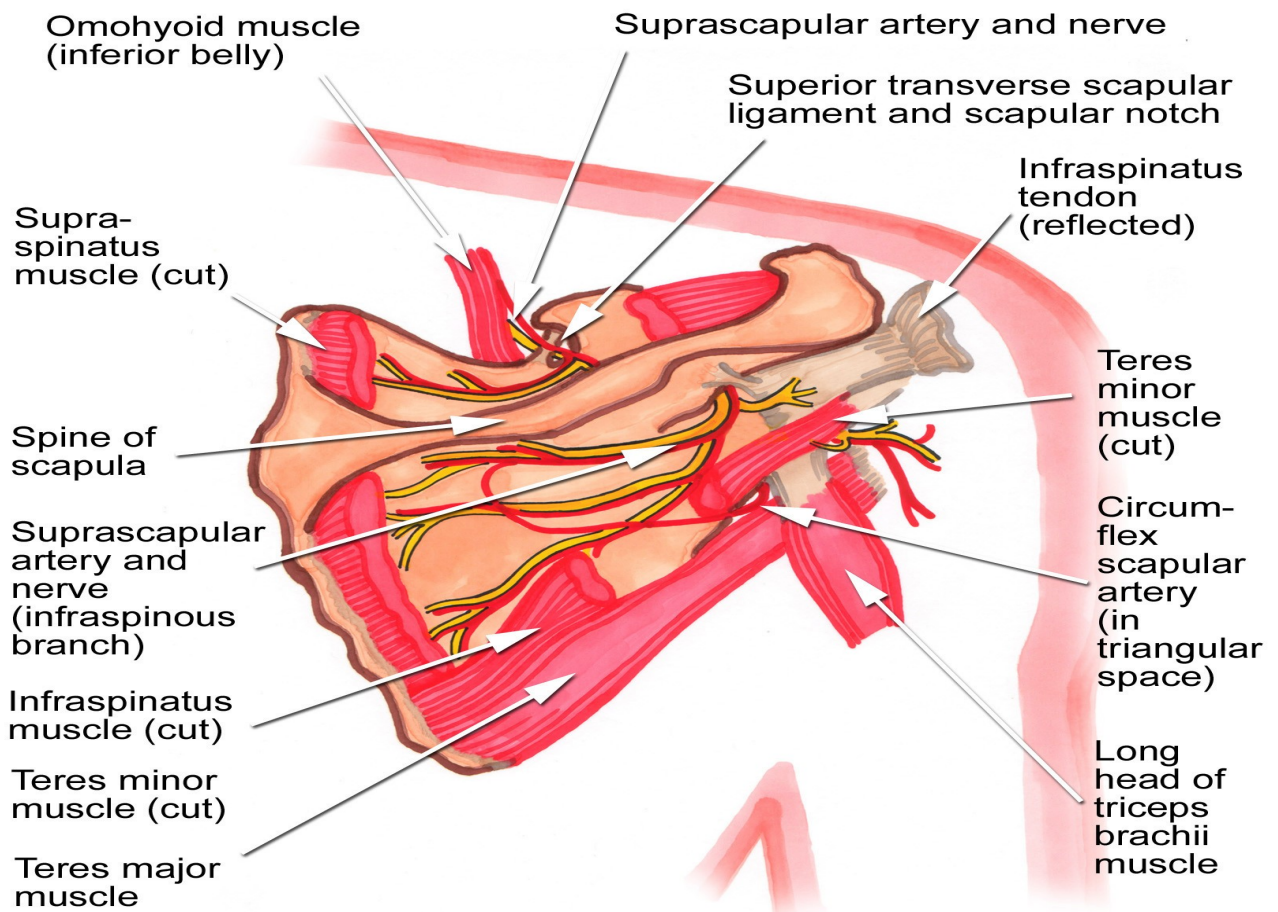
Several somatic and peripheral neural blockade procedures are useful for therapeutic and diagnostic purposes. Although the opportunity to block specific nerves can be considered limitless in the hands of an experienced interventionist with appropriate radiographic guidance, only some of the available procedures are mentioned to highlight their usefulness as potential tools for a neurologist involved in the diagnosis and treatment of pain.

Suprascapular nerve block

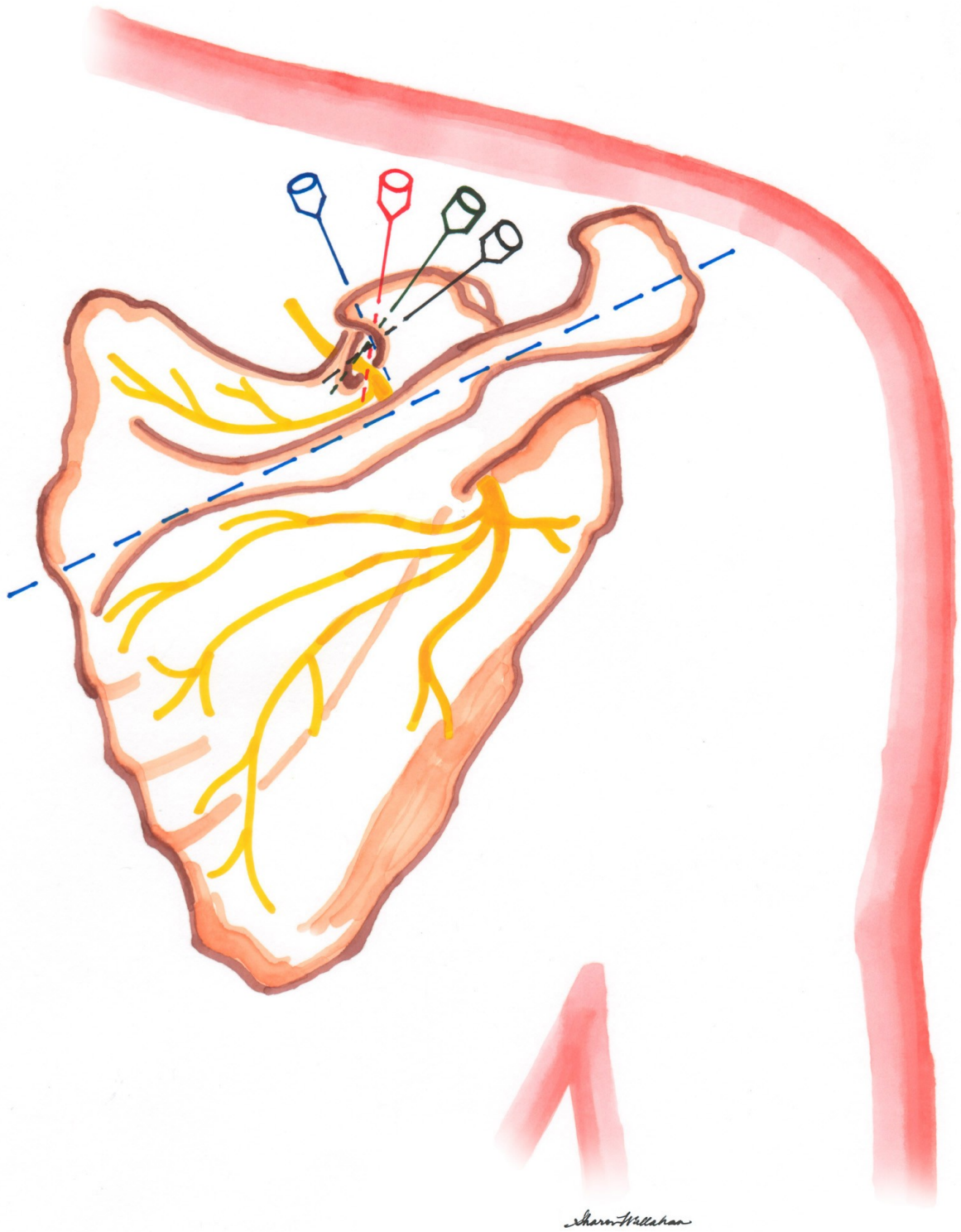
The suprascapular nerve branches from the brachial plexus and serves as the primary sensory supply for the shoulder joint. Suprascapular nerve block can be helpful for the management of severe pain caused by bursitis, periarthritis, or arthritis when intra-articular and periarticular injection of LA and steroids are contraindicated, ineffective, or to be avoided.

Suprascapular nerve block provides anesthesia to the shoulder joint, which allows physical therapy to implement improved range of motion caused by adhesive capsulitis or excessive periarticular muscle guarding. Technically the procedure is easy to perform; however, satisfactory blockade is not achieved uniformly in all cases. When blockade is inadequate, concomitant use of radiography or a peripheral nerve stimulator can provide more accurate placement of the needle and improve anesthetic administration.

To perform a suprascapular nerve block, the practitioner locates the suprascapular notch by first forming 2 bisecting lines—one extending along the spine of the scapula and another that bisects this line and extends to the inferior angle of the scapula.



Using the technique advocated by Bonica, an 8-cm, 22-gauge needle is introduced through a skin wheal of LA placed in the outer triangle about 1.5 cm from the bisection point. The shaft of the needle is directed anteriorly, caudally, and medially into the supraspinatus fossa just lateral to the suprascapular notch. The needle is withdrawn until its point



Sharn Willahan



lies within the subcutaneous tissue and then re-introduced to a point that is approximately 5 mm medial to the first contact. The needle should enter the notch; contact with the nerve is verified if paresthesia is evoked. If no

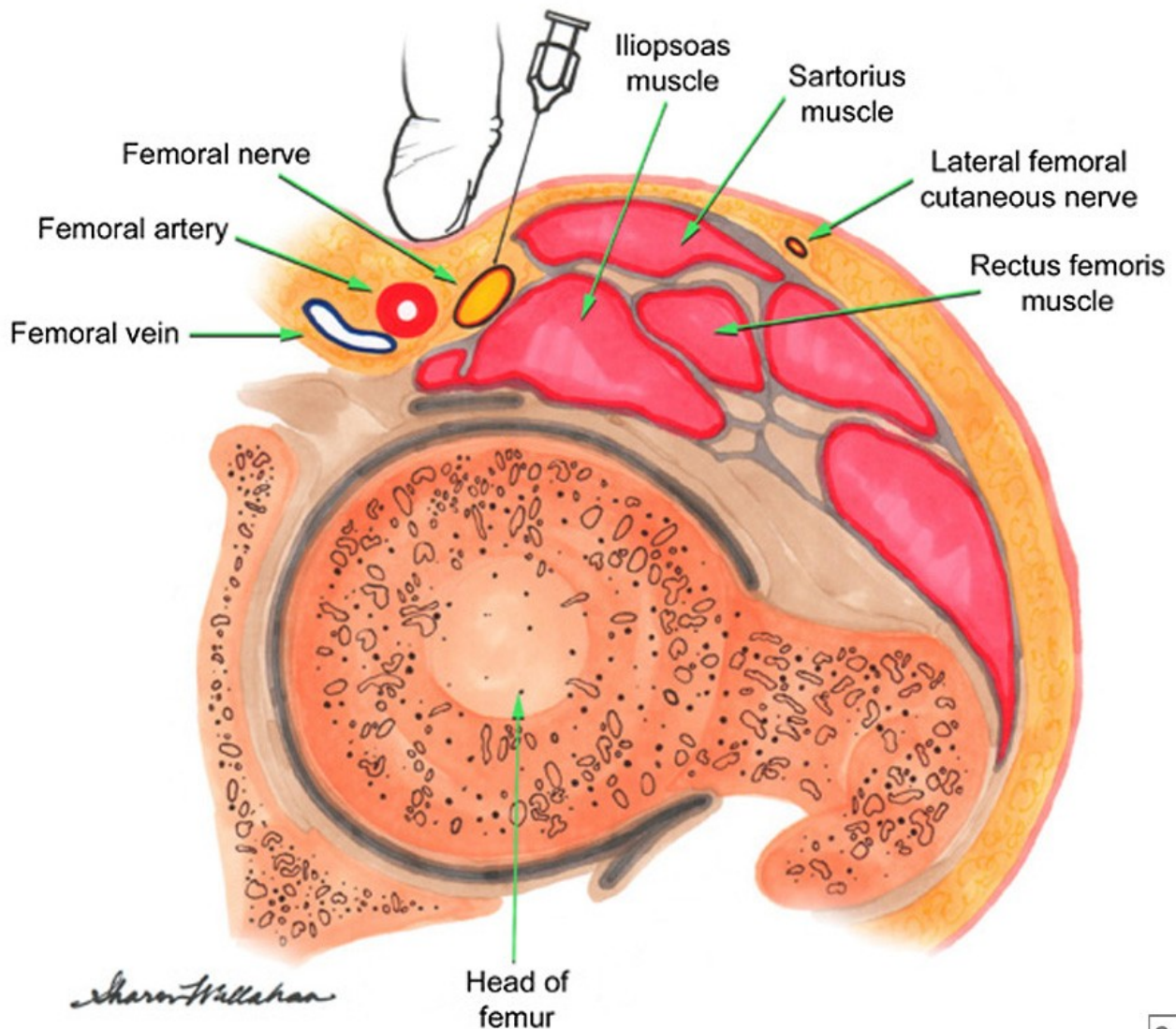
paresthesia is elicited, sequential insertions may be necessary, or location of the nerve can be facilitated by electrical nerve stimulation.

Bupivacaine (3-5 mL) or other long-acting LA, in addition to a short-acting LA, should provide an adequate block for diagnostic purposes, and thereafter, allow appropriate physical therapy intervention.

Femoral nerve block

Femoral nerve block just below the inguinal ligament can be used as a diagnostic tool in patients who present with anterior thigh pain or can be combined with a sciatic nerve block to produce sympathetic neural blockade of the lower extremity. Femoral nerve block can alleviate severe pain related to posttraumatic or postoperative causes (eg, fracture of the neck of the femur).

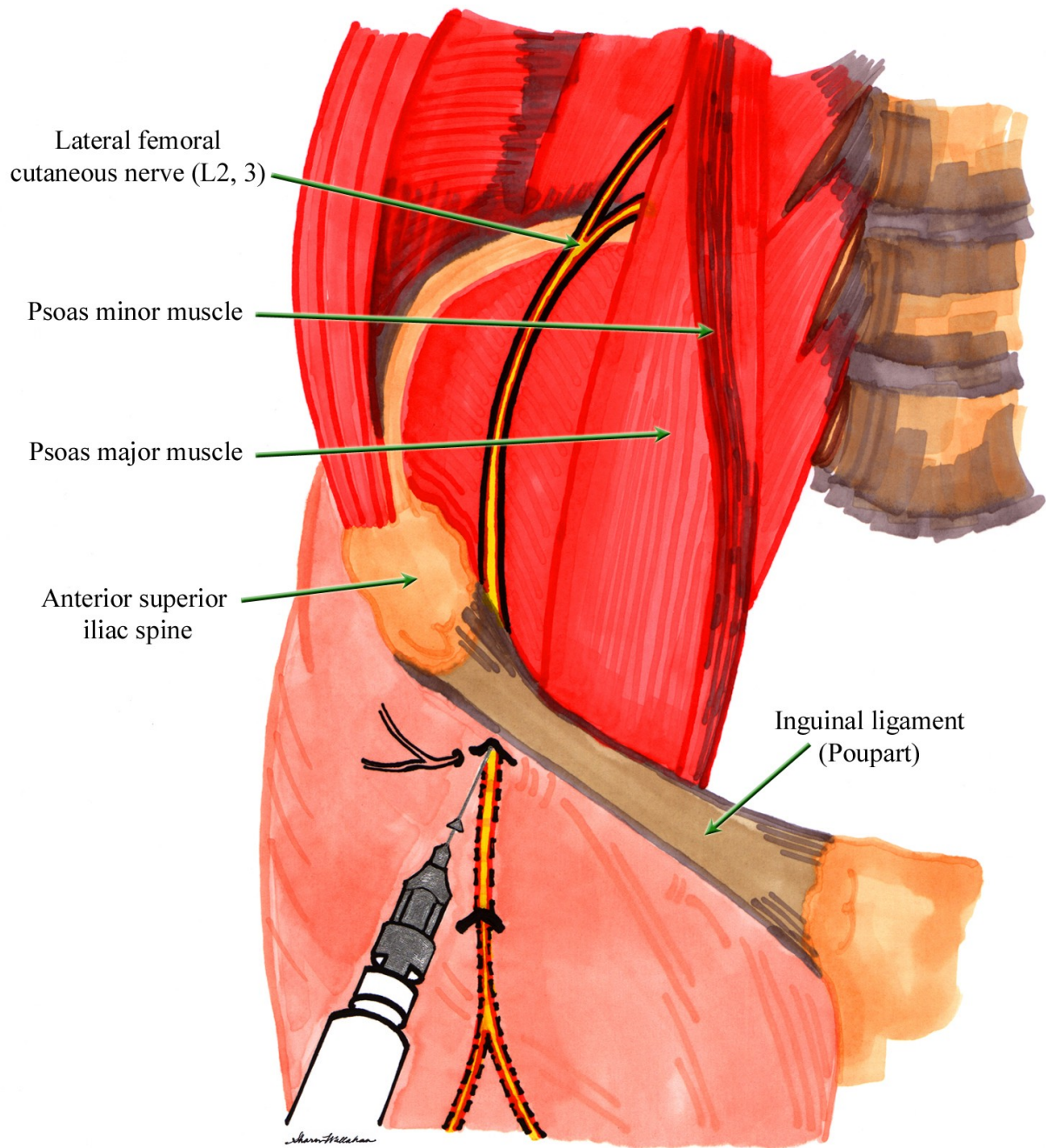
Using the technique described by Bonica, this procedure is performed with the patient in a supine position. The midpoint of a line joining the anterosuperior iliac spine and pubic tubercle usually overlies the femoral artery. A short-acting LA is employed to raise a skin wheal approximately 1 cm lateral to the junction of the femoral artery in the inguinal ligament.



Femoral nerve block technique (cross-sectional view). See text for details.

While palpating the artery under the second finger of the left hand, a 5-cm, 22- or 25-gauge, short-beveled needle is introduced with the right hand through the skin wheal and advanced perpendicularly through the skin until paresthesia is elicited in the distribution of the femoral nerve. An electrical nerve stimulator can be used if paresthesias are not elicited easily. Usually 8-10 mL of 1% lidocaine with epinephrine produces analgesia for 3-4 hours, whereas the same volume of 0.25% bupivacaine with epinephrine produces analgesia for 6-8 hours. If longer analgesia is required, the concentration of bupivacaine can be increased to 0.5% with epinephrine or a continuous block can be applied by placing an infusion catheter at the site.

A lateral femoral cutaneous nerve block confirms the presumptive diagnosis of lateral femoral cutaneous neuralgia or meralgia paresthetica and may provide symptomatic relief. Using the technique described by Bonica, a 5-cm, 22- or 25-gauge, short-bevel needle is introduced through a skin wheal of 1% lidocaine that is 1.5 cm caudal to the anterosuperior iliac spine just below the inguinal ligament at an angle of approximately 60° to the skin.

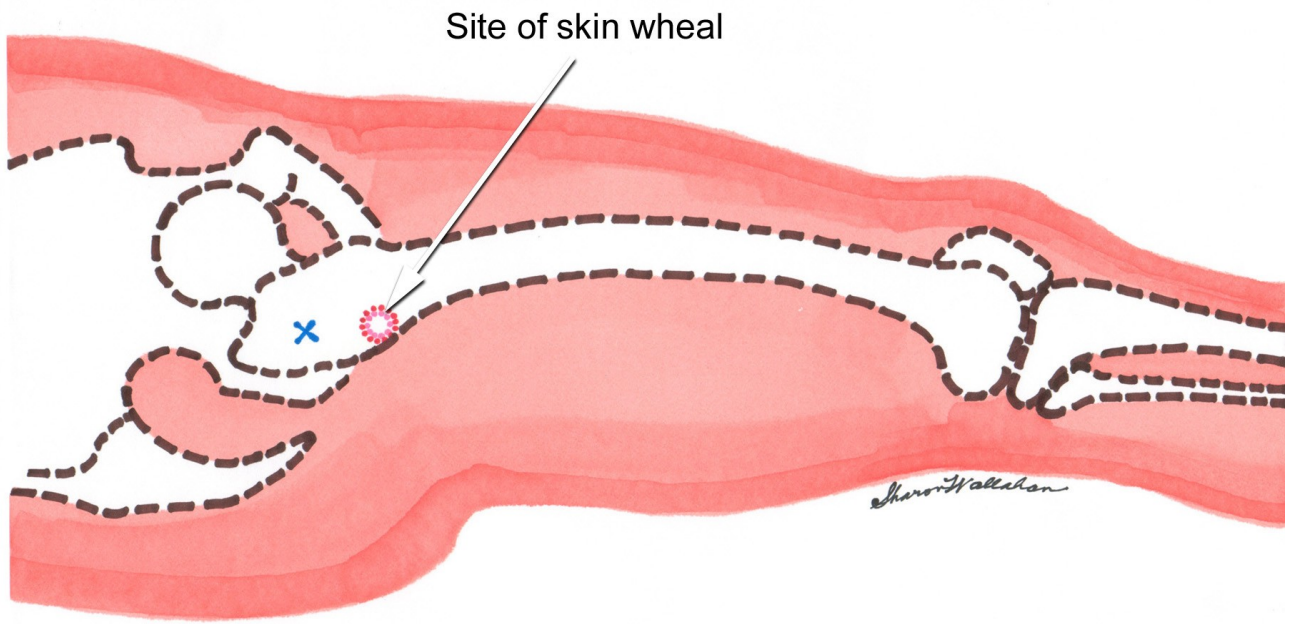


Lateral femoral cutaneous nerve block technique. See text for details.

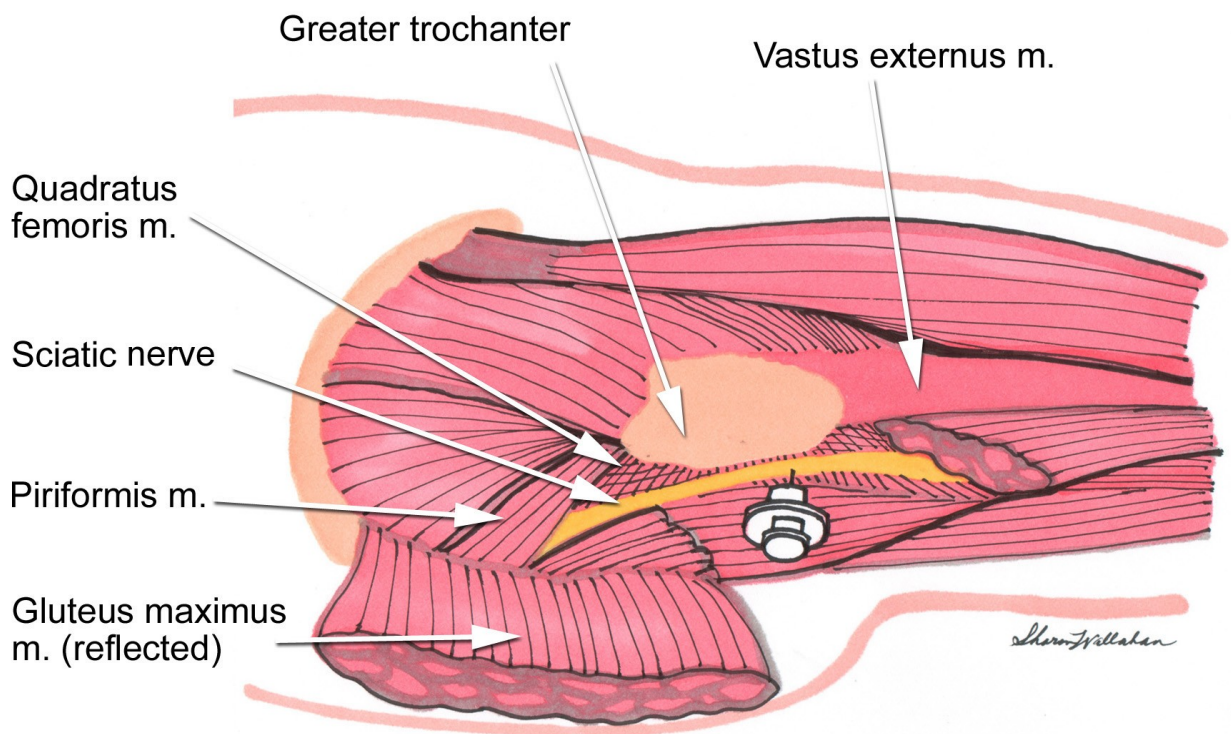
Usually a volume of 5-8 mL of LA is required; addition of corticosteroids may produce therapeutic relief for meralgia paresthetica. Oral medications (tricyclic antidepressants or anticonvulsants) can be added for improved pain relief.

Sciatic nerve block

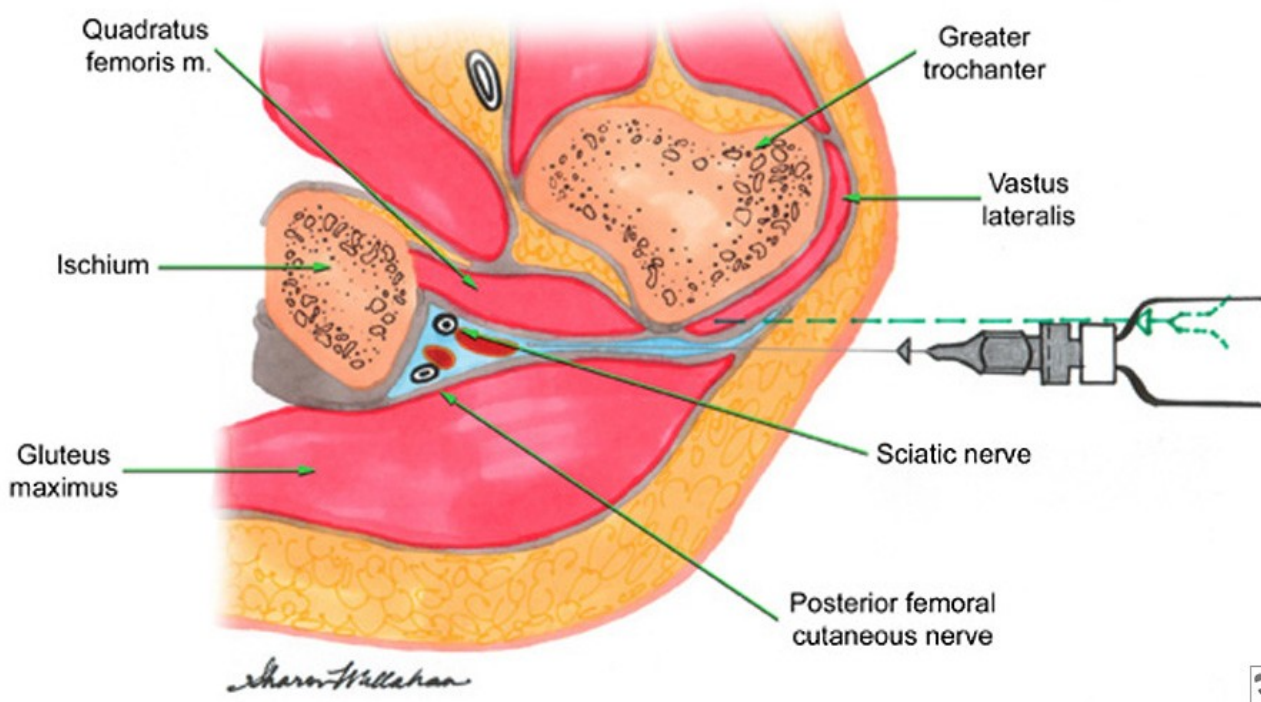
Sciatic nerve block is effective for the control of severe, acute posttraumatic or postoperative pain, or to provide sympathetic interruption of the foot, leg, and posterior thigh. By combining sciatic, femoral, and obturator nerve blocks, analgesia and sympathetic blockade of the entire lower limb can be achieved. The patient is placed in supine position for the following technique described by Bonica.



Sciatic nerve block technique (lateral approach). See text for details.



Sciatic nerve block technique (lateral approach). See text for details.



Sciatic nerve block technique, lateral approach (cross-sectional view).

A 5-cm, 25-gauge needle attached to a 10-mL Luer-Lok syringe containing anesthetic solution is introduced through a skin wheal and advanced slowly through the skin and vastus lateralis muscle until it reaches the posterior surface of the greater trochanter.

LA solution is injected as the needle is introduced and advanced; then an additional 1-2 mL of LA is injected onto the periosteum when the needle contacts the posterior surface of the greater trochanter. This leaves a track of anesthesia for subsequent introduction of the larger needle, a 12- or 15-cm, 22-gauge needle with a 10-mL Luer-Lok syringe containing a greater strength of LA (eg, 0.5% bupivacaine with epinephrine). The depth from skin to periosteum is noted and the needle withdrawn until its tip resides in the subcutaneous tissue.

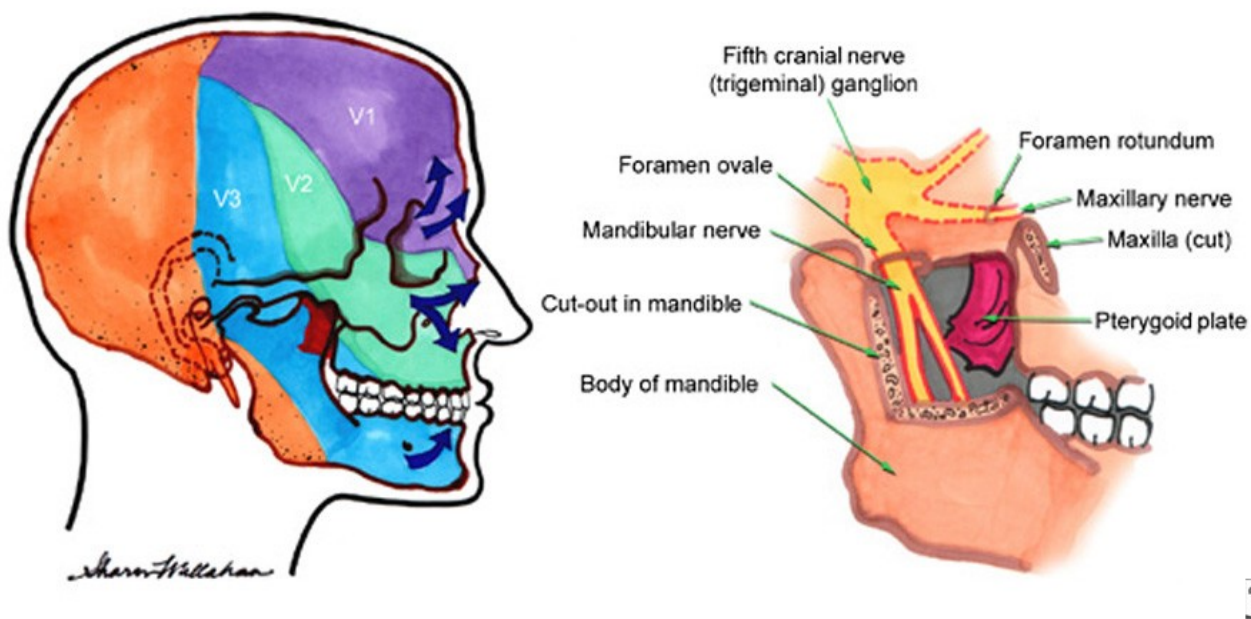
The skin is then moved 1 cm posteriorly and the needle is advanced again parallel to the previous track, the trajectory of which should be parallel to the floor. When the needle has traveled twice the original distance of the first needle, it should be in proximity or contact with the sciatic nerve and elicit paresthesia. The needle should be advanced cautiously and slowly, stopping as soon as the patient experiences paresthesia, to prevent needle penetration and damage to the axons of the sciatic nerve. The LA solution should be injected around the nerve structure to prevent axonal damage.

Neural blockade of median, ulnar, radial, and pudendal nerves also can be used for therapeutic reduction of severe, acute postoperative or posttraumatic pain, and for diagnostic purposes.

Craniofacial neural blockade

Occipital nerve block can be applied for diagnostic, prognostic, and therapeutic purposes in patients with headache, neuralgia, and other painful conditions of the posterior aspect of the head. Using the technique described by Bonica, the greater occipital nerve is blocked by needle placement just above the superior nuchal line and approximately 2.5-3 cm lateral to the external occipital protuberance. If reaching the nerve and eliciting paresthesia are difficult, then 5 mL of LA can be injected on the medial side of the artery, 2 mm superficial to the skull. Frequently, care must be

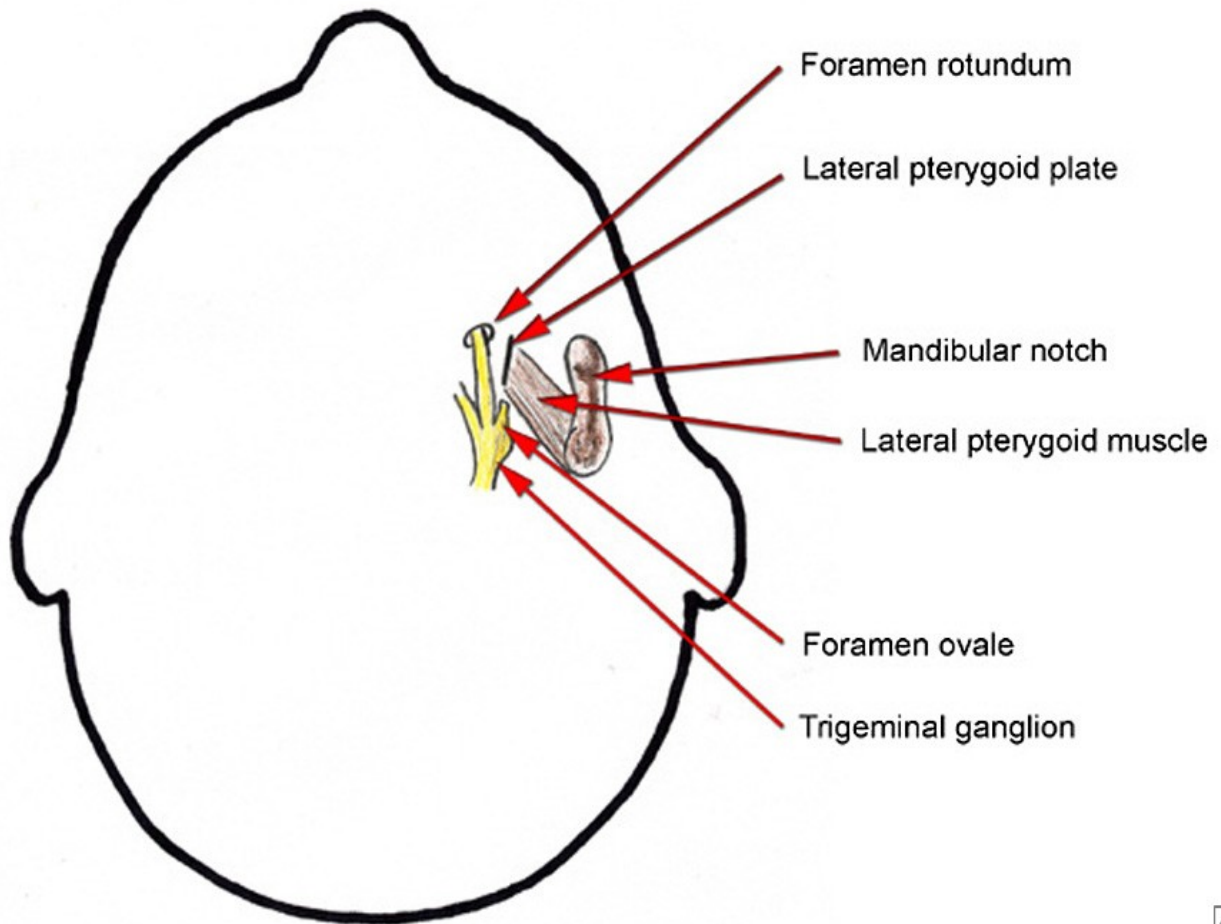
taken during this block not to allow anesthetic fluid to spread laterally, as it may affect the glossopharyngeal nerve, causing hoarseness and difficulty in swallowing.



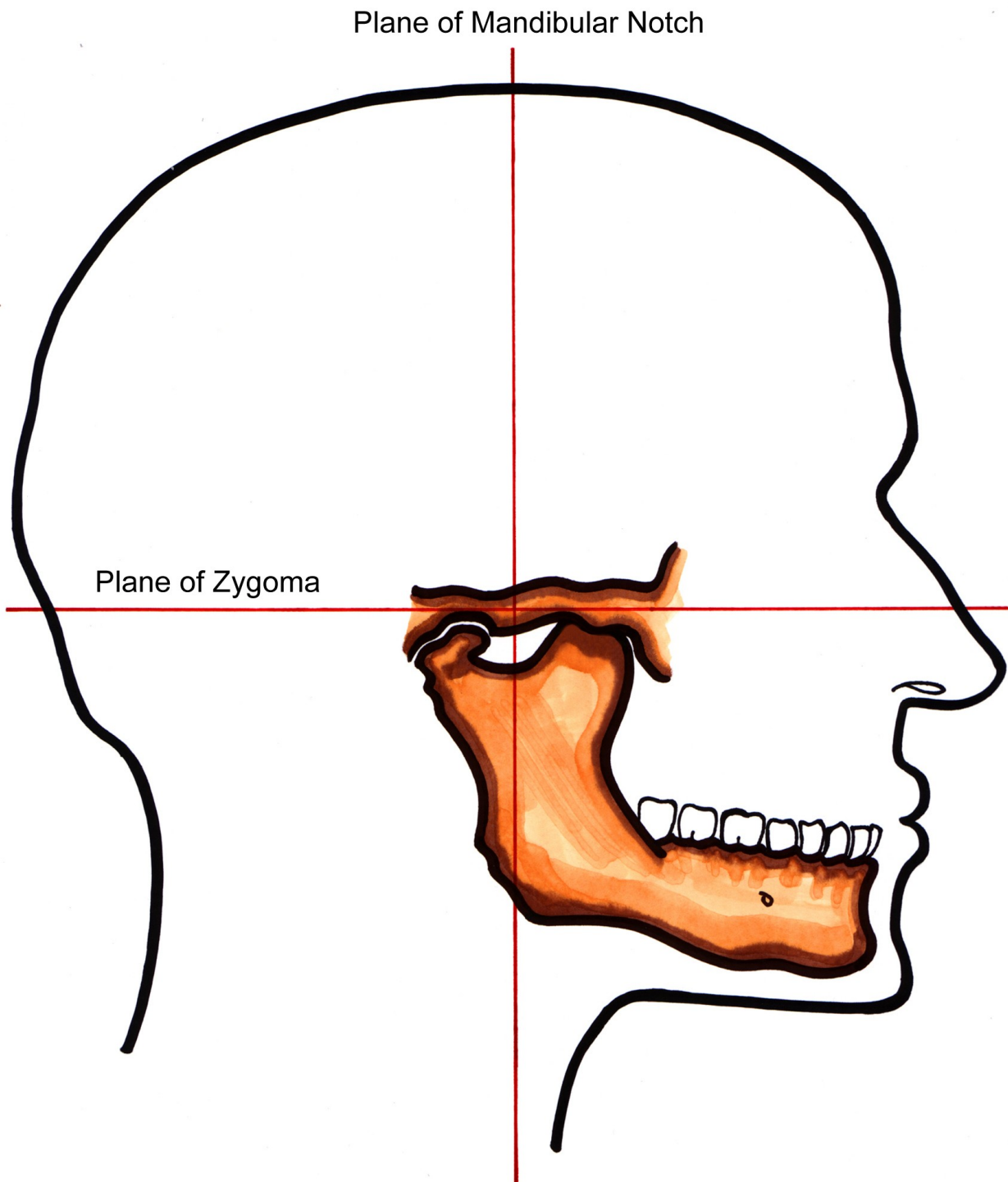
Anatomy of the fifth cranial nerve ganglion (trigeminal) along with innervation and peripterygoid relationship.

Trigeminal ganglion block commonly is used for diagnostic and prognostic purposes when considering trigeminal neurolysis for patients with trigeminal neuralgia. The trigeminal ganglion is located intracranially, situated lateral to the internal carotid artery and cavernous sinus and posterosuperior to the foramen ovale.

The foramen ovale is approximately 1 cm in diameter and serves as the cranial opening through which the mandibular nerve exits; it lies approximately in the same horizontal plane as the zygoma at the level of the mandibular notch, immediately dorsolateral to the pterygoid process.

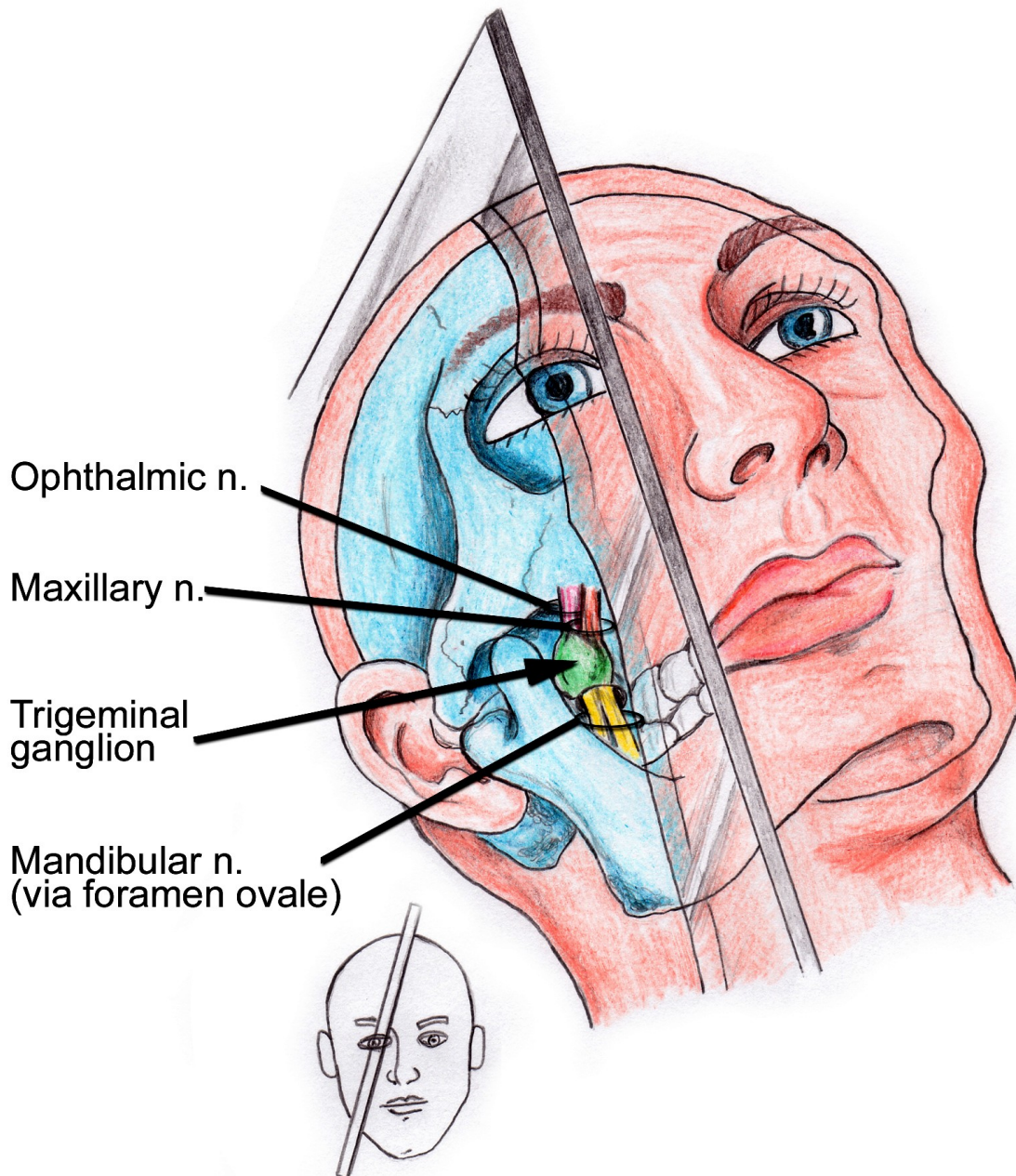


Anatomy of the fifth cranial nerve (trigeminal) ganglion and foramen ovale (cross-sectional view).

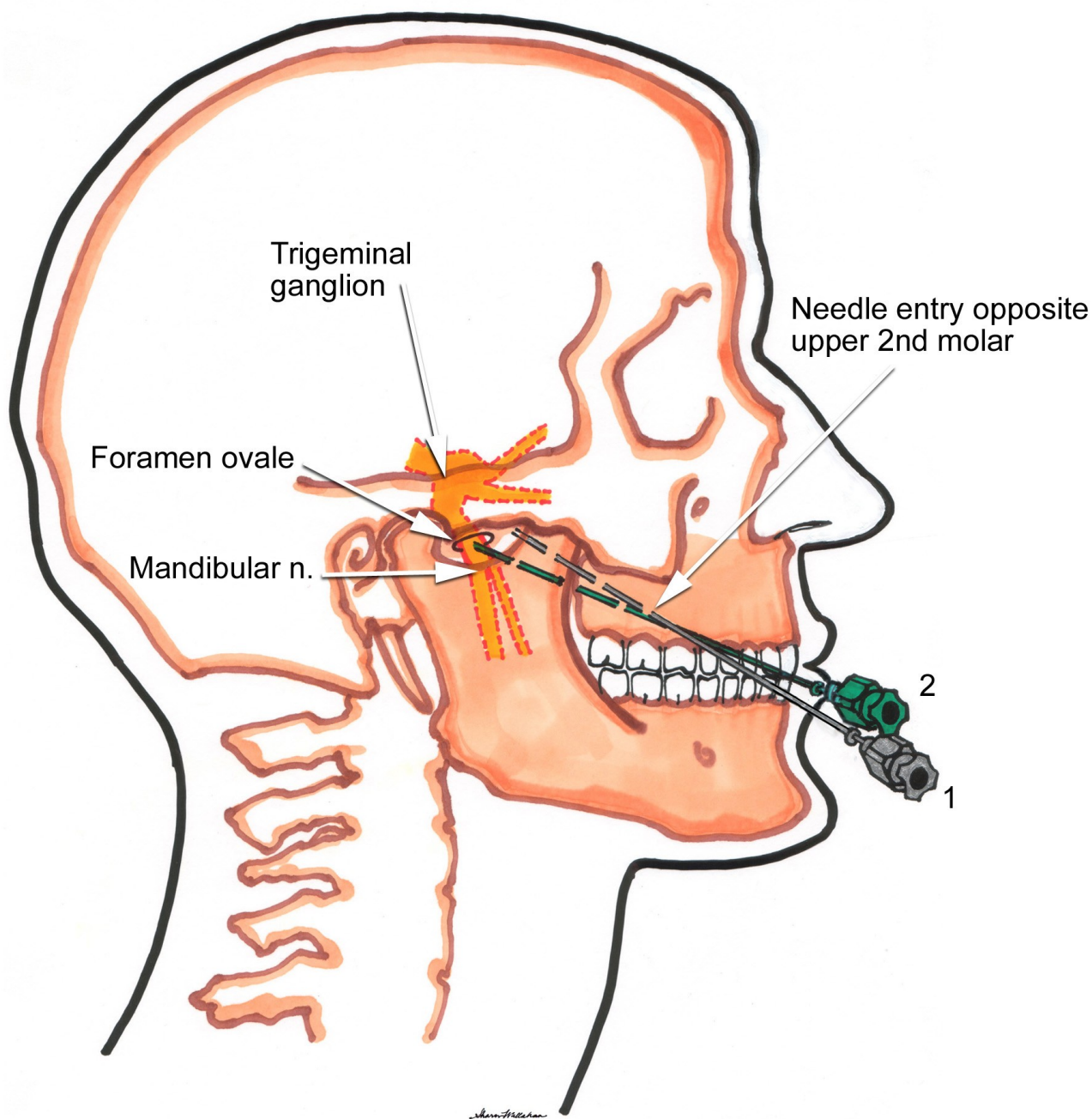


Lateral view of mandibular notch and plane of zygoma. Correlate with cross-sectional view of the fifth cranial nerve (trigeminal) ganglion and foramen ovale.

Trigeminal ganglion blockade should be performed only by skilled and experienced interventionists. Using the technique described by Brown, the patient is placed in a supine position.



Anatomy and needle-insertion plane of trigeminal ganglion block technique. See text for details.



Anatomy and technique of trigeminal ganglion block. See text for details.

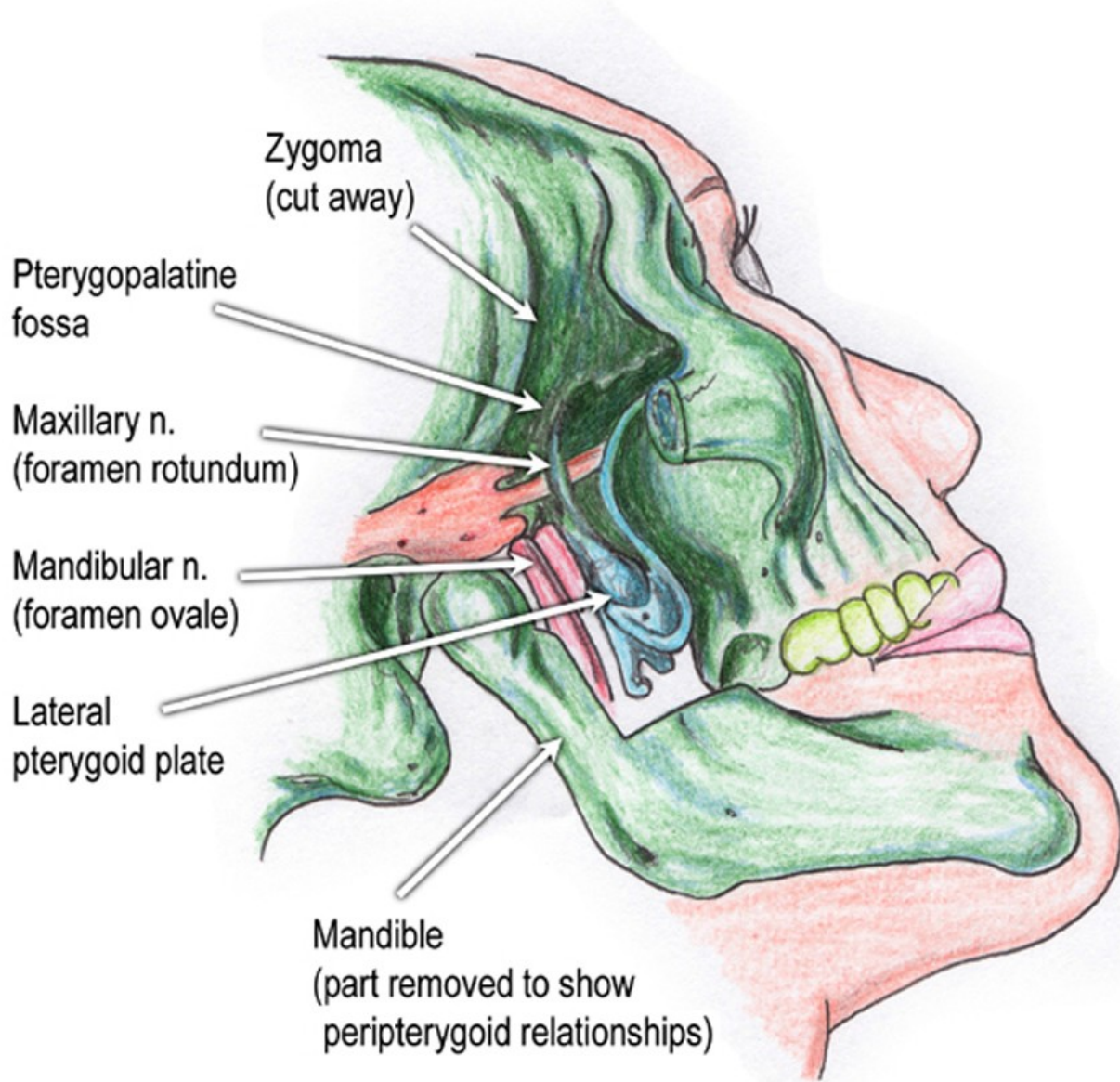
A 22-gauge, 10-cm needle is inserted through a skin wheal approximately 3 cm lateral to the corner of the mouth and medial to the masseter muscle in a direction that bisects the plane formed by the midpoint of the pupil with the patient staring at the ceiling. This allows the needle tip to contact the infratemporal surface of the greater wing of the sphenoid bone, immediately anterior to the foramen ovale at a depth of 4.5-6 cm.

Once the needle is positioned firmly against this bony target, it is withdrawn and redirected in a stepwise manner until it enters the foramen ovale at a depth of about 6-7 cm, approximately 1.5 cm beyond the initial needle length required to contact the bone. As the foramen is entered, paresthesia in the mandibular distribution usually is evoked.

Further slight and careful movement of the needle may elicit paresthesia in the distributions of the ophthalmic and maxillary nerves. These additional paresthesiae verify a periganglionic placement of the needle tip.

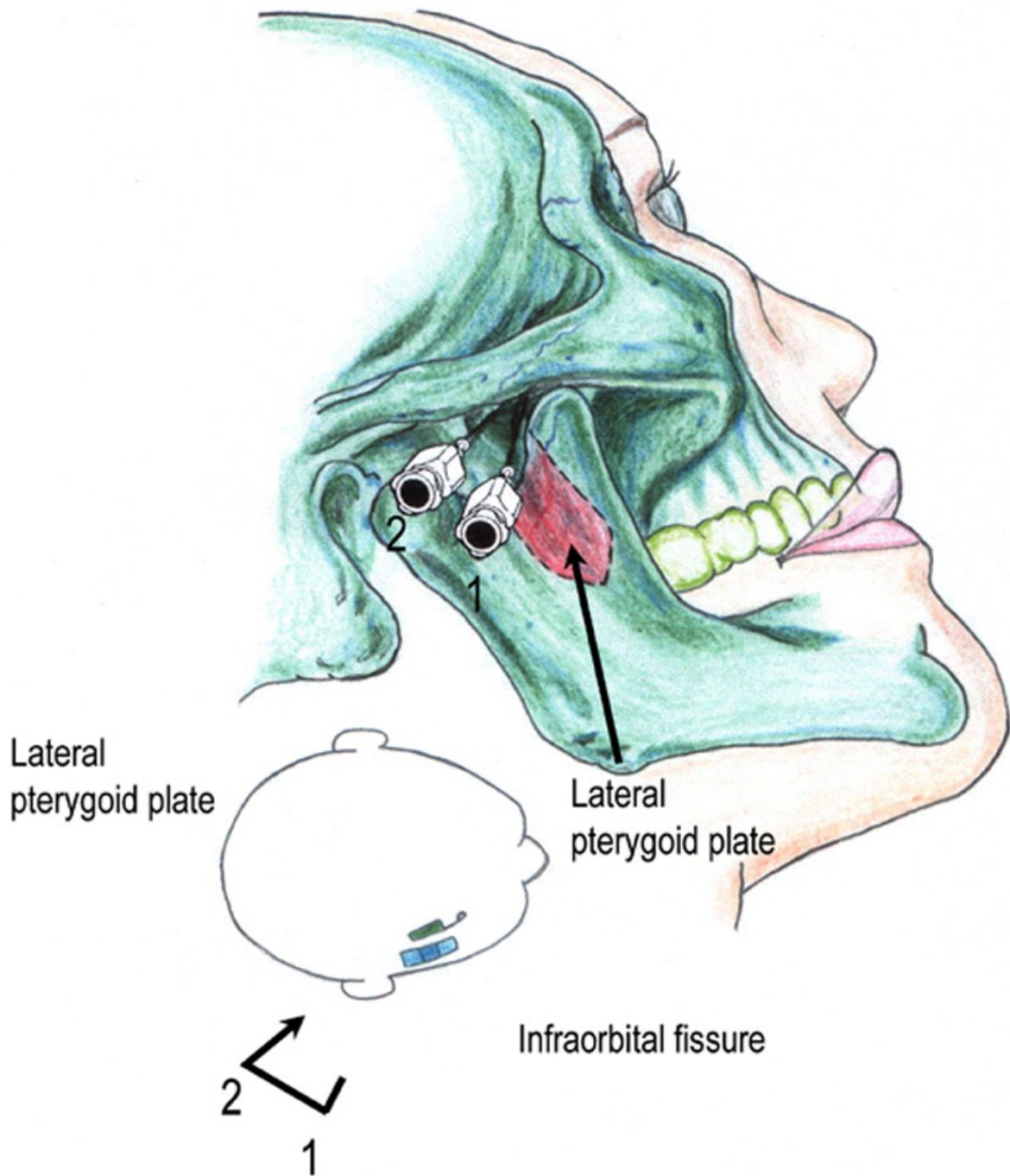
Aspiration should be performed first to check for CSF because the posterior two thirds of the trigeminal ganglion is enveloped in the reflection of the dura. One milliliter of a short-acting LA then can be injected. If neural blockade is incomplete after 5-10 minutes, an additional 1-2 mL of LA can be injected or the needle can be repositioned to obtain a more complete block. The most concerning complication with this procedure is subarachnoid injection. Moreover, because the needle passes through a highly vascular region, hematoma formation is a possibility.

Maxillary nerve blockade also can be useful for diagnosis and treatment of facial neuralgia. The maxillary nerve is entirely sensory and exits through the foramen rotundum. Using the technique described by Brown, the patient is placed in supine position with the head and neck rotated away from the side to be blocked.



Pertinent anatomy with regard to the maxillary block.





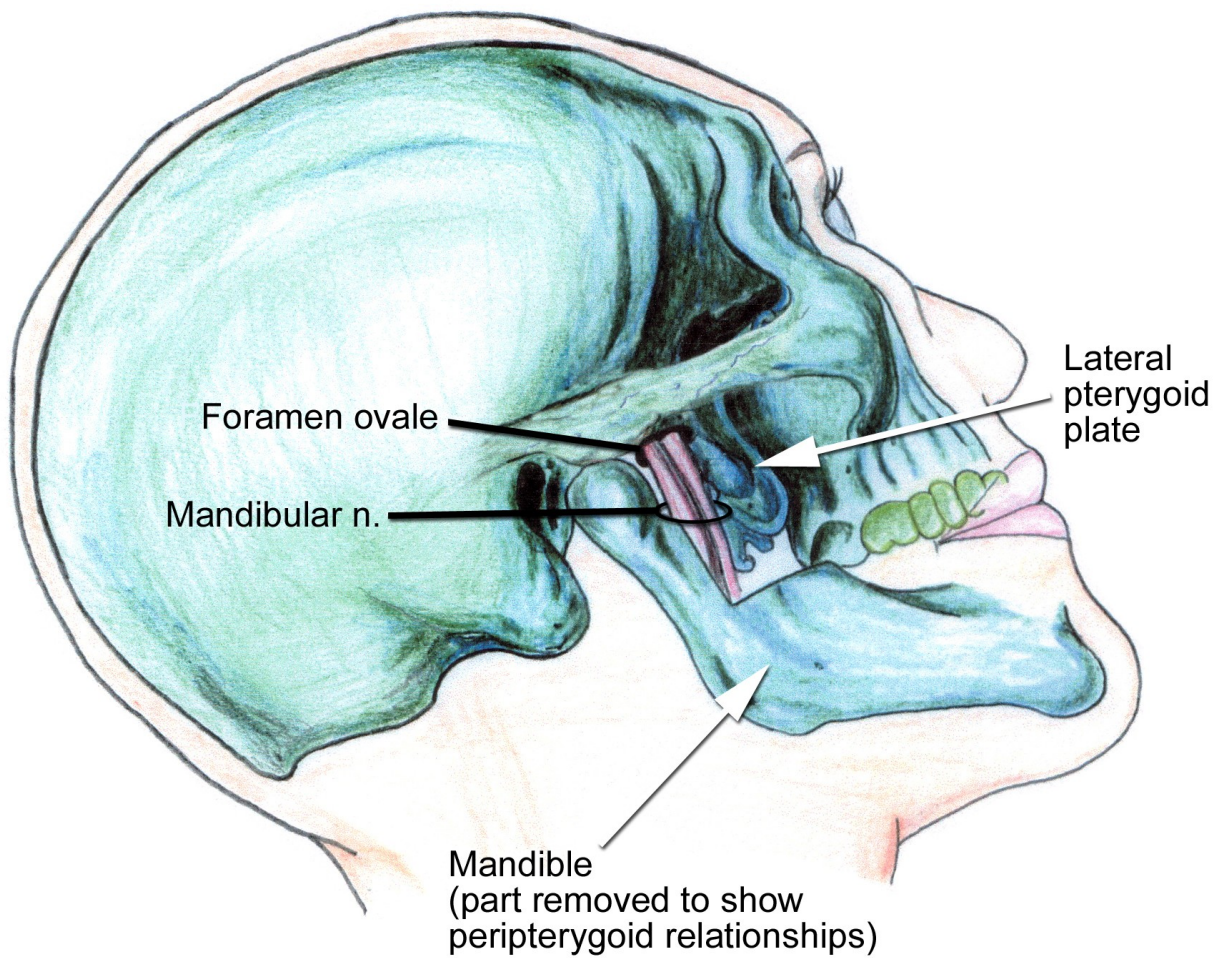
Anatomy of maxillary block. See text for details.

A 22-gauge, 8-cm needle is inserted through the mandibular notch and advanced in a medial and cephalad direction until it meets the lateral pterygoid plate at a depth of approximately 5 cm.

The needle is then withdrawn and redirected in a stepwise manner by walking the bevel off the pterygoid plate, to a depth 1 cm beyond initial contact, until it lies within the pterygopalatine fossa. Once the needle rests in a satisfactory position, 5 mL of LA is injected. Because of the maxillary nerve's proximity to the infraorbital fissure, LA may spill into the orbit and affect eye movement or vision. Because the vascularity of this region is rich, hematoma formation is a possible complication; some subjects may develop a black eye following this block, again because of the close proximity of the orbit.

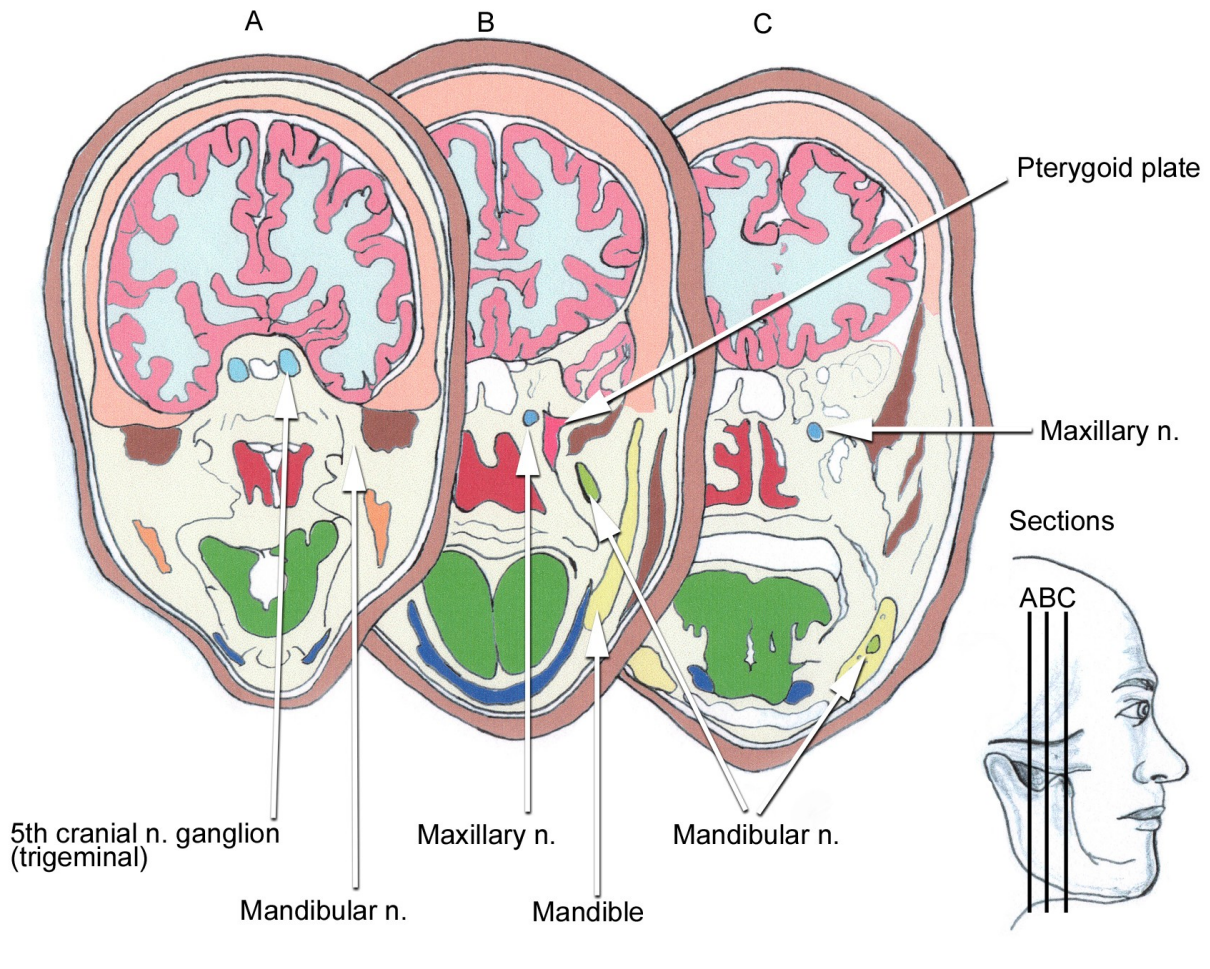


Mandibular nerve block is similarly useful for diagnosis and treatment of facial neuralgia. The mandibular nerve is primarily a sensory nerve and exits the cranium through the foramen ovale, traveling parallel to the posterior margin of the lateral pterygoid plate, then descending inferiorly and laterally toward the mandible.



Pertinent anatomy with regard to the mandibular block.

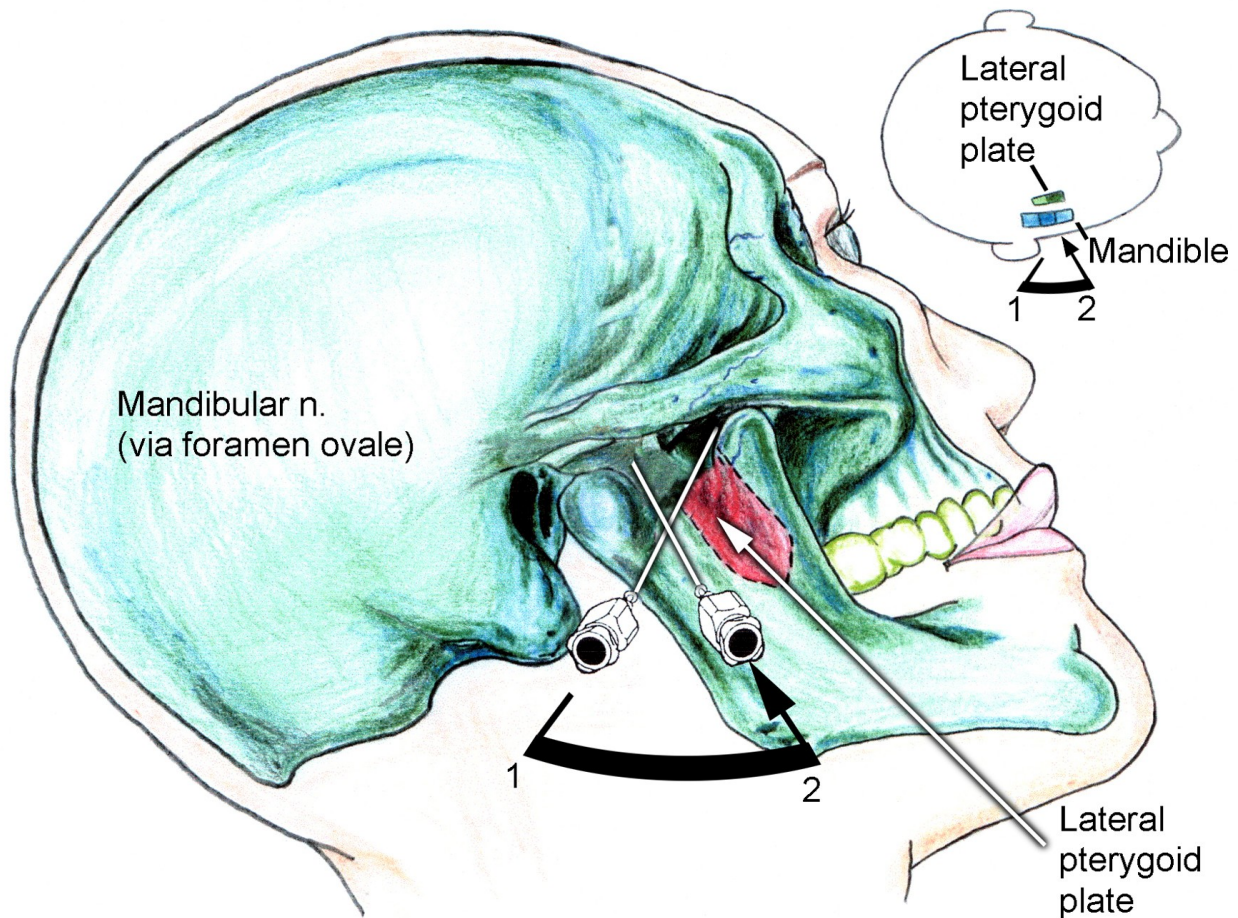




Pertinent coronal anatomy with regard to trigeminal nerve block.

The anterior division of the mandibular nerve is principally motor and supplies the muscles of mastication, whereas the posterior division is principally sensory and supplies the skin and mucous membranes overlying the jaw and skin anteriorly and superior to the ear.

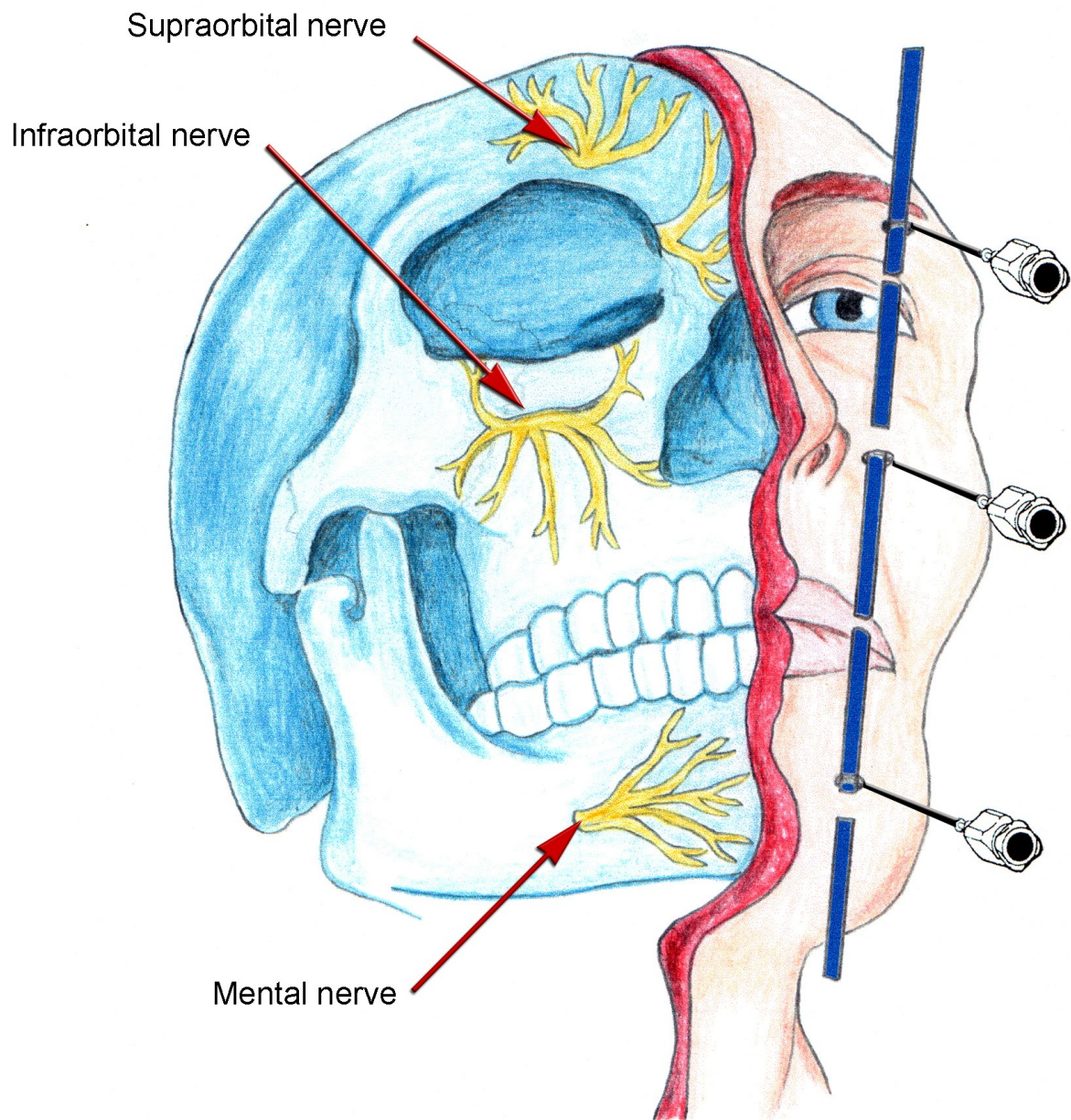
The Brown technique for performing this block begins with the patient in supine position with the head and neck turned away from the side to be blocked.



Anatomy of mandibular block and needle insertion technique. See text for details.

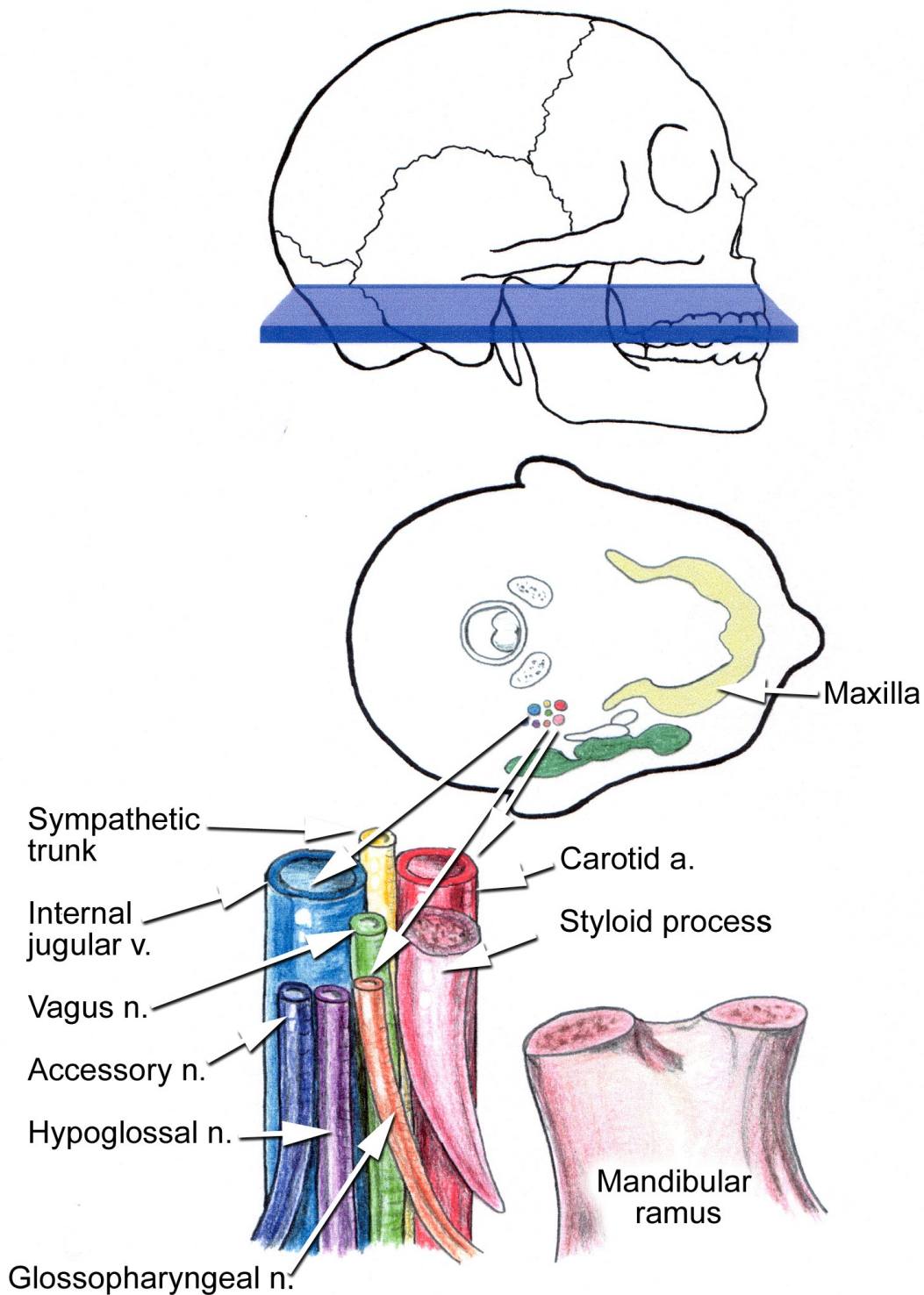
The patient is asked to open and close the mouth gently so that the operator can identify and palpate the mandibular notch. A 22-gauge, 8-cm needle is inserted in the midpoint of the mandibular notch and directed at a slightly cephalad and medial angle through the notch to the lateral pterygoid plate at a depth of approximately 5 cm. The needle is then withdrawn to a subcutaneous position and carefully walked off the posterior border of the pterygoid plate in a horizontal plane. The needle should not be advanced more than 0.5 cm past the depth of the pterygoid plate because the superior constrictor muscle of the pharynx can be pierced easily. When the needle is in appropriate position, 5 mL of LA can be administered. Complications include hematoma formation and subarachnoid injection.

Distal trigeminal blocks can be performed to target specific distal branches of the 3 divisions of the trigeminal nerve, specifically the supraorbital branch of the ophthalmic nerve, infraorbital branch of the maxillary nerve, and mental branch of the mandibular nerve. These blocks are performed with a 25-gauge needle directed at the superficial foraminal site, where approximately 2-3 mL of LA can then be injected.



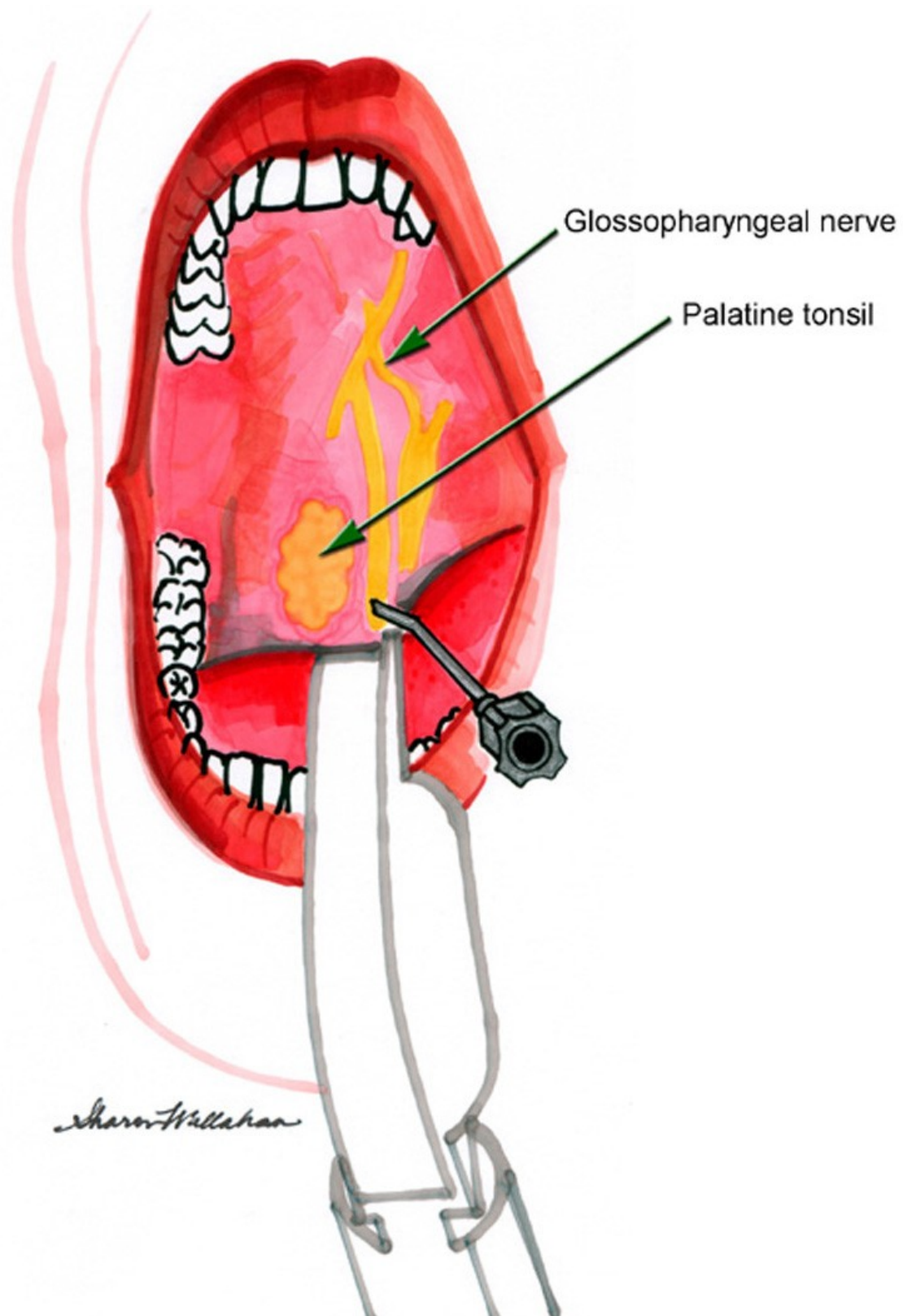
Distal trigeminal block technique. See text for details.

Glossopharyngeal nerve block also is performed for diagnosis and management of neuralgia. The glossopharyngeal nerve exits the jugular foramen at the base of the skull in close association with structures of the cheek, including the parotid gland and vagus nerve. It then descends into the neck between the internal and external carotid arteries.



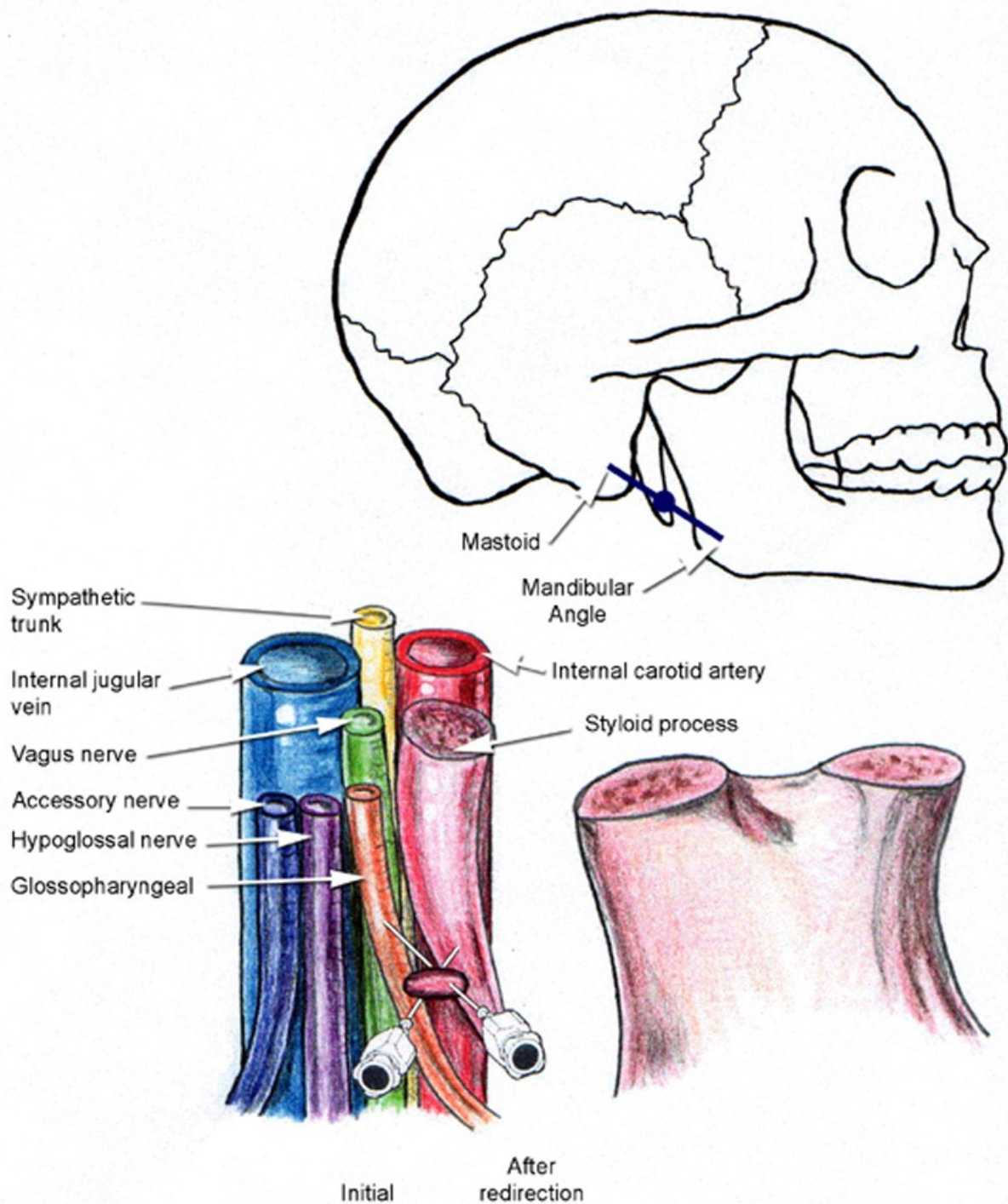
Peristyloid anatomy (cross-sectional view).

A glossopharyngeal block can be carried out intra-orally or using a peristyloid technique. If the block is performed intra-orally, the patient must be capable of opening the mouth, and adequate topical anesthesia of the tongue is necessary to allow needle placement at the base of the tonsillar pillar. While using this approach, care must be taken because of the proximity of the glossopharyngeal nerve to the internal carotid artery, which lies immediately lateral to the tip of the correctly positioned needle.



Intraoral anatomy and glossopharyngeal block technique. See text for details.

The peristyloid approach, also described by Brown, begins with the patient in a supine position with the head neutral.



Glossopharyngeal block, peristyloid technique.

A 22-gauge needle is inserted at the midpoint of a line between the mastoid process and angle of the mandible and advanced until it reaches the styloid process. Palpation of the styloid process should be maintained while the needle is inserted until it reaches this structure. The needle is then pulled back and redirected to slip off the posterior border of the styloid process. Careful aspiration for blood is necessary because of the intimate relationship of both the internal jugular vein and carotid artery to the glossopharyngeal nerve.

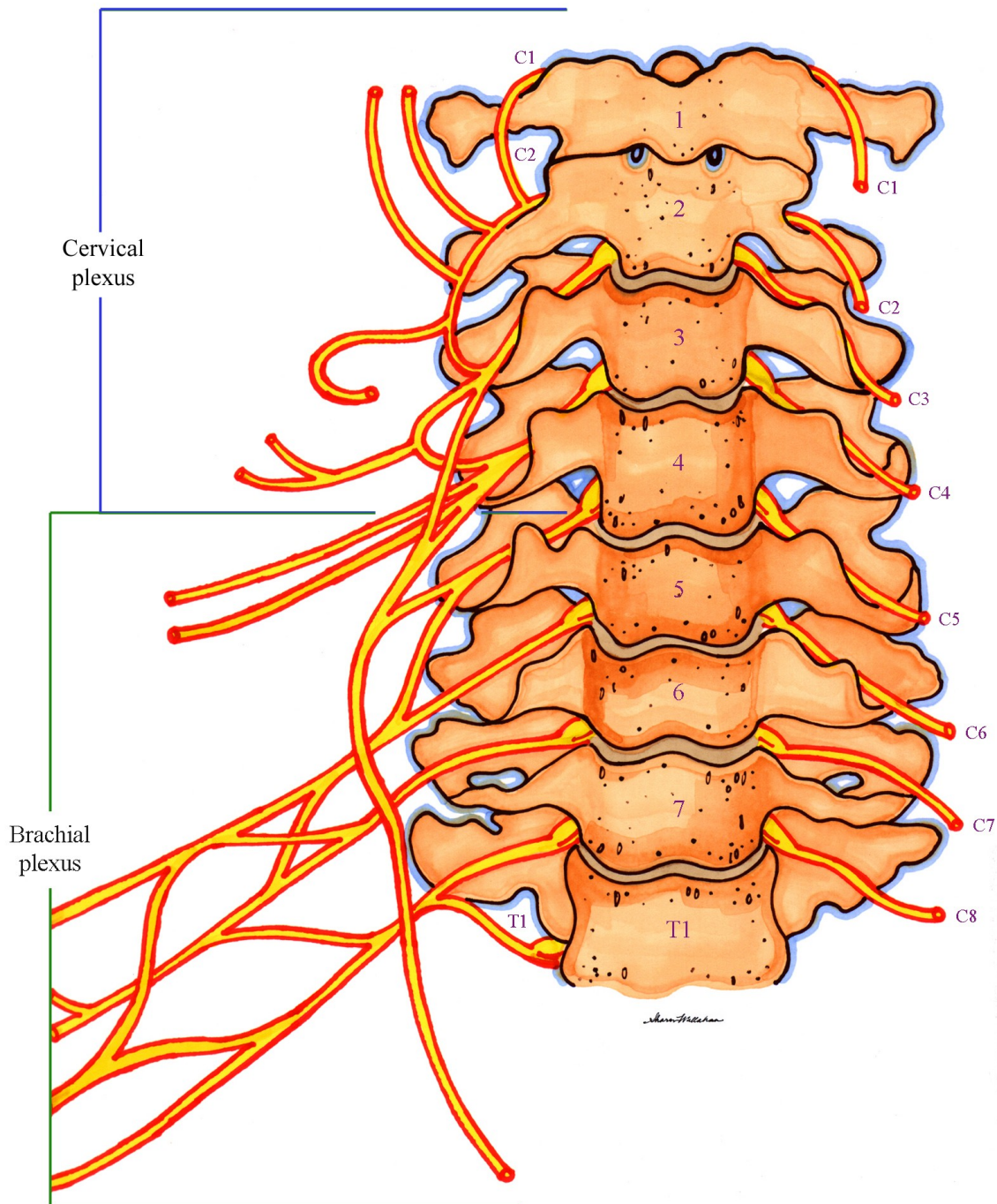
Other blocks, including cervical plexus, superior laryngeal, translaryngeal, and retrobulbar blocks, usually are performed best by anesthesiologists or surgical subspecialists. These blocks are usually performed to achieve regional

anesthesia, although a retrobulbar block can be useful diagnostically for determining the etiology of eye pain.
Technical Principles of Spinal Neural Blockade

Nerves emanating from the spinal cord can be blocked in the paravertebral region or at certain points along their course. These procedures usually are performed for management of severe acute or chronic pain affecting the trunk or extremities, to relieve painful muscle spasm, or to block sympathetic nervous system dysfunction in affected limbs.

Cervical spinal nerves can be blocked to alleviate pain caused by segmental neuralgia or by primary spinal lesions (eg, nerve root compression caused by disk protrusion, spondylosis, or neoplasm). In some cases, these blocks provide prognostic information and if successful can be followed by neurolytic or neuroablative interruption of involved pathways, particularly when the pain is caused by cancer. Also, selective nerve root blocks often are used to determine whether a patient will respond to surgical decompression.

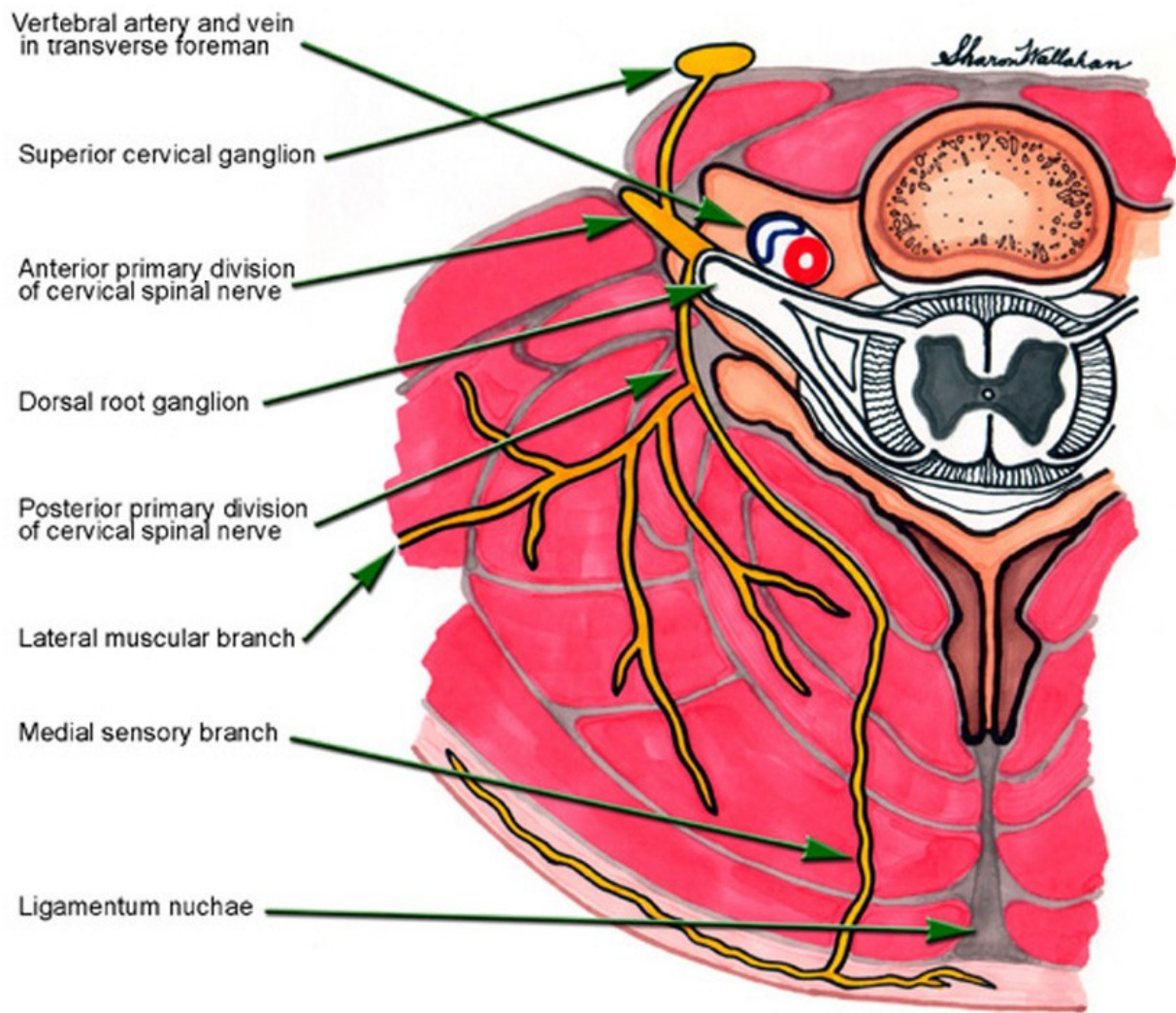
Cervical nerve roots (C1-C8) pass laterally through their respective foramina within the sulcus of each transverse process and exit at the level above the vertebral segment for which they are numbered.



Anatomy of the cervical nerves (anterior view).

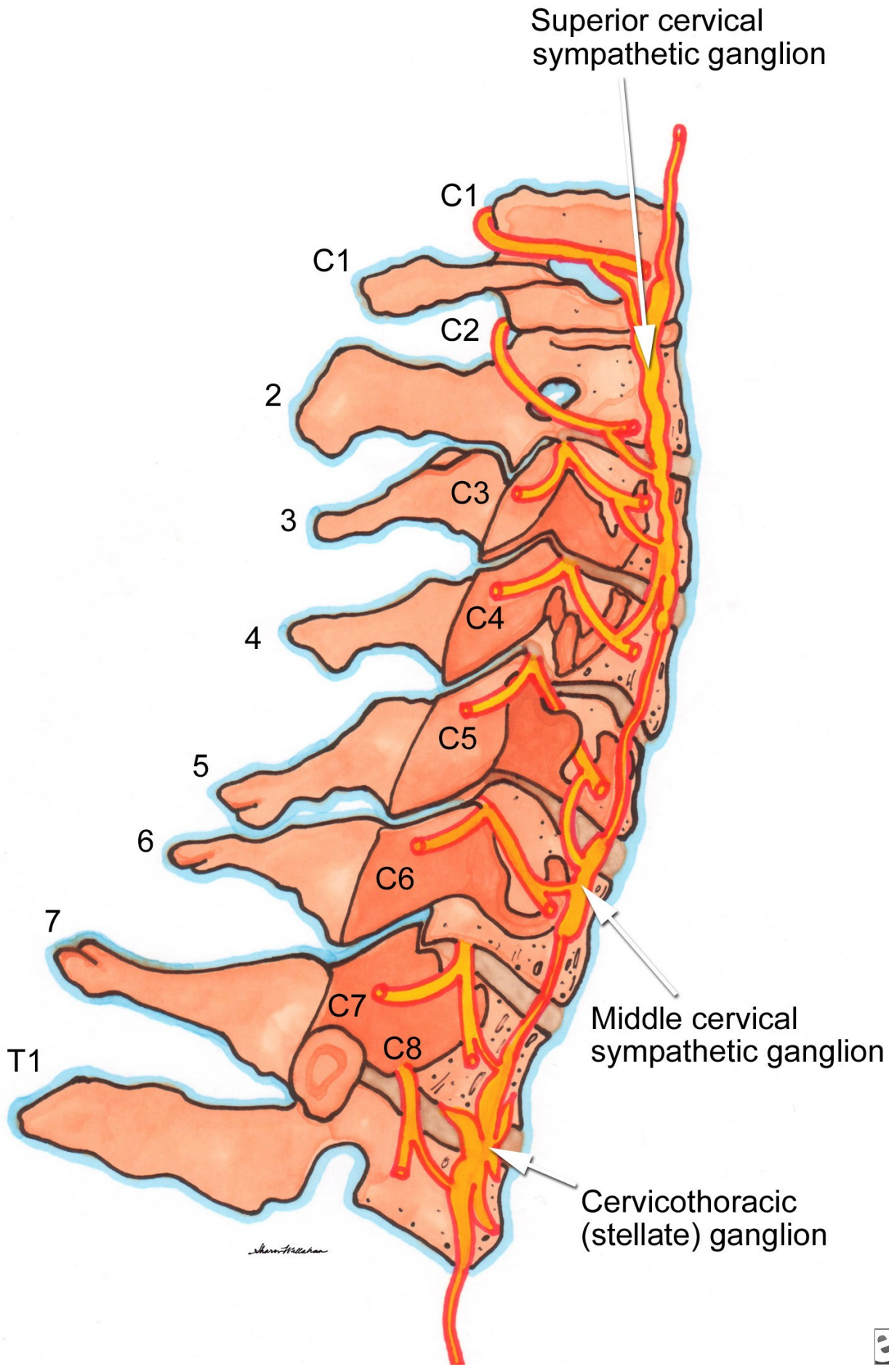
The posterior tubercle of the tip of each transverse process is larger and more superficial, and therefore is easier to palpate than the nearby anterior tubercle. Ventral and dorsal divisions of each cervical nerve root join to form the dorsal root ganglion, which lies just posterior to the ascending vertebral artery.

Just lateral to the dorsal root ganglion, the posterior primary division or dorsal ramus passes posteriorly, dividing into a lateral muscular branch and a medial sensory branch.



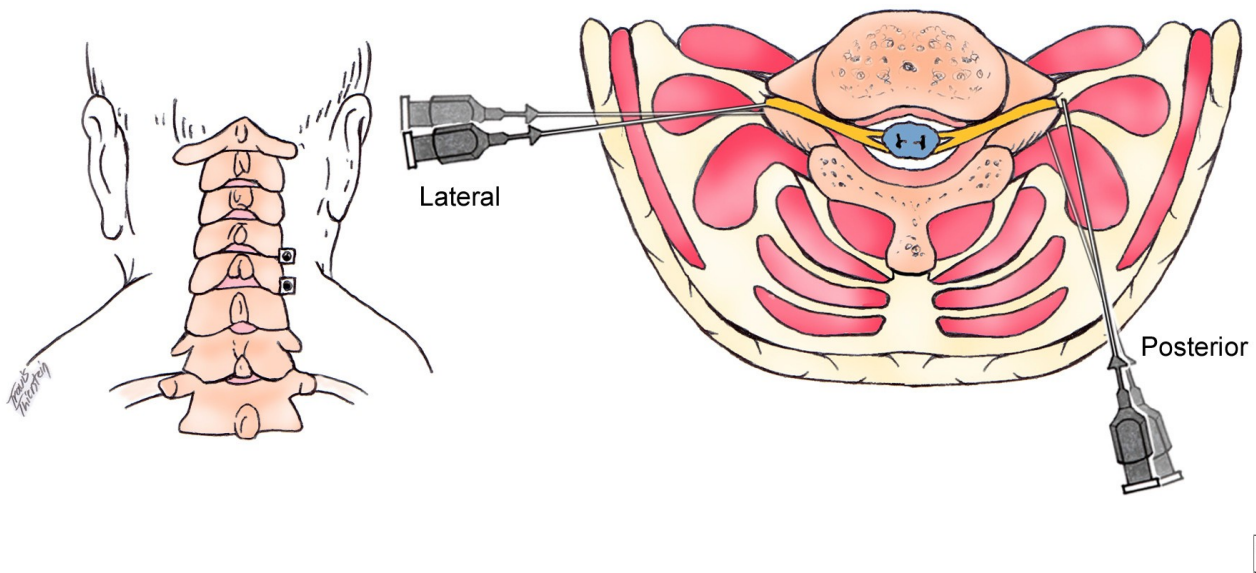
Anatomy of the cervical nerves in the neck (transverse section).

The anterior primary division or ventral ramus continues its anterolateral course, sending gray ramus communicantes to the nearby sympathetic ganglion situated adjacent to the anterolateral surface of the vertebral body.



Anatomy of the cervical nerves (lateral view).

Each cervical nerve root can be blocked paravertebrally by approaching the nerve in a lateral or posterior direction as it lies within the shallow sulcus of the transverse process. The posterior approach is technically more difficult but may be necessary in patients with contraindications due to skin infection, carcinoma, or other pathological processes in the lateral structures of the neck. Furthermore, neural blockade of the C8 nerve root can be achieved only from a posterior approach by slowly passing the needle caudally and slightly medially over the transverse process of C7, until the patient reports paresthesia in the C8 distribution.



Paravertebral block technique (posterior approach). See text for details.

Because selective nerve root blocks often are performed for diagnostic purposes, only small quantities of LA should be used to prevent any confusion that may occur if anesthesia spreads to adjacent nerve roots simultaneously. Nerve roots are identified by eliciting dermatomal paresthesia; radiographic confirmation is useful to ascertain the level and to prevent needle injury to the target nerve root(s).

Complications of paravertebral block include accidental injection into the subarachnoid, subdural, or epidural space. This is particularly hazardous in the cervical region, where anesthetic may diffuse and cause phrenic nerve and respiratory paralysis. Accidental injection of LA into the vertebral artery may lead to transient loss of function in vital brainstem areas and cause impaired consciousness, coma, and seizures. This complication requires immediate cardiorespiratory and circulatory support until the LA is redistributed and metabolized. Other possible adverse events include anesthesia or injury to the cervical sympathetic chain (with development of Horner syndrome), the superior or recurrent laryngeal nerve, or the trunk of the vagus nerve. Because of the risks of unintended recurrent laryngeal and phrenic nerve block, limiting the procedure to a unilateral injection at any one treatment setting is advisable.

Intra-articular cervical zygapophyseal joint blocks should be performed by a highly skilled interventionist experienced with the use of fluoroscopy for needle placement. The C3-4 to C6-7 levels can be accessed using either a posterior or lateral approach. Only the lateral approach is feasible for the C7-T1 joint because of the steep slope. An oblique approach to the C2-3 zygapophyseal level is necessary because the joint tends to slope caudally and medially.

Injection techniques described by Bogduk et al usually are accomplished with the patient in a prone position, but if necessary, the patient can be in the sitting position. A 22- or 25-gauge needle is directed into the midpoint of the target joint from a posterior paraspinal site usually 2 or more segments caudally and along an oblique trajectory that coincides with the plane of the joint as viewed by C-arm fluoroscopy. The needle is directed through the skin upward and ventrally through the posterior neck muscles, until it touches the posterior aspect of the targeted facet; the needle then can be readjusted until it enters the joint cavity. Repeated posteroanterior and lateral screening by fluoroscopy allows the operator assurance that the needle maintains its intended course. The bevel of the needle should enter the target joint at its midpoint.

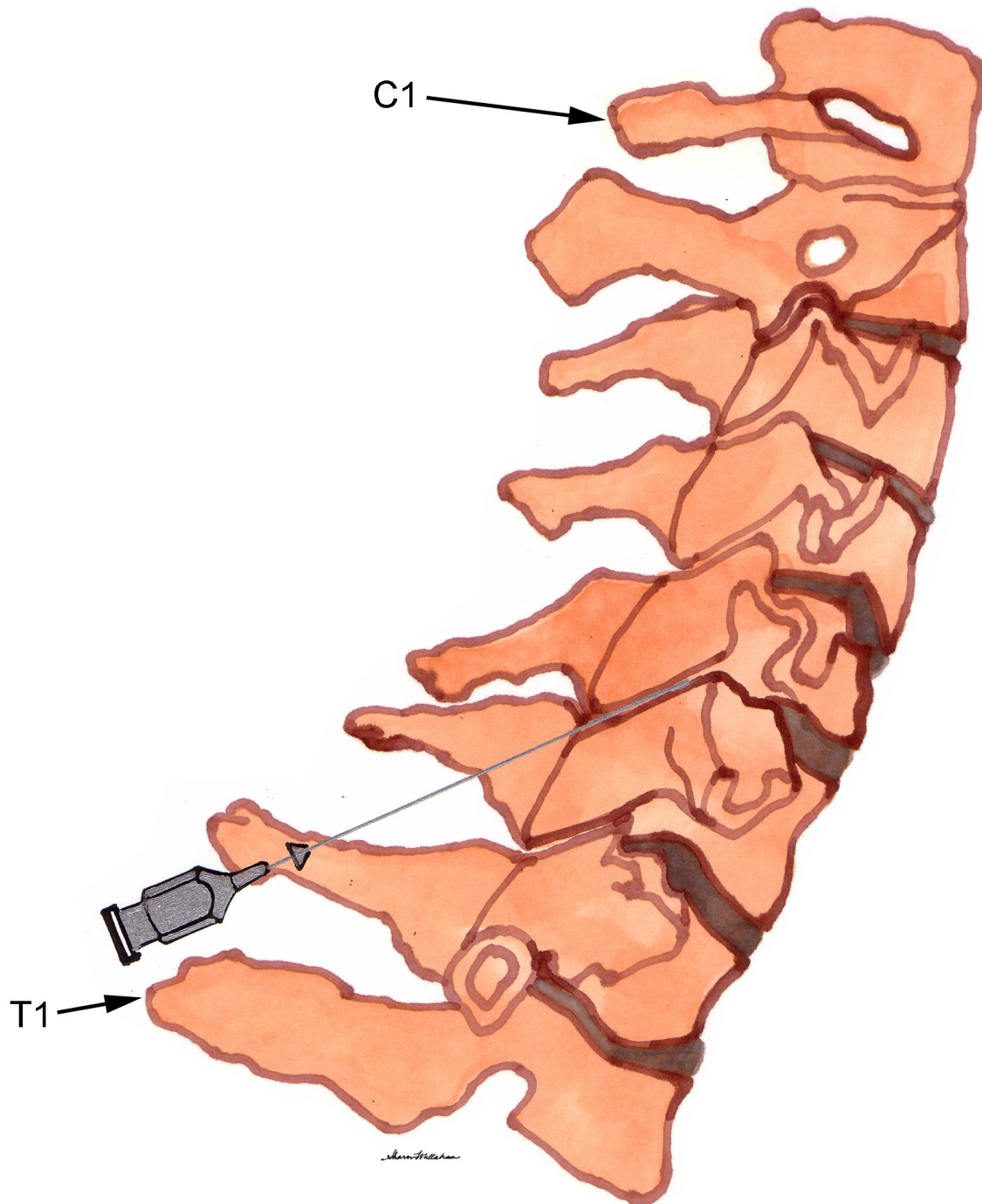
Once the needle is located in the joint, injection of contrast medium should produce an arthrogram and confirm accurate needle placement. Contrast injection may induce a pain response from the awake patient; the practitioner should record carefully whether the pain is concordant (identical to the pain under investigation), partly concordant (similar, but not identical), or nonconcordant (a different, new pain experience). After the patient's pain response is recorded, the injection of LA with or without corticosteroids provides further diagnostic information on whether the pain can be ablated satisfactorily.

Although the goals of this procedure are controversial, therapeutic response is a primary goal. The capacity of the joint can be gauged from the injection of the contrast medium; it is usually less than 1 mL. The capacity of zygapophyseal joints should be considered to prevent rupture of the joint capsule and unwanted spread of the injected agent(s) into surrounding tissues, which may exacerbate the patient's pain syndrome and confound the identification of the joint as a pain generator. Other possible complications include inadvertent trauma and puncture of the deep cervical artery en route, the facet-joint capsule, the vertebral artery or vein, the ventral ramus of the spinal nerve, the epidural space, or in some cases the spinal cord.

The lateral approach to the cervical zygapophyseal joints described by Bogduk et al is performed with the patient in the lateral position. In these cases, the patient may be gently rolled ventrally or dorsally, or the x-ray beam adjusted to tilt along the transverse plane of the target joint. The needle is directed, then advanced, toward the superior or inferior articular process at the midpoint of the target joint. This is done carefully so that the needle does not pass deeper into or through the joint into the epidural space.

When the needle reaches the joint, the operator gently probes and pierces the capsule to enter the joint space, which can be sensed by the operator as a loss of resistance. Only minimal penetration is required. A small amount of contrast medium (0.3 mL) is used to obtain an arthrogram and to record the patient's pain response. The C2-3 zygapophyseal joints are blocked using the lateral approach already described. This level is considered important, and especially pertinent to neurologists, because several studies suggest that this articulation level is the most common source of cervicogenic headache.

Cervical medial branch blocks also can produce anesthesia of cervical facets. These medial branches of the cervical dorsal rami travel across the waist of the articular pillars -- a point proximal to the origin of the articular branches. Here, the nerves have a constant relationship to bone and are more easily accessible by either a posterior or lateral approach.



Cervical spine local injection technique into the cavity of the right C5-C6 zygapophyseal joint (lateral view). See text for details.

Atlantoaxial joint block should be performed only by highly skilled interventionists. The intra-articular target of this joint is at the midpoint of its radiographic silhouette as seen in posteroanterior views. This procedure is potentially hazardous with little margin for error because of the proximity of the dural sac, spinal cord, and vertebral artery.

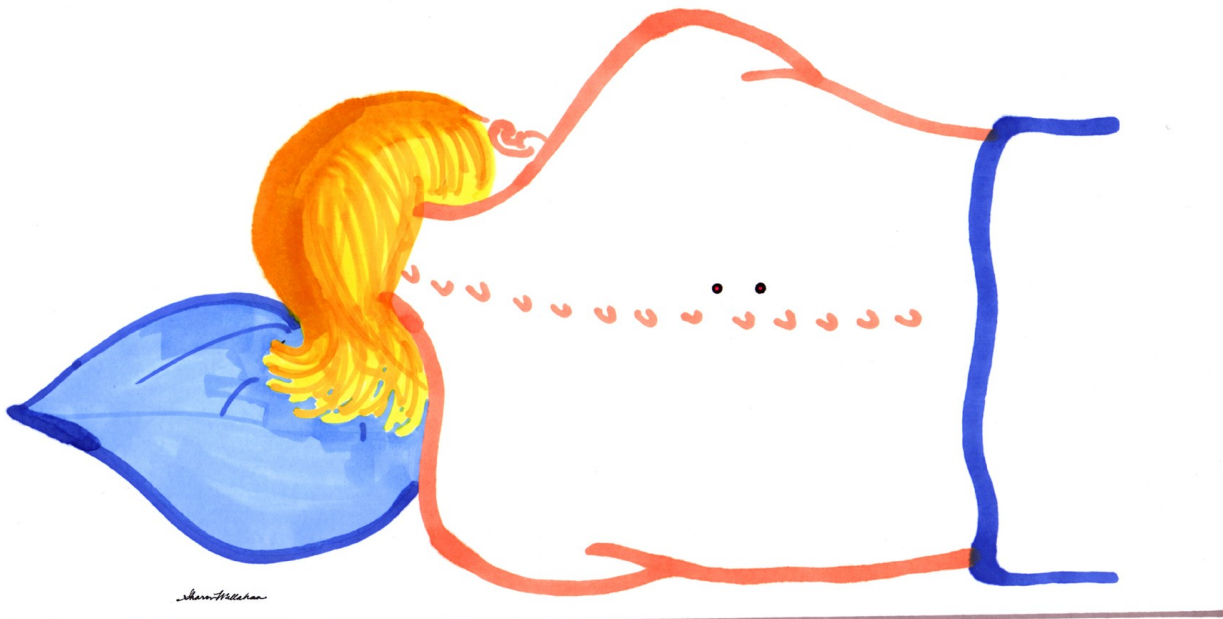
Cervical epidural steroids are administered with similar techniques, which are described in the section on lumbar epidural injections. This procedure has not had widespread appeal because operators are unwilling to risk complications involving the cervical spinal cord. Nevertheless, in well-trained hands, under radiographic control, the

procedure can be accomplished safely.

Thoracic paravertebral somatic blocks can alleviate pain involving the thoracic paraspinal regions, chest, and abdomen. Paravertebral blocks are helpful in determining the cause of nociception when patients complain of thoracic segmental neuralgia caused by vertebral pathology, such as osteoporosis, metastatic fracture, or nerve root impingement. Thoracic paravertebral blocks also can be used to quell the pain of acute and chronic herpes zoster syndromes; severe acute postthoracotomy pain; pain from skeletal muscle spasm; pain from fractures involving the head or neck portion of the rib or surgical or traumatic injuries to the chest wall or upper abdomen; or segmental neuralgia caused by osteoporosis.

The technique of thoracic paravertebral block as described by Bonica entails the insertion of a 4- or 8-cm, 22-gauge, short-beveled needle through a skin wheal approximately 4-5 cm lateral to the spinous process. The needle then is directed anteriorly and medially at an angle of 45° to the midsagittal plane. The needle is advanced until it strikes the appropriate transverse process and then is withdrawn to a subcutaneous position. The needle is next redirected slowly to pass underneath the transverse process until the tip reaches the nerve and appropriate paresthesia is elicited. Procedural risks are evident because of the proximity of the needle tip to the posterior border of the lung; if the needle penetrates the intervertebral foramen, dural and even spinal cord punctures are possible.

To circumvent the risks of this procedure, Bonica developed a paralaminar technique with the patient in the lateral position. A 5- to 8-cm, 22-gauge, short-bevel needle is inserted through a skin wheal of short-acting LA and advanced to the lateral edge of the lamina. After contact with the lateral edge of the lamina, the needle is withdrawn until its point is subcutaneous and the skin is moved laterally, approximately 0.5 cm. The needle is then readvanced until it reaches a point just lateral to the upper edge of the lamina, here engaging the uppermost part of the superior costotransverse ligament just below the adjacent transverse process. A 2-mL glass syringe filled with saline solution is then attached to the needle. As long as the tip of the needle is within the ligament, the operator can perceive some resistance to injection.



Lateral position of patient for thoracic paravertebral somatic nerve block (paralaminar technique).

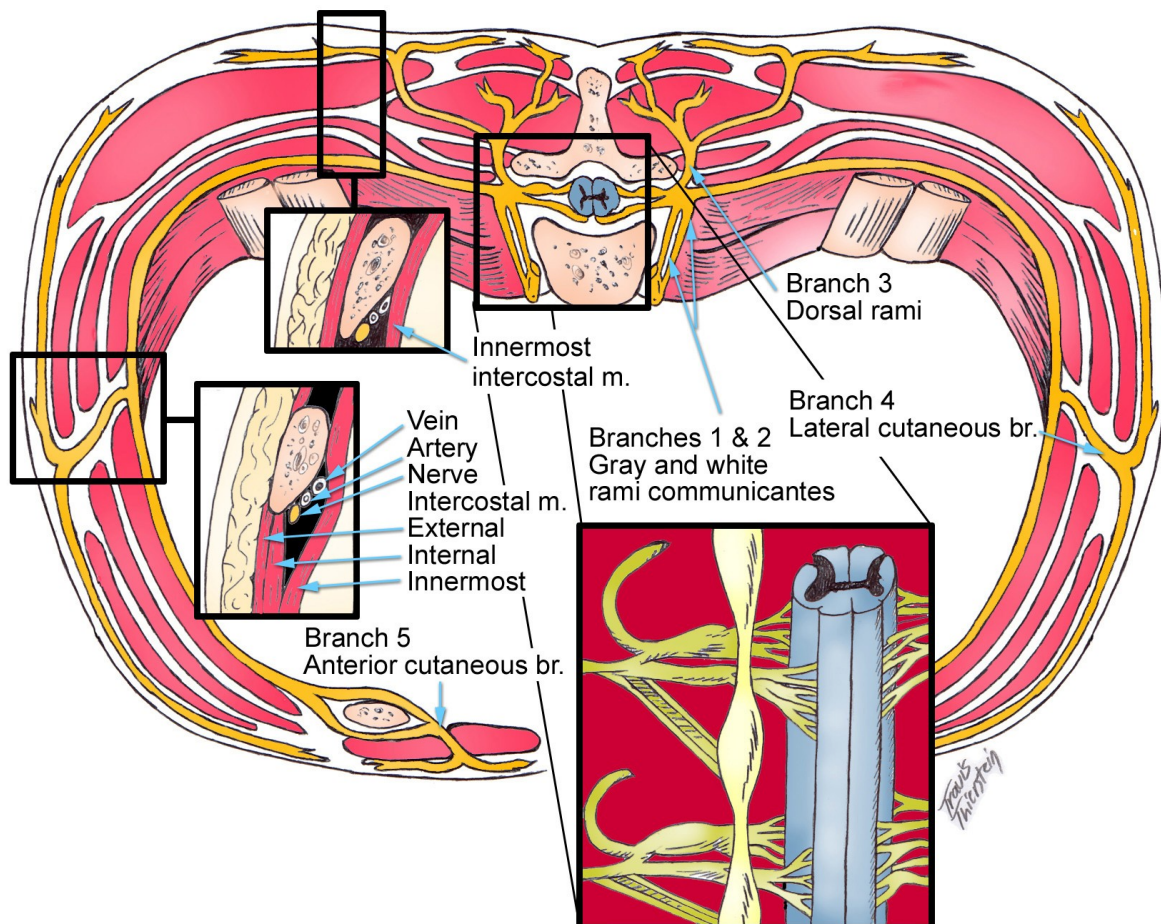
By exerting constant pressure on the plunger of the syringe with the right hand, the needle is advanced slowly with the left hand until lack of resistance is discerned. When this occurs, the needle has passed through the costotransverse ligament into the paravertebral region and the needle tip is likely to be in near proximity to the targeted nerve root. If paresthesia is not elicited, a peripheral nerve stimulator can be used to ensure that the bevel of the needle is positioned adjacent to the nerve. For diagnostic purposes, 3 mL of 1% lidocaine or 0.25% bupivacaine

can be injected. For treatment of acute severe pain, 5 mL of 0.375-0.5% bupivacaine with epinephrine is usually necessary. Production of a prolonged continuous block of multiple levels involves a larger injectate of 10-15 mL of 0.375-0.5% bupivacaine with epinephrine through a fixed catheter. Possible complications include accidental subarachnoid or epidural injection, intravascular injection, and pneumothorax.

Nerve Blocks

Intracostal nerve block is a useful procedure for defining potential source(s) of pain in the chest and abdominal wall. Intracostal neural blockade at the posterior axillary line relieves pain of somatic origin but does not relieve pain arising in the thoracic or abdominal viscera, which are supplied by nociceptive fibers that follow sympathetic pathways located near the vertebral column. Intracostal nerve blocks also can offer relief of severe posttraumatic, postoperative, or postinfectious pain in the thoracic or abdominal wall. Other indications include severe pain involving rib fractures or dislocation of the costochondral joints at the sternum, chest pain associated with pleurisy, pain associated with herpes zoster or intracostal nerve entrapment in the abdominis rectus sheath, and postoperative pain from thoracotomy, sternotomy, and after renal surgery through flank incisions. Caution should be used when performing bilateral intracostal blocks, since ventilation may be impaired.

The intracostal nerves are the ventral rami of T1 through T11; however, the twelfth ventral ramus becomes the subcostal nerve and travels between the transversus abdominus and internal oblique muscles of the abdomen.



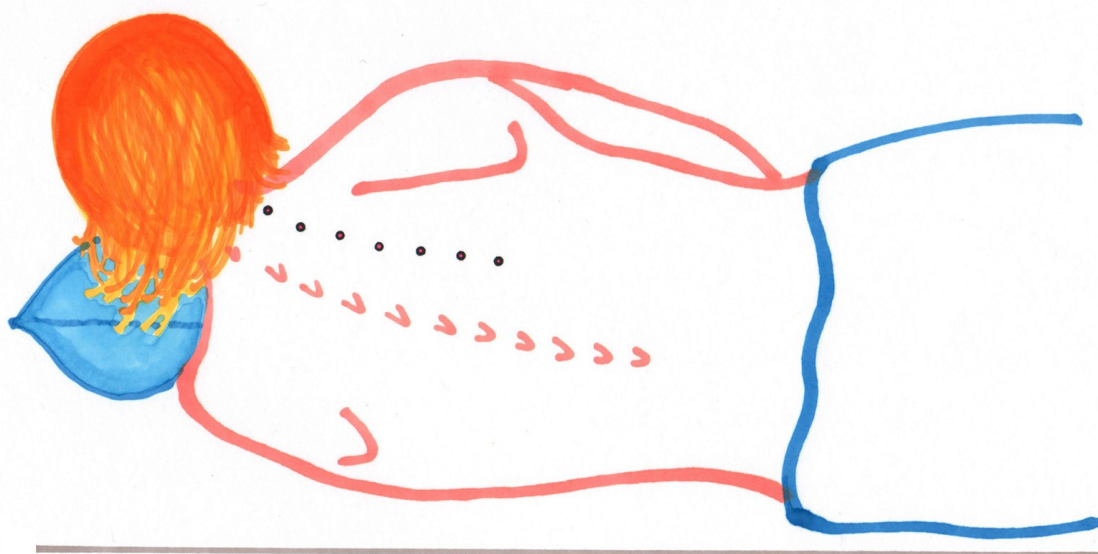
Anatomy of intercostal nerves (cross-sectional view).

The intracostal nerve provides preganglionic sympathetic fibers to the sympathetic chain via the white rami communicantes and receives postganglionic neurons from the sympathetic chain through the gray rami communicantes. These gray rami join the spinal nerves near their exit from the intervertebral foramina.

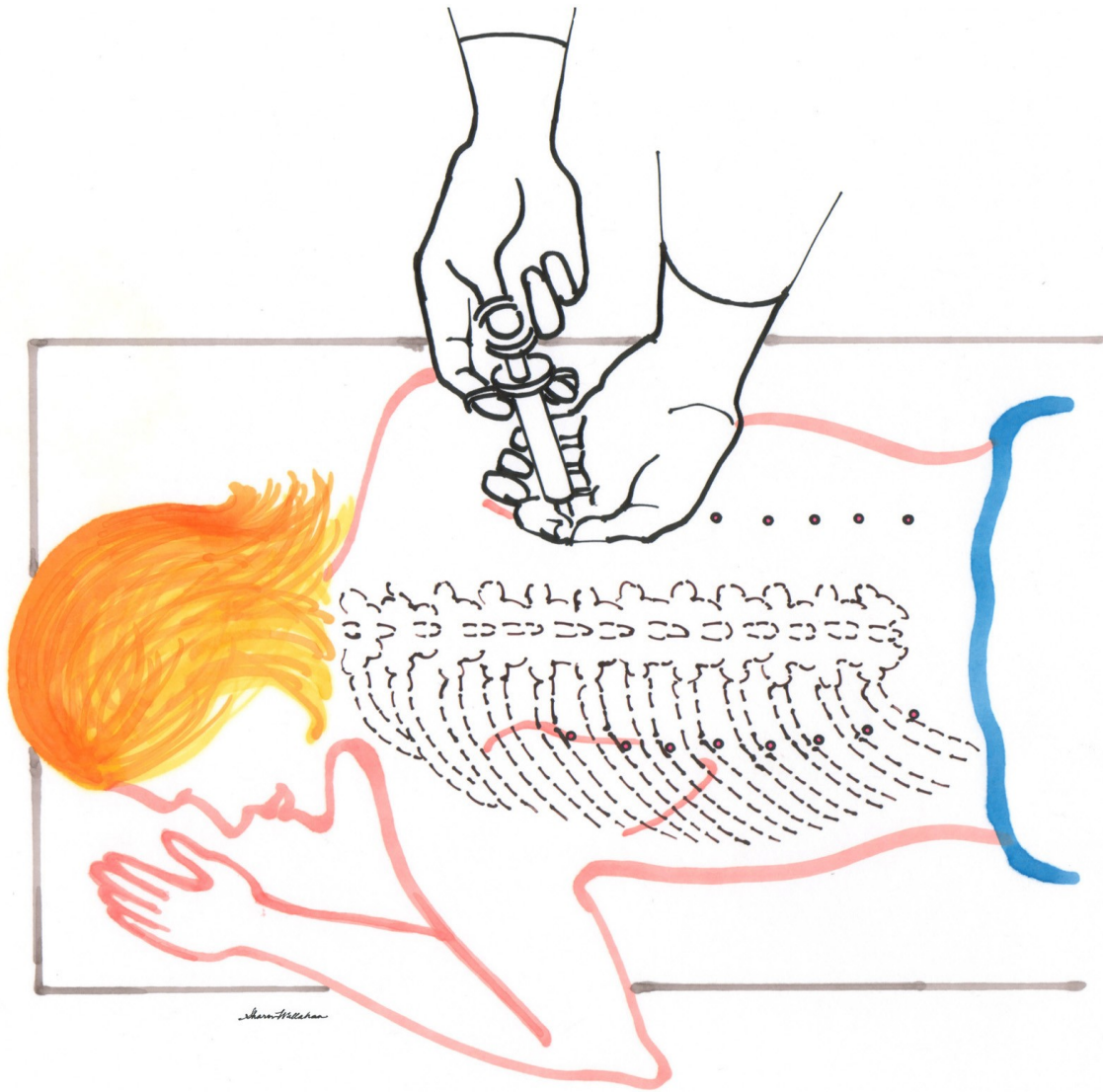


A short distance beyond the intervertebral foramina, the nerve root divides into the posterior and anterior primary divisions. The posterior primary division carries sensory and motor fibers to posterior cutaneous and muscular tissues, which are paravertebral. The primary anterior division that becomes the intercostal nerve gives rise to the lateral cutaneous branch just anterior to the midaxillary line, which sends subcutaneous fibers anteriorly and posteriorly. The intercostal nerve continues to the anterior trunk where it terminates as the anterior cutaneous branch.

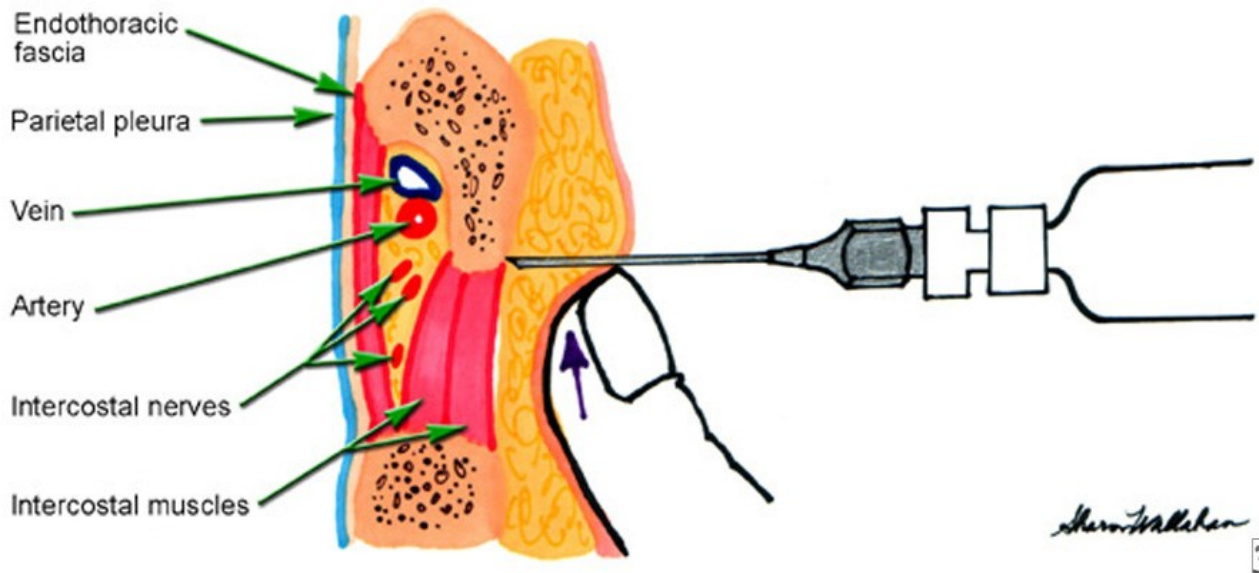
The posterior intercostal block, as described by Bonica, is carried out easily at the angle of the rib, where it is the most superficial and easiest to palpate. The patient is placed in the lateral position with the target side up if performing a unilateral block or in prone position if performing bilateral blocks. A 3-cm, 25-gauge, short-beveled needle is inserted through a skin wheal at the lower edge of the posterior angle of the rib. The second finger of the left hand is placed over the intercostal space and the skin is pushed gently cephalad so that the lower edge of the rib above can be palpated simultaneously. This technique protects the intercostal space, thus reducing the risk of passing the needle into the lung.



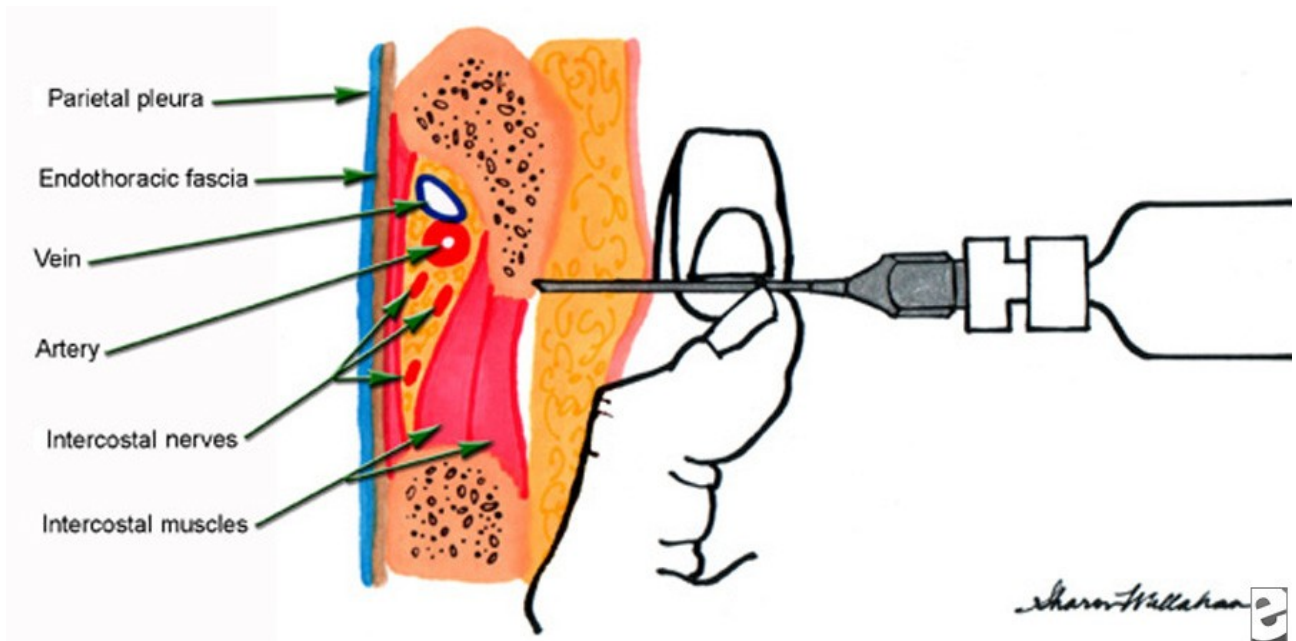
Position of patient for posterior intercostal block technique.



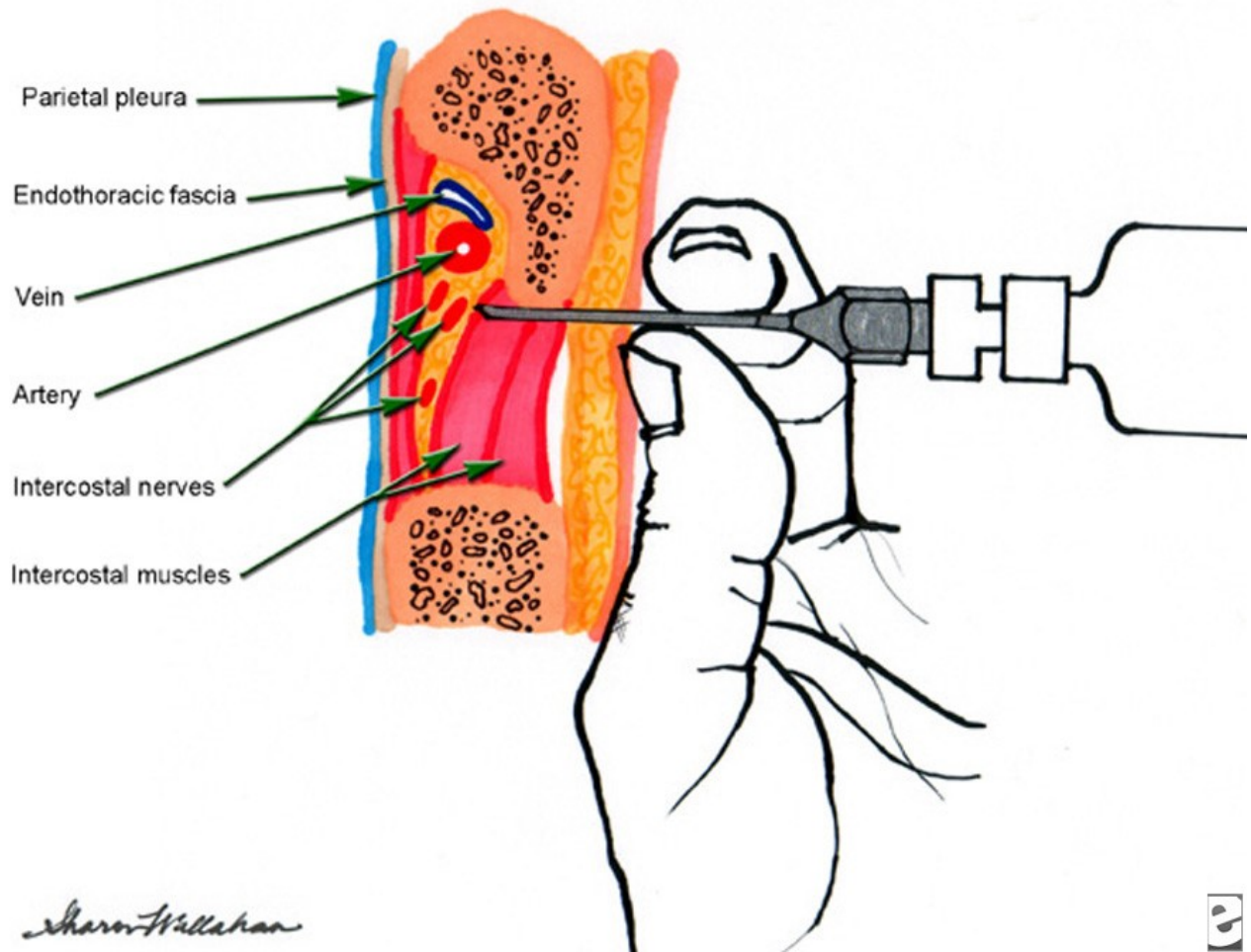
Intercostal block technique. See text for details.



A - Posterior intercostal block technique. See text for details.



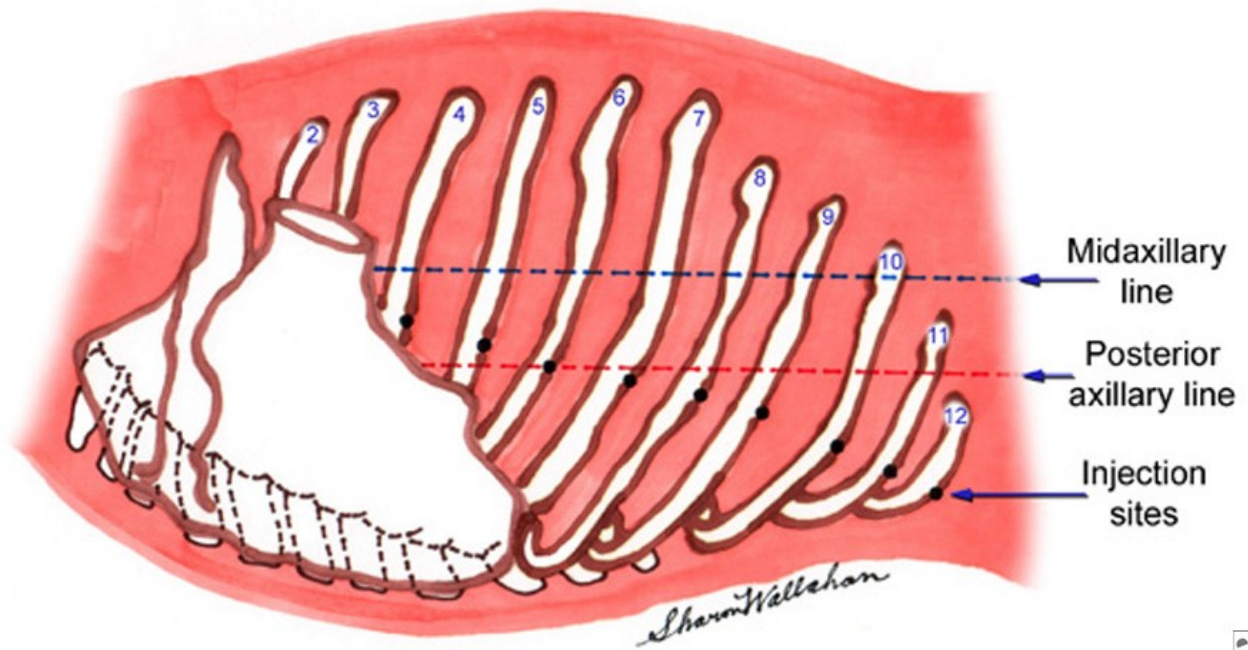
B - Posterior intercostal block technique. See text for details.



C - Posterior intercostal block technique. See text for details.

The needle is advanced until the lower part of the lateral aspect of the rib is reached. After reaching the rib, the needle is grasped with the thumb and index finger of the left hand about 3-5 mm above the skin surface. The skin is moved caudally with the left index finger to allow the needle to slip just below the lower border of the rib and then the needle is advanced until the left thumb and finger grasping the needle become flush with the skin. Aspiration is attempted; if negative, 3-4 mL of LA solution is injected. This LA solution diffuses several centimeters distally and proximally to involve the sympathetic chain, which may also block visceral nociceptive pathways, thus helping to relieve pain, which arises from painful viscera as well. Injection of larger volumes will result in both paravertebral and epidural spread of the drug, which may cause arterial hypotension if many segments are involved.

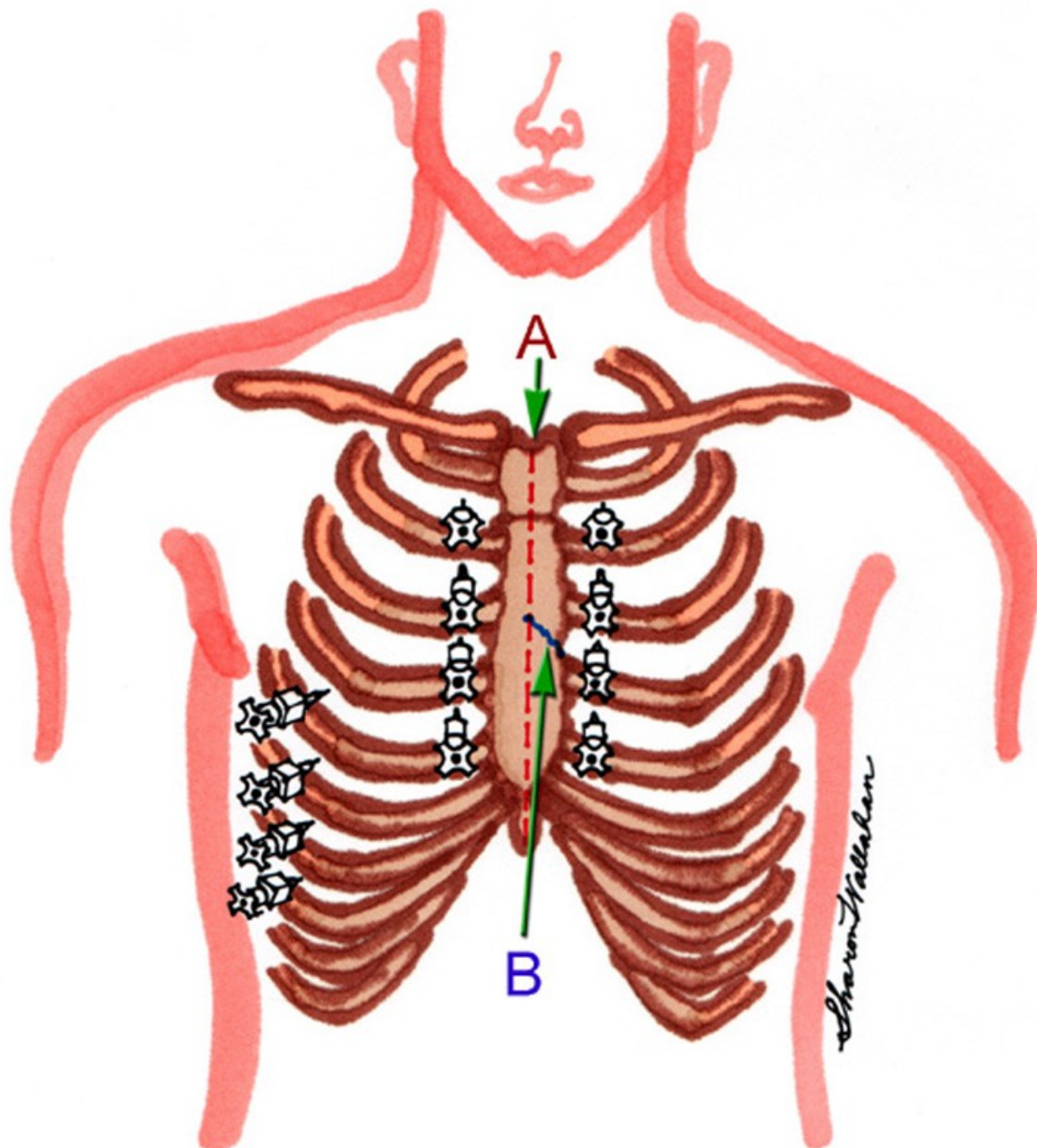
The lateral intercostal block technique described by Bonica is performed 3-4 cm posterior to the midaxillary line where the lateral cutaneous nerve pierces the intracostal muscles and divides into anterior and posterior branches. The anterior branch supplies skin and subcutaneous tissues of the anterolateral chest and abdominal wall as far as 7 cm from the midline; the posterior branch supplies tissues as far as 7-10 cm from the spine. A block at this site is unlikely to diffuse to the paravertebral region and therefore is preferable to differentiate thoracic and abdominal visceral pain from somatic pain caused by disorders of the chest and abdominal wall.



Lateral intercostal block along the posterior axillary line.

Intercostal block at this site produces less ventilatory impairment than blocks at other sites; therefore, it is often considered preferable for patients with pulmonary disorders. Because a block at this site does not relieve postoperative pain from the viscera, however, supplementary pharmacologic analgesia may be necessary.

Anterolateral intercostal block is performed in the anterior axillary line proximal to the takeoff of the anterior cutaneous branches of the thoracic intercostal nerves and is useful for alleviating the pain of sternotomy, fracture of the sternum, and dislocation of costicartilage articulations.



Anterolateral intercostal nerve block technique. See text for details.

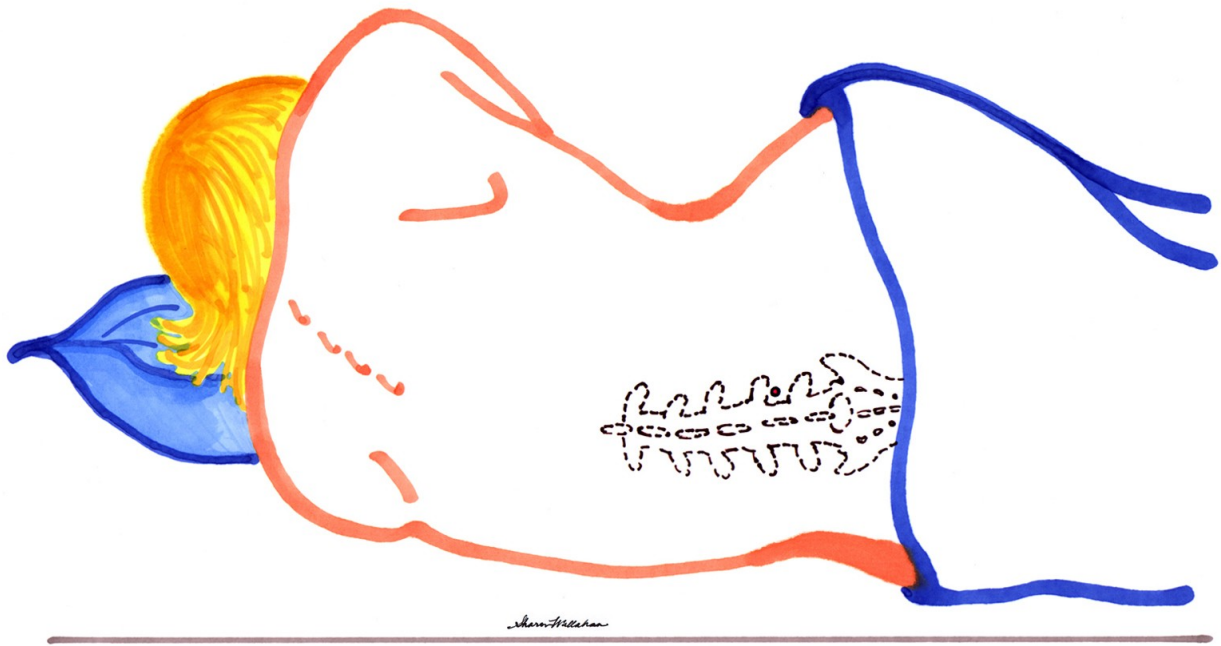
This technique also can be used to block the cephalad 3 or 4 abdominal intercostal nerves just proximal to the costochondral articulation to provide analgesia in the upper abdominal wall. Like the lateral intracostal block, this procedure does not interrupt visceral nociceptive pathways.

Thoracic zygapophyseal joint blocks have received little attention in the literature. The orientation of these facet joints does not lend them to the posterolateral approaches used for intra-articular injections as in the cervical or lumbar spine. Furthermore, the exact course of the medial branches of the thoracic dorsal rami and the pattern of innervation of these joints has not been researched adequately.

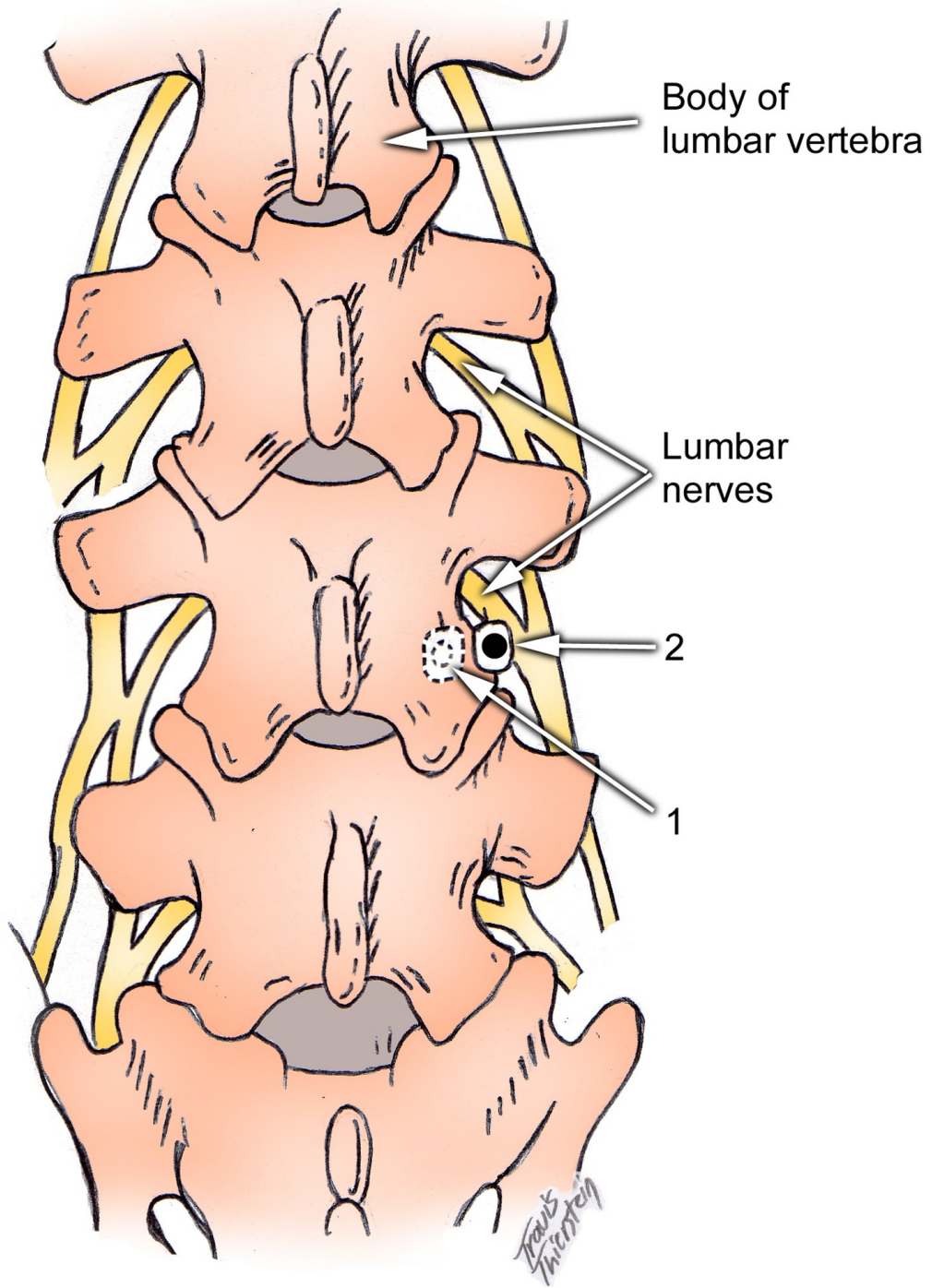
Paravertebral lumbar somatic nerve root blocks can provide diagnostic information and also pain relief associated with painful nerve root compression, vertebral body and intervertebral disk pathology, and peripheral nerve disorders. These blocks can also reduce reflex spasm of the hip adductor muscles in patients with spasticity or paraplegia.

Needle placement can be verified by radiography, eliciting paresthesia, or using a nerve stimulator.

The technique described by Bonica begins with the patient in a prone position. A skin wheal is placed 1.5 cm lateral to the upper portion of the spinous process. A 5-cm, 25-gauge needle is directed vertically downward, while tissues along the way are infiltrated with 5-7 mL of a dilute LA solution (eg, 0.5% lidocaine or 0.125% bupivacaine) until the needle impinges upon the lamina of the vertebra. An 8-cm, 22-gauge needle is inserted perpendicular to the skin in the parasagittal plane through the anesthetized area, until the second needle reaches the uppermost part of the lateral edge of the lamina. After contact with the lamina, the needle is marked 1.5 cm above the skin. The needle is then withdrawn until it is subcutaneous in location, then moved laterally approximately 1.5 cm and advanced past the lamina to a depth of 1.5 cm, where it should make contact with the nerve, eliciting paresthesia.



Position of the patient for lumbar paravertebral somatic block technique.



Lumbar paravertebral somatic block technique. See text for details.

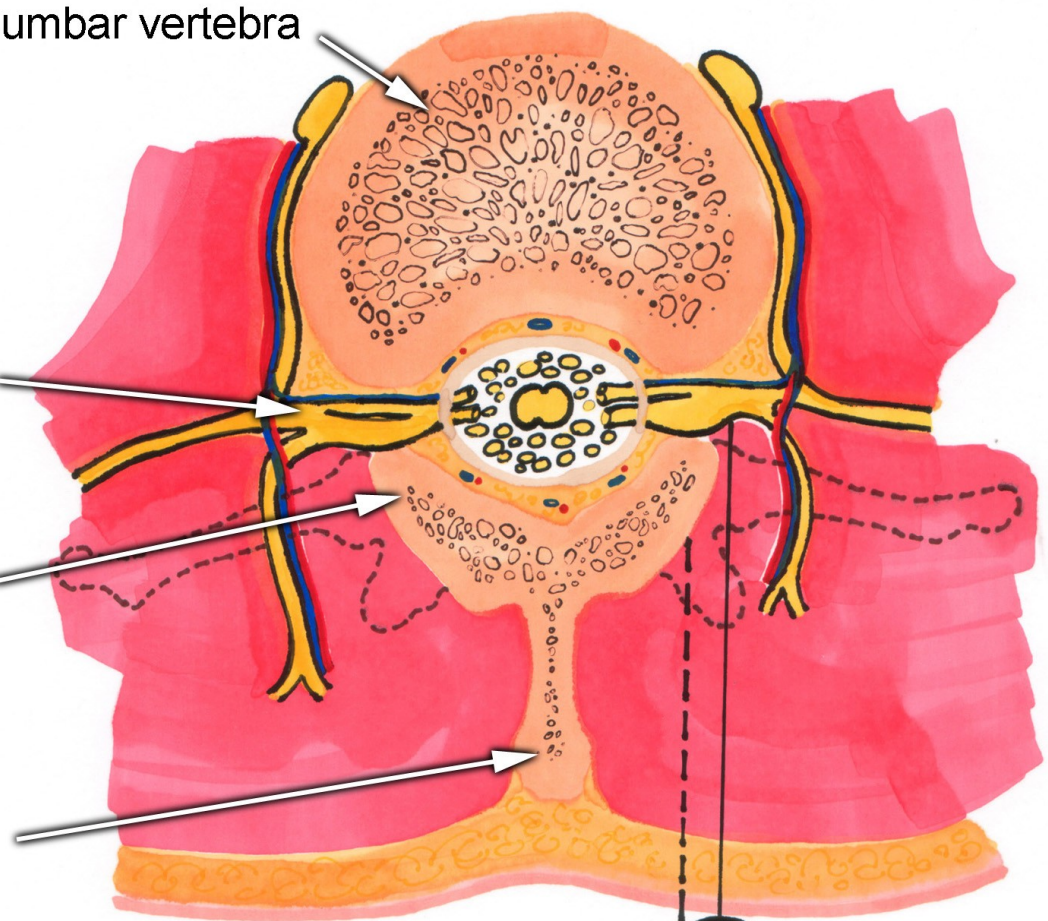


Body of lumbar vertebra

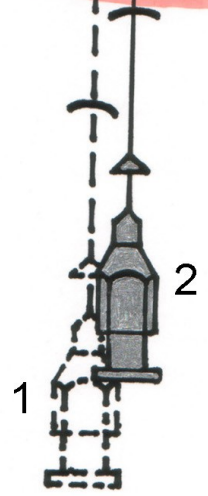
Lumbar nerves

Lamina

Spinous process

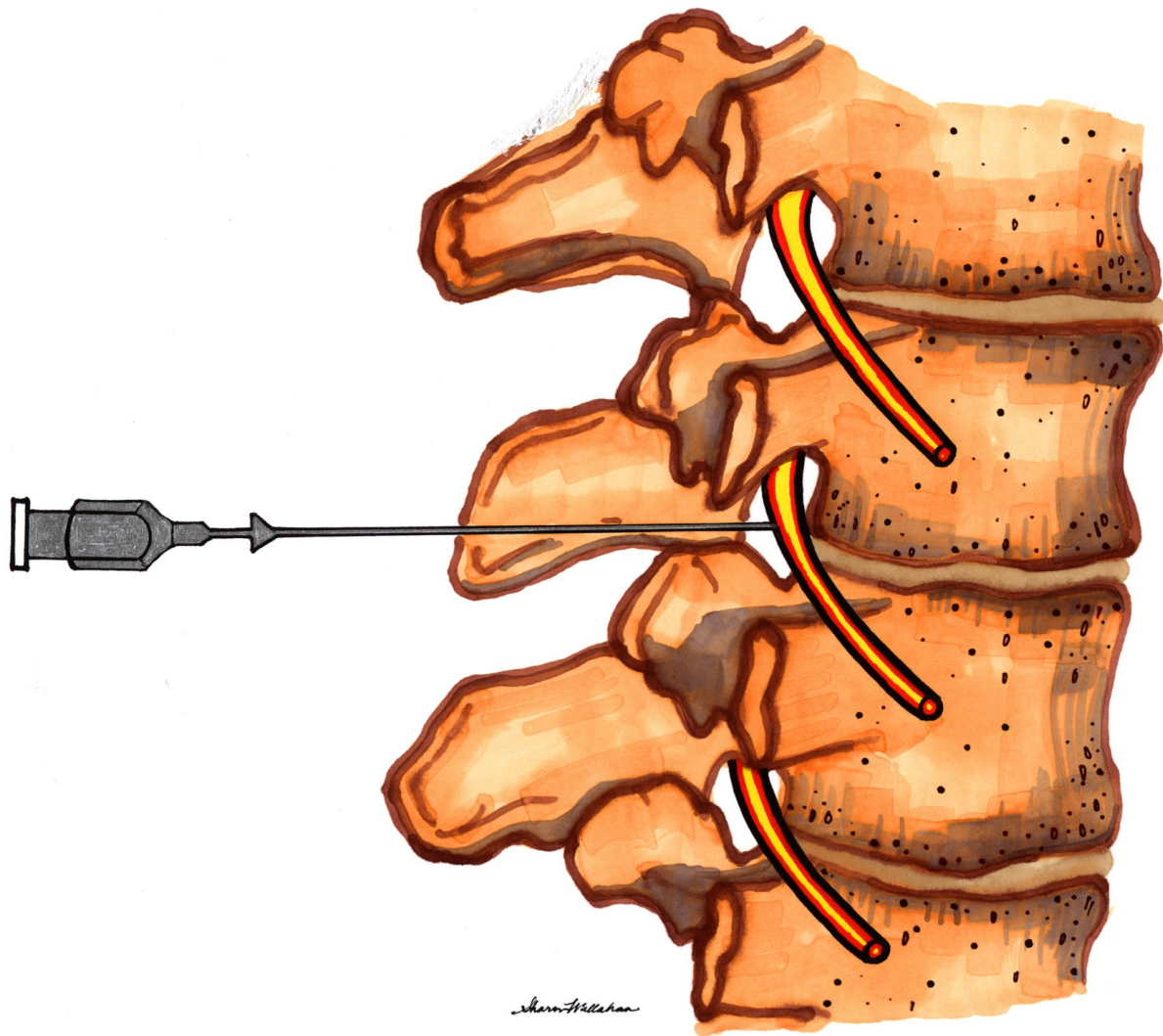


Sharon Willahan



Lumbar paravertebral somatic block technique. See text for details.





Lateral view showing needle position of lumbar paravertebral somatic block technique.

For diagnostic or prognostic purposes, 2 mL of a potent LA solution (eg, 0.5% bupivacaine with epinephrine) typically is injected after radiographic verification of the position of the needle bevel. This volume is sufficient to block the nerve as it exits from the intervertebral foramen, provided the needle tip is within 1-2 mm of the nerve. For therapeutic purposes, 5 mL of solution can be used to prolong analgesia but this is likely to spread to one or more adjacent segments. Multiple nerve roots can be addressed by injecting 25-30 mL of LA solution into the psoas compartment, which contains the lumbar plexus. This will spread sufficiently to block sympathetic nerves, the lumbar plexus, and lumbosacral trunks.

Intra-articular lumbar zygapophyseal joint blocks, as described by Bogduk et al, are performed with the patient lying prone. Upper lumbar facet joints originate in the sagittal plane; therefore, the joint space is usually evident on posteroanterior fluoroscopy with the patient prone. The lower lumbosacral zygapophyseal joints are oriented obliquely at an angle of approximately 45° to the sagittal plane.

To permit visualization of the joint space, the patient has to be rotated appropriately and supported in an oblique prone position or a C-arm fluoroscopy unit capable of tilting the x-ray beam. A 22- or 25-gauge, 9-cm spinal needle is the most practical for accessing the target joint cavity. Finer needles enter the joint space more easily but are apt to stray during penetration of the back muscles. If this difficulty is encountered, then a double needle technique can be used, in which a large gauge needle is introduced to the target joint and a finer needle is passed through the larger needle to penetrate the joint capsule.

The operator relies on feel to determine when the needle enters the joint and limits penetration of the joint no farther than its center. Correct placement of the needle inside the midpoint of the joint is confirmed by injection of a

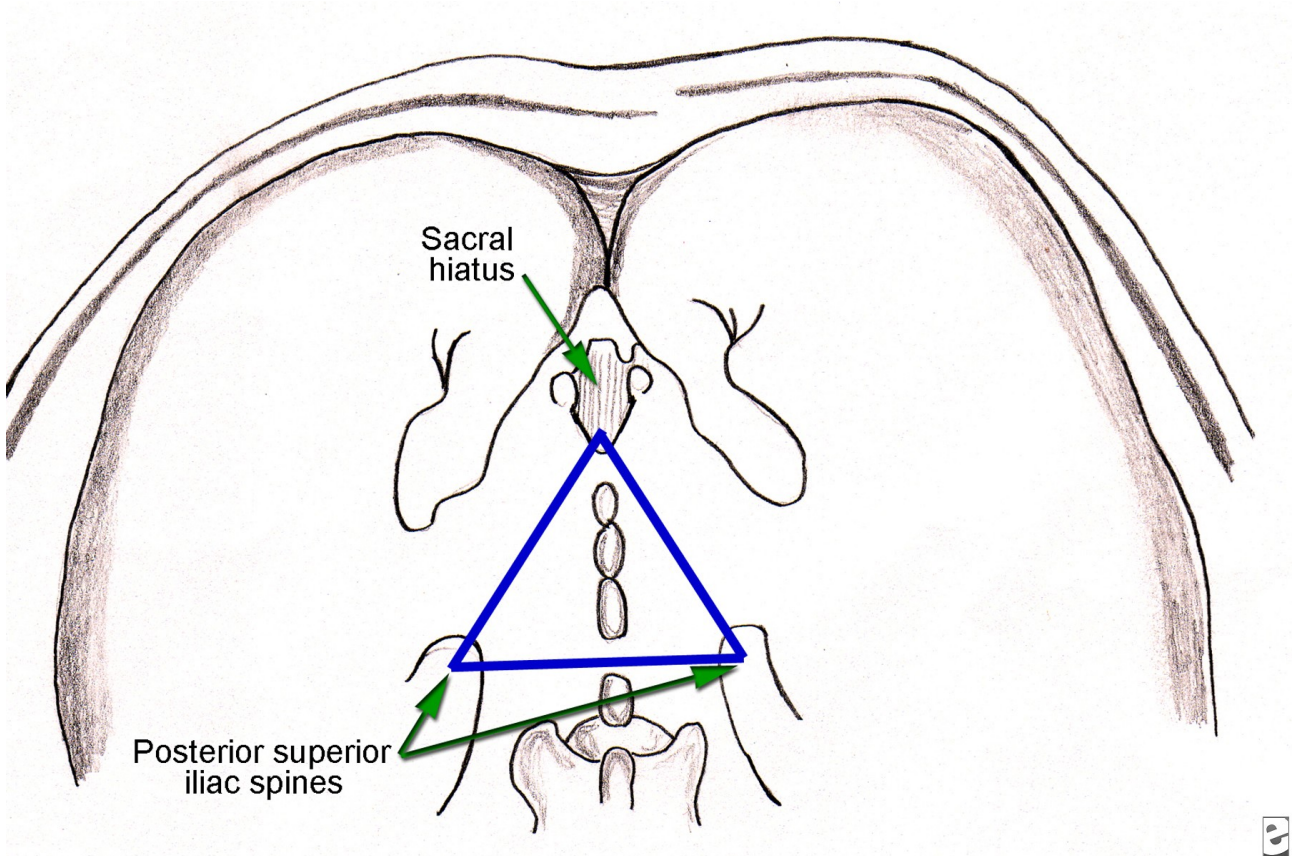
small quantity of contrast medium (less than 0.3 mL) using a small syringe (2-5 mL) to minimize injection pressure. If the needle is inside the joint, the arthrogram smoothly outlines the smooth perimeter of the joint space. At this point in the procedure, the patient should be questioned in detail whether the character of pain that is perceived as a result of the dye injection is concordant to the pain for which treatment is being sought. Once intra-articular placement has been verified, LA can be used to ablate the pain and provide additional diagnostic verification. Addition of corticosteroids has been reported to provide therapeutic benefit; however, no more than a total of 1 mL of any solution combination should be injected.

Lumbar medial branch blocks, whose technique also has been described by Bogduk et al, are performed under fluoroscopic guidance. The needle target for medial branch blocks of L1-L4 is on the dorsal surface of the transverse process, where it joins the superior articular process. The medial branch of each of these lumbar segments crosses the transverse process of the segment below; for example, the L4 medial branch crosses the L5 transverse process and so on. A 22- or 25-gauge, 9-cm spinal needle typically is used to perform the block. Larger needles are easier to maneuver through the back muscles, while finer needles are averted more easily from their intended course.

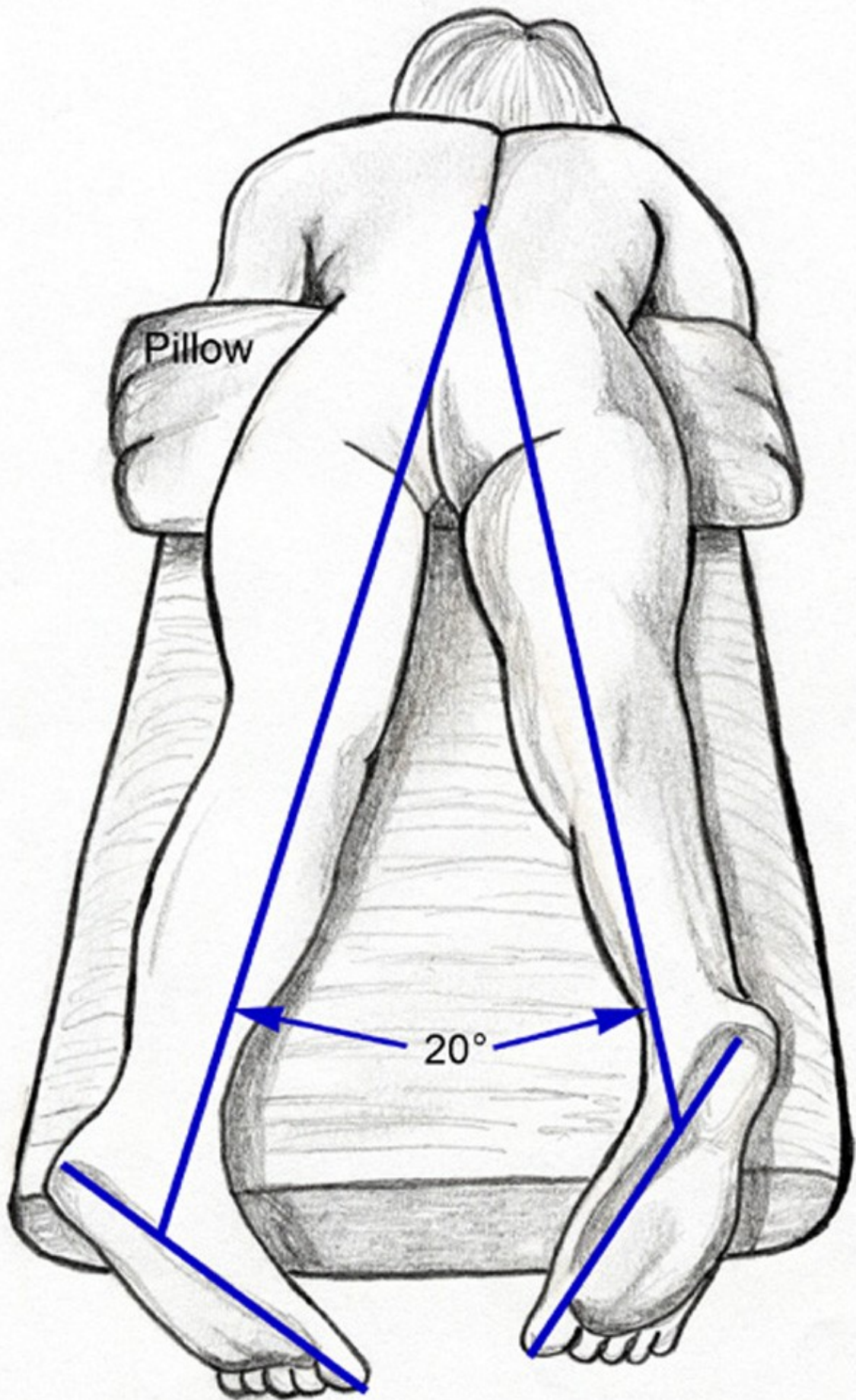
Sacroiliac joint blocks are challenging because the joint cavity lies deep to the corrugated interosseous surfaces of the sacrum and ilium and is joined by the dense interosseous sacroiliac ligament. Entry into the joint is most practical below the interosseous ligament deep to the gluteus maximus muscle along the upper margin of the greater sciatic notch. Using the technique described by Bogduk et al, the patient is placed in the prone position and a 25-gauge spinal needle is inserted through a skin wheal into the gluteus maximus and advanced until it engages the posterior aspect of the sacrum.

The operator must be cautious to avoid the greater sciatic foramen and redirect the needle toward the lower end of the joint space. Once the needle enters this slitlike opening, it is wedged between the sacrum and ilium and should be in the correct position. Penetration should be just deep enough to engage the slit; further penetration may cause the needle to emerge from the ventral surface of the joint. Contrast medium then is injected to verify placement and pain concordance. LA with or without supplementary agents may be injected for diagnostic and therapeutic purposes.

Caudal blockade has become more popular as a method of inducing epidural anesthesia and for catheter entry to locate specific spinal pain generators. To perform caudal blockade using the technique described by Brown, the patient is placed in a lateral decubitus or prone position. The prone position is more amenable to accurate identification of midline anatomical targets in adults. A pillow placed beneath the lower abdomen produces slight flexion of the lumbar spine. Mild sedation improves patient comfort. Relaxation of the gluteal muscles is induced in the prone patient by a 20° hip adduction and the feet pointed inward by internal rotation of the hips. A 22-gauge or larger needle is recommended for adult patients, and some needles allow introduction of a catheter, which can be directed under fluoroscopy using a steering wire.



Surface anatomy of caudal block and sacral hiatus localization.



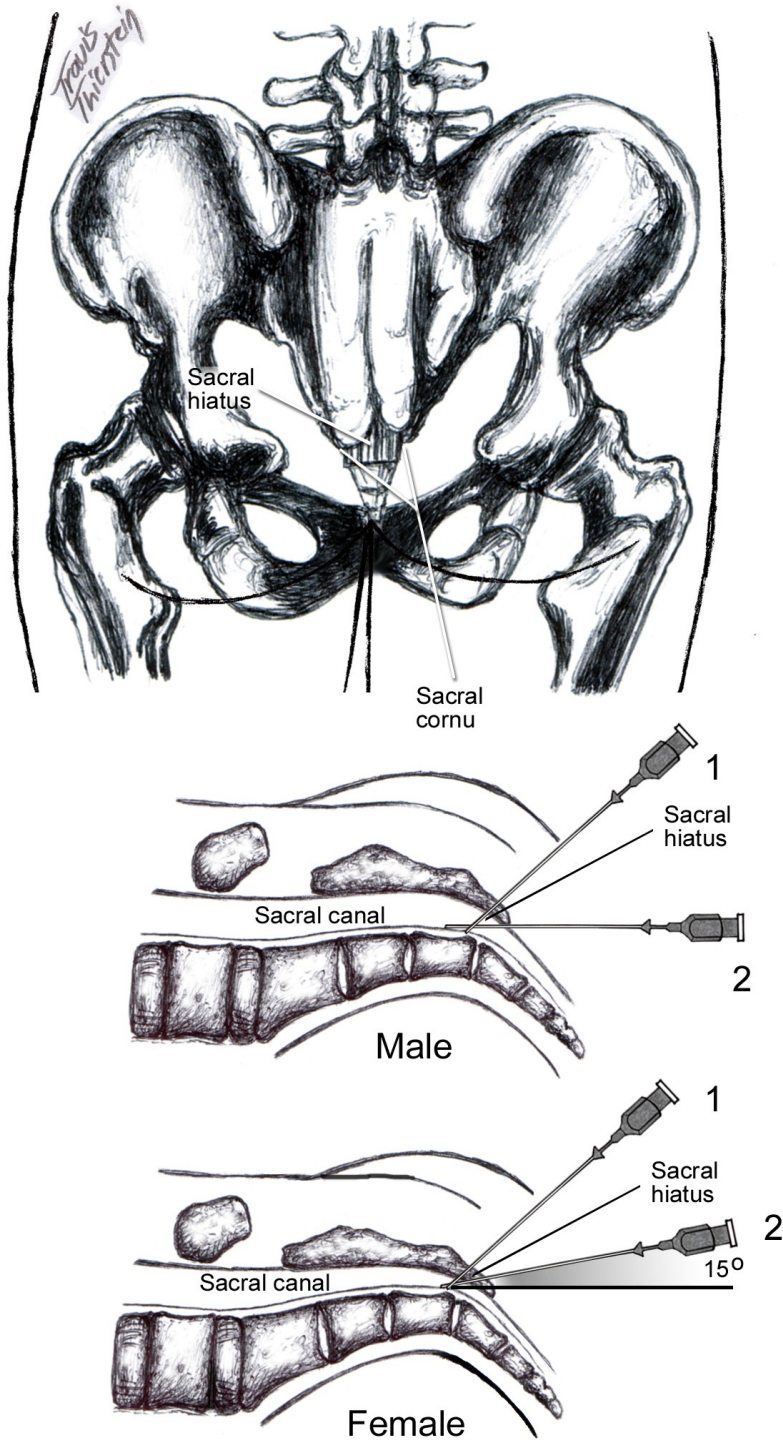
Pillow

20°

Toes pointing inward



Patient in the prone position for caudal block technique.



Caudal block technique. See text for details.

After the sacral hiatus is identified, the index and middle fingers of the palpating hand are placed on the sacral cornu and the caudal needle is inserted at an angle approximately 45° to the sacrum. As the needle is advanced, the operator can sense a reduction in resistance when the needle enters the caudal canal. The needle is advanced until bone is contacted on the dorsal aspect of the ventral plate of the sacrum. The needle is then withdrawn slightly and redirected at an angle more parallel to the skin surface. In male subjects this angle is usually about parallel to the



tabletop, whereas in female patients a slightly steeper angle is often necessary. After the needle is redirected, it should be advanced approximately 1-1.5 cm into the caudal canal. Further needle advancement should be avoided to prevent unintentional intravascular cannulation or dural puncture. At this point in the procedure, a catheter can be threaded and directed by fluoroscopy to the desired spinal level and structures.

Caudal anesthesia and neural blockade carry the same complications that can accompany lumbar epidural anesthesia; however, the incidence of subarachnoid puncture is much lower with the caudal technique. The dural sac ends approximately at the level of S2; therefore, unless the needle is inserted deeply within the caudal canal, subarachnoid puncture is unlikely. The most commonly encountered problem with caudal anesthesia is ineffective neural blockade.

Subarachnoid block, also termed spinal anesthesia (SA), can be achieved with small amounts of LA (eg, 100-150 mg procaine, 50-100 mg lidocaine, 5-15 mg bupivacaine) placed into the subarachnoid space where it readily mixes with the CSF. SA produces a rapid onset of analgesia, because the drug comes into direct contact with neural structures, especially nerve axons, without first traversing the epineurium and perineurium. Furthermore, SA is a relatively simple procedure when administered by experienced hands, and it allows better control of the degree and duration of neural blockade. LA solution can be made hyperbaric (ie, specific gravity CSF), which allows the spinal level of the block to be controlled by changing the position of the patient.

Notwithstanding, these advantages of SA have limited value when managing patients with acute pain, and SA rarely is indicated as a therapeutic tool for patients with chronic pain. SA is frequently useful as a prognostic block prior to a subarachnoid injection of a neurolytic agent or for diagnostic purposes.

Differential subarachnoid block can be used as a diagnostic procedure in differentiating pain caused by somatic nociceptive sensory nerves, sympathetic hyperactivity, and pain from a primarily central source, including that of psychogenic etiology. Classically, this is performed by an anesthesiologist who inserts a microcatheter into the subarachnoid space. Bonica described a technique using a 32-gauge polyamide catheter, 91 cm long, which can be inserted through a 25- or 26-spinal gauge spinal needle. During the procedure, cardiorespiratory monitoring, as well as sympathetic, sensory, and motor neural assessment, should be ongoing. After insertion of the catheter, 8-10 mL of saline solution are infused as control. Some anesthesiologists have advocated aspiration of 8 mL of CSF and then CSF re-injection because of the controversial belief that isotonic saline solution may induce a change in sensation.

The operator then injects 8-10 mL of 0.25% procaine, which should produce a sympathetic neural blockade; sympathetic neural functions are monitored, as well as any reported changes in the patient's pain. Subsequently, 8-10 mL of 0.5% procaine is injected to produce a sensory block, which can be assessed by pinprick, touch, and pinch. Finally 8-10 mL of 1% procaine is injected to produce a motor blockade. During each stage of the procedure, the patient's pain intensity, spinal level of the sensory block, and neurophysiological and behavioral changes, as well as the quality of the analgesic effect, are monitored.

Pain that responds to isotonic saline or "placebo" is presumed to have a nonnociceptive origin; therefore, possible contributing psychogenic factors should be evaluated. If a sympathetic blockade accompanied by objective evidence of sympathetic interruption alleviates the pain, sympathetic hyperactivity may account for a component of the pain. Elimination of the pain with 0.5-1% procaine should indicate that the pain has a somatic origin. Failure of any solution to block the pain also implies a central or psychogenic etiology.

Extradural or epidural blockade can be varied to suit the spinal segmental level of the patient's symptoms. Blockade can be achieved with a single injection of LA through a needle placed at the appropriate segmental level or by introduction of a catheter through a thin-walled 18- or 17-gauge needle placed at the spinal level, which is considered clinically to be the optimum site for injection. Injections into the lumbar epidural space can be accomplished through either a caudal or lumbar approach.

The lumbar approach involves passing the needle through the intralaminar space along the midline through the interspinous ligament or slightly to the side of the ligament, then penetrating through ligamentum flavum to enter the epidural space. Perceived advantages of the lumbar route are (1) the needle is directed more closely to the assumed site of pathology, (2) the drug to be injected can be delivered directly to its target (ie, more target specific), and (3) lesser volumes of the injected solution can be used.

Continuous epidural block often is used to eliminate chronic persistent pain secondary to somatic, visceral, or sympathetic etiologies. This procedure can be used for relieving the severe pain associated with pancreatitis, biliary colic, renal or ureteral colic, multiple fractures of the ribs, and severe posttraumatic pain. Postoperative pain of the thorax, abdomen, pelvis, and/or lower limbs is also a common indication. In all these acute conditions, blockade provides not only analgesia by interruption of nociceptive pathways from somatic structures and viscera, but also blocks reflex muscle spasm, sympathetically induced ileus, and neural endocrine responses that may codevelop with acute injury and disease. Continuous epidural anesthesia also can be achieved using minute doses of soluble opioids.

Clinical Application of Spinal Injection Techniques

Epidural corticosteroid injections reportedly were used first in 1952 by Robecchi and Capra, who claimed to provide relief of lumbar and sciatic pain in a woman after periradicular injection of hydrocortisone into the first sacral root. Sacral epidural injection of steroid by the transforaminal route was largely popularized in Italy and involved passing a needle through the first dorsal sacral foramen to gain access to the first sacral nerve roots.

Caudally administered solutions require a substantial volume so that the injectate reaches the lumbar nerve roots, which lie approximately 10 cm or more cephalad to the site of injection. Frequently, a threaded catheter inserted under fluoroscopic guidance provides more precise anatomical application, thereby avoiding the complications associated with injection of a large volume of fluid. Traditionally, clinicians and investigators have used methylprednisolone or triamcinolone, mixed with variable, often large, volumes of LA and isotonic saline or sterile water, for spinal injections.

Corticosteroids may be administered into the lumbar epidural space through either a caudal or lumbar approach, with the latter approach advocated as more target specific and requiring smaller volumes of injectate. For the same reason, many spine specialists advocate transforaminal steroids because this route of administration is placed more precisely at or near the presumed painful nerve root. Once the drug is injected into the epidural space, the operator has no control over dispersal, which is governed by injection volume and pressure and the anatomy of the epidural space.

Normal epidural ligaments or epidural scarring may obstruct passage of injectate to the desired site. To overcome these perceived difficulties, some operators advocate delivering the drugs into the epidural space immediately surrounding the nerve root. Therefore, the target nerve root is approached with the needle under radiographic guidance along an oblique paravertebral approach. Targeting the root, and not the epidural space, is more likely to deliver the corticosteroid solution to the affected nerve root.

The rationale for use of epidural steroids was based on the belief and some supporting literature, including animal studies, that lumbosacral radiculopathies may have an inflammatory component. A study performed by Ryan and Taylor arbitrarily divided 70 patients into 2 groups -- those with "compressive radiculopathy" and those with "irritative radiculopathy." The former group was characterized by sciatica with sensory, motor, and reflex disturbances, while the latter was characterized by sciatica alone. Intrathecal and epidural injections of corticosteroids produced better therapeutic responses in the group with "irritative radiculopathy." Furthermore, responders tended to have higher CSF protein levels and a shorter duration of illness, particularly with sciatica lasting less than 2 weeks.

Therefore, epidural corticosteroids may provide relief in some cases of radiculopathy owing to their anti-inflammatory properties; however, research has demonstrated that methylprednisolone has a direct inhibitory action upon nociceptive input. In summary, many practitioners advocate the use of epidural corticosteroids as treatment for inflammatory-type radicular pain and preclude their use for axial or referred somatic pain. Since nerve root inflammation has not been implicated as the only cause of back pain, no confirming data exist to support the use of epidural steroids for low back pain alone.

Epidural corticosteroids should be used with caution or avoided in some cases of congenital anomaly or prior surgery that has altered the normal anatomy of the epidural space, when corticosteroids may unmask an infection, in patients with coagulopathy, and in patients susceptible to fluid retention and congestive heart failure. Epidural corticosteroids are absorbed systemically, which may cause suppression of adrenal function for up to 2-3 weeks.

Other "red flags" that should warn practitioners considering use of corticosteroids include patients with significant contributing operant and psychosocial factors, clinical presentation suggestive of somatization, nonmechanical back pain, disability related to the lumbosacral syndrome under treatment, normal straight leg raising, and pain that is not decreased by medication of any type. Factors that seem to have no bearing on the decision to use corticosteroids include age, pattern and frequency of pain intensity, results of physical examination, and presence or absence of structural pathology.

Corticosteroids have been advocated using the same techniques and operational procedures as described previously in this article for somatic, transforaminal, and epidural neural blockade. The issues associated with the use of epidural corticosteroids include those attributed to injection technique and local anesthetics. Infection is possible following any injection but is an exceedingly rare complication of epidural corticosteroids and has been documented only in several case reports.

Arterial hypotension has been reported as a complication of epidural steroids unrelated to LA toxicity. Other adverse effects ascribed to corticosteroids have included nausea, vomiting, respiratory insufficiency, insomnia, and facial flushing.

The technical risks of epidural steroid injection include bloody tap, nerve root injury, and dural puncture. Dural puncture usually is associated with postural or low-pressure headaches, which are increased in intensity when the patient is vertical and improve in deliberate fashion when the patient moves to a horizontal position. Bed rest, fluid intake, and caffeinated beverages usually resolve such headaches; however, in some cases, placement of a blood patch is necessary over the presumed site of dural puncture.

Uncontrolled studies on the use of caudal epidural corticosteroids have shown benefit in 33-77% of patients. One study indicated no significant difference in outcome between a group of 24 patients treated with caudal injections of procaine 1% and methylprednisolone 80 mg and another group of 24 patients treated with procaine alone. Two additional studies purported to show that caudally administered LA mixed with corticosteroids yielded a clinical benefit, but comparison data were found to lack statistical significance. Another 3 studies reporting the same results were methodologically flawed. Overall, the published literature supports the therapeutic use of caudal epidural steroids for the treatment of radicular symptoms; however, the scientific evidence is weak.

On assessment, the published medical literature also is favorably disposed toward the use of lumbar epidural LA and corticosteroid combinations for radicular symptoms, although more negative studies have emerged evaluating the lumbar epidural approach than evaluating the caudal approach. Dilke et al studied 100 patients with unilateral sciatica who received either active treatment consisting of lumbar epidural injection of 40 mL of 0.75% lignocaine with 80 mg of methylprednisolone and 25 mg of hydrocortisone, or a control injection of 1 mg of isotonic saline solution into the interspinous ligament. Significantly, more patients receiving the active treatment had their pain "clearly relieved." Three months after treatment, a greater proportion of the active treatment group had no pain, took no analgesics, and had resumed work. However, some of the treatment group had undergone subsequent surgery or other nonsurgical treatments; these latter differences were not statistically significant.

Other randomized controlled studies have shown conflicting results and been attacked as methodologically flawed. Clinical judgment remains the mainstay of support for or against the use of lumbar epidural steroid injections. Some advocate their use in the early management of selected patients with sciatica or radiculopathy; however, benefits are usually temporary.

Diagnostic spinal synovial joint blocks are used to assess whether the pain stems entirely from the zygapophyseal joints. No established clinical or radiographic features are recognized uniformly that enable practitioners to assign the posterior articulations as probable pain generators. Furthermore, degenerative features on CT scan have shown poor specificity and sensitivity in implicating these as causative of pain, and joints that appear normal have been demonstrated to be symptomatic. Aprill et al have mapped typical referral patterns that occur with provocative injections into the synovial zygapophyseal joints.

Cervicogenic headache involving the occiput and posterior portion of the head has been demonstrated as a result of injections into the C2-3 facet and lateral atlantoaxial joint. Provocation at C3-4 tends to span the entire cervical area but not to extend into either occiput or shoulder girdle. Provocation at C4-5 sends pain into the angle formed by the neck and top of the shoulder girdle. Provocation at C5-6 tends to produce pain over the supraspinous fossa to the acromion, and provocation at C6-7 provokes pain that radiates into the ipsilateral scapula. Reproducible pain patterns have been harder to establish in similar injection studies of lumbar spine facets, although provocation of these joints at L4-5 or L5-S1 usually results in pain referred into the low back, gluteal, and posterior thigh regions.

Nevertheless, the facet joints of the lumbar spine have been implicated as a source of low back pain since 1911. Injections of intra-articular anesthetic have provoked and alleviated pain. Although some spine specialists and interventionists advocate facet injections as a treatment method, several studies, including a large prospective study and 3 randomized controlled trials, showed no significant long-term benefit. Intra-articular facet injections, which are costly and invasive, should be considered as an adjunctive method for diagnostic identity of pain generator(s), and if convincing pain relief is obtained from intra-articular anesthetic block, the practitioner should remain open-minded in addressing the zygapophyseal joints as a potential pain source. Estimates for the prevalence of lumbar facet syndromes range widely in the published medical literature from as low as 7% to as high as 75%.

The prevalence of cervical zygapophyseal joint pain has been studied and estimated at 65%. Intra-articular corticosteroids have been used for presumptive zygapophyseal joint pain involving the lumbar and cervical spine. A carefully designed, double-blind study of intra-articular steroids versus saline for lumbar zygapophyseal joint pain revealed no clinically significant differences between groups at 1- or 6-month follow-up. No controlled studies of the value of intra-articular steroids for neck pain have been published. The concept of denervating painful zygapophyseal joints has been explored.

Some investigators have identified modest benefit from medial branch neurolysis with phenol. Percutaneous radiofrequency neurotomy has been advocated for neurolysis of the medial branch or for facet articular denervation as a treatment for both neck and back pain. A recent double-blind, randomized, controlled study comparing percutaneous radiofrequency neurotomy for chronic cervical zygapophyseal joint pain showed approximately 50% improvement in pain for a mean duration greater than 8 months when compared with an identical sham procedure.

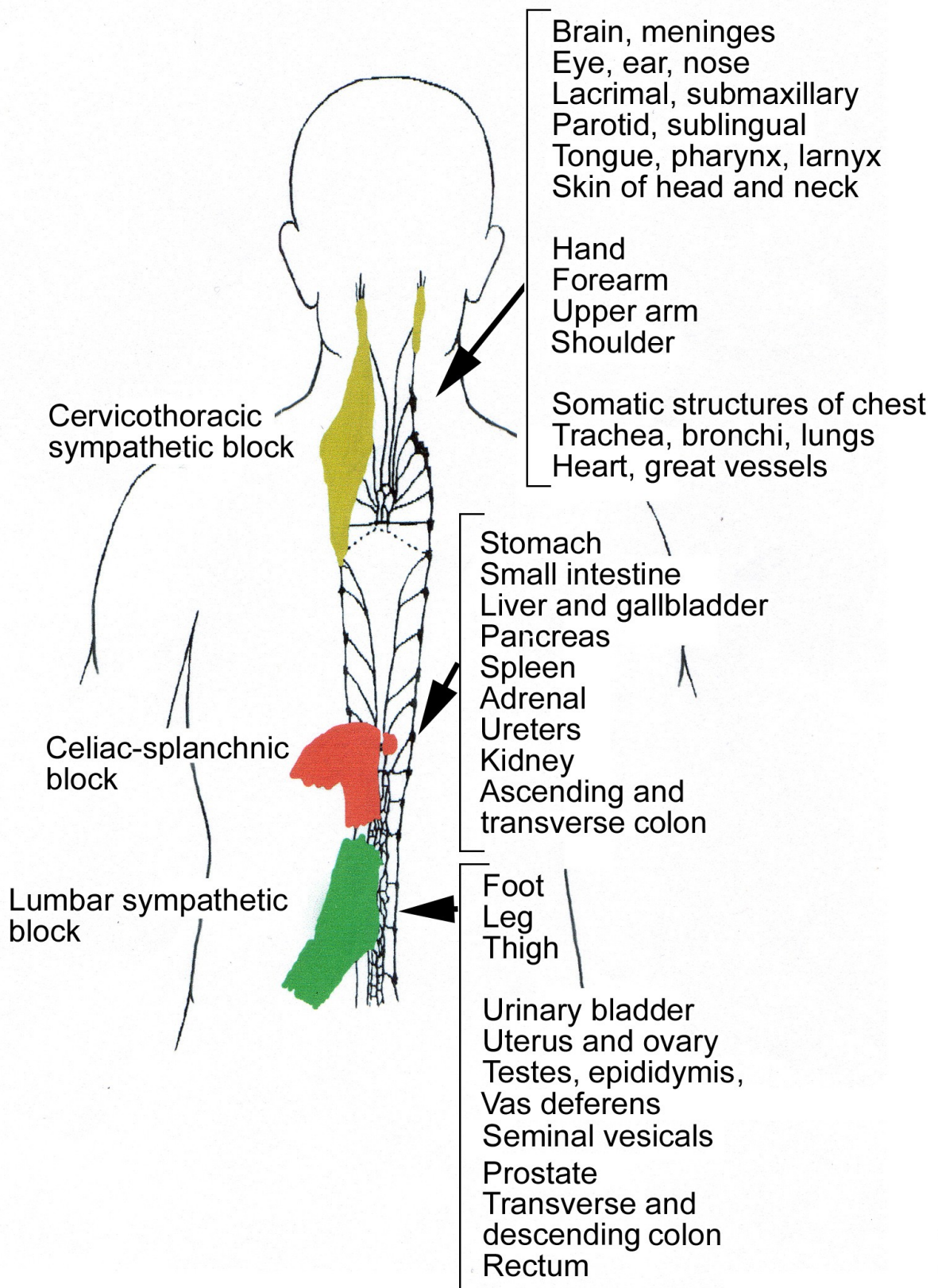
A prospective, randomized, double-blind study of injections into diskography-confirmed painful disks showed no significant difference in benefit between corticosteroids and LAs. Other interventions used to disrupt painful epidural adhesions have included hyaluronidase, hypertonic saline, and corticosteroids. Intrathecal morphine and dorsal column stimulation have been proposed as options in specific cases of severe, disabling, and intractable low back pain.

Sympathetic Nervous System Blockade

Complex regional pain syndromes (CRPS) develop as an exceedingly disproportionate consequence relative to the causative trauma affecting the limbs. Causalgia (CRPS 2) is a painful disorder that results from traumatic nerve injuries, most commonly when such damage is partial. Causalgia is a syndrome of sustained, diffuse, burning pain; allodynia (pain produced by nonnoxious stimuli) with hyperpathia (painful overreaction to stimuli); and vasomotor and sudomotor disturbances.

When advanced, CRPS 2 is associated with trophic changes of the affected tissues. Treatment of CRPS 1 and 2 entails sympathetic denervation of the entire limb, thus LA volume and concentration with diffusion must be sufficient to block the entire portion of the sympathetic chain that supplies the affected extremity. Following sympathetic interruption, patients should be questioned and urged to keep a diary as to the extent and duration of relief from burning pain, hyperpathia, allodynia, and sudomotor changes.

Three "critical sites" can be used to interrupt the peripheral sympathetic nervous system: the cervicothoracic (stellate) ganglion, celiac plexus, and lumbar sympathetic plexus. Usually, injection of 15-20 mL of an LA solution into the proper fascial plane near the stellate ganglion allows for sufficient spread to block the sympathetic chain from the superior cervical ganglion to the T5 ganglion, thereby inducing interruption of sympathetic innervation to the head and neck, upper extremities, heart, and most of the esophagus and lungs. Likewise, sufficient spread of 15-25 mL of an LA injectate near the celiac plexus should interrupt all sympathetic (and vagal), efferent, and afferent fibers serving the viscera in the upper abdomen. Injection of 15-20 mL at the anterolateral surface of the L2 or L3 vertebral body interrupts sympathetic innervation to the ipsilateral lower extremity and pelvis.

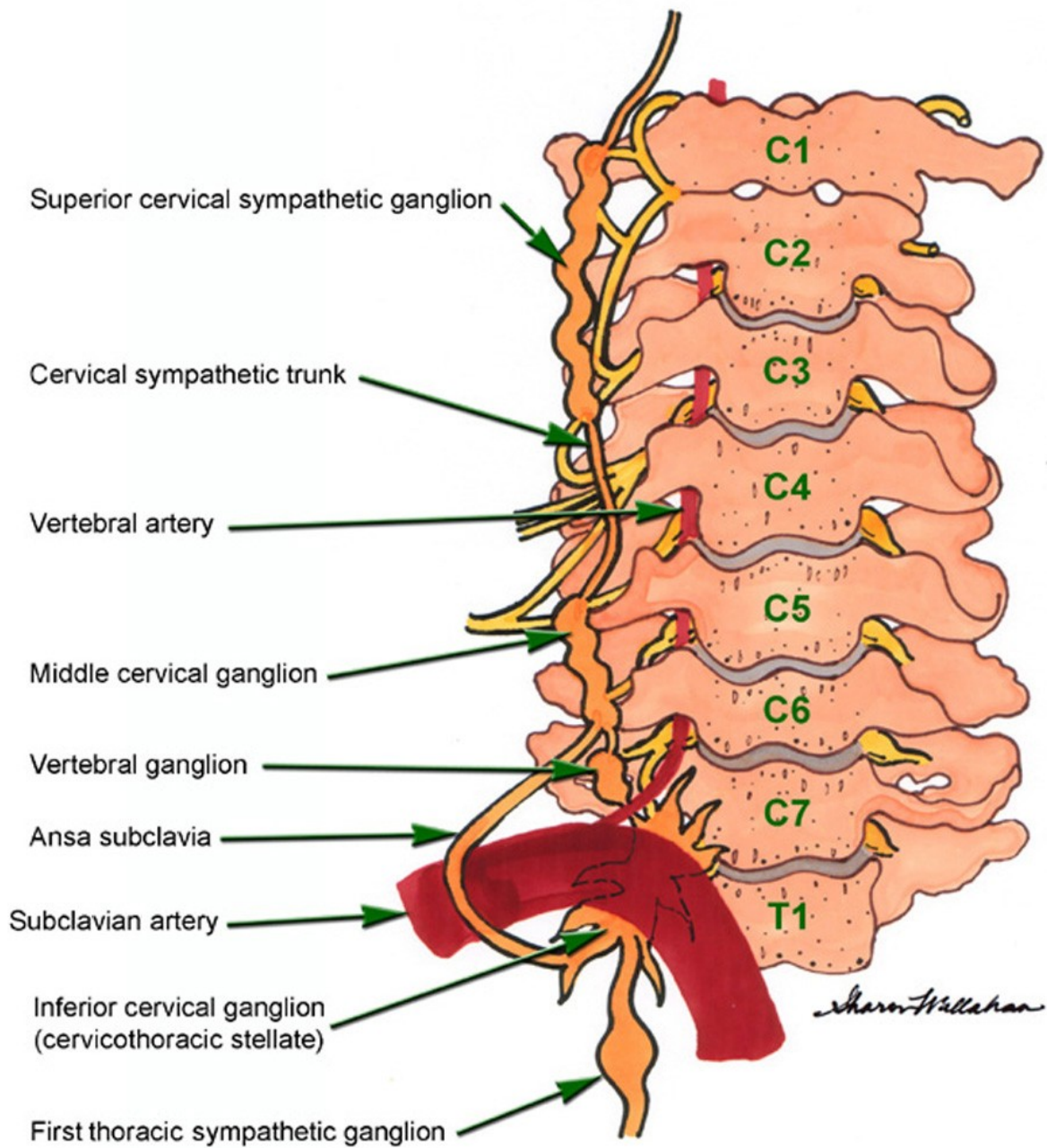


Three important sites that can be used to interrupt the peripheral sympathetic nervous system. The colored areas represent patterns of diffusion of local anesthetic solutions for the following techniques: cervicothoracic sympathetic block, celiac-splanchnic block, and lumbar sympathetic block.

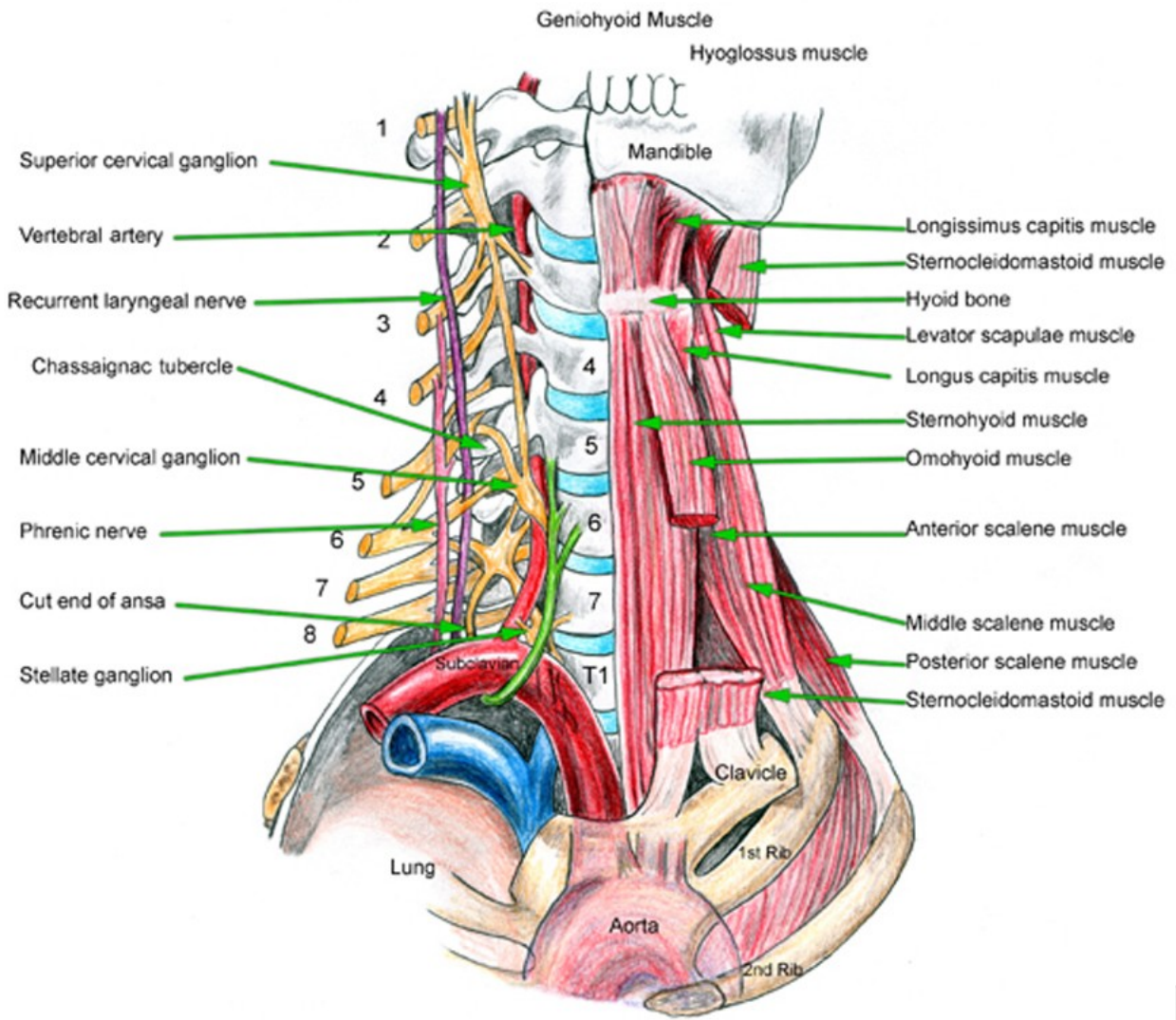
Sympathetic blockade is often useful for other pain disorders, including postamputation pain syndromes and peripheral vascular disease, such as acute or chronic occlusive arterial disease and vasospastic disorders. Blockade of sympathetic nerves to the thoracic or abdominal viscera often alleviates severe visceral pain that is not amenable to other therapies. Thoracic visceral pain, such as that of acute myocardial infarction and angina pectoris, may activate reflex coronary vasoconstriction by segmentally induced sympathetic stimulation, which conversely further aggravates cardiac ischemia.

In these cases, cervicothoracic sympathetic blockade and, if necessary, neurolytic sympathectomy may be considered useful as adjunctive treatments. Celiac plexus block or continuous segmental T5-T10 block can be used to interrupt nociceptive afferents associated with pancreatitis, biliary and ureteral colic, and adynamic ileus, as well as painful visceral conditions caused by malignancy. Sympathetic blockade at the appropriate segmental level also has been prescribed in cases of acute herpes zoster and postherpetic neuralgia.

Cervicothoracic sympathetic block is also referred to as "stellate ganglion block;" and it usually is performed by an experienced anesthesiologist for the indications previously outlined. Using the technique described by Brown, the patient is placed in supine position with the neck in slight extension. The operator then identifies the sixth cervical vertebral tubercle by locating the cricoid cartilage and moving the fingers laterally until they reach this easily palpable structure. The anesthesiologist then places the index and third fingers between the carotid artery laterally and the trachea medially at the level of C6. A short 22- or 25-gauge needle is inserted until it contacts the transverse process of C6. The needle is then withdrawn approximately 1-2 mm and 5-10 mL of LA injected. Care must be taken not to perform intravascular injection or LA blockade of the recurrent laryngeal and phrenic nerves.

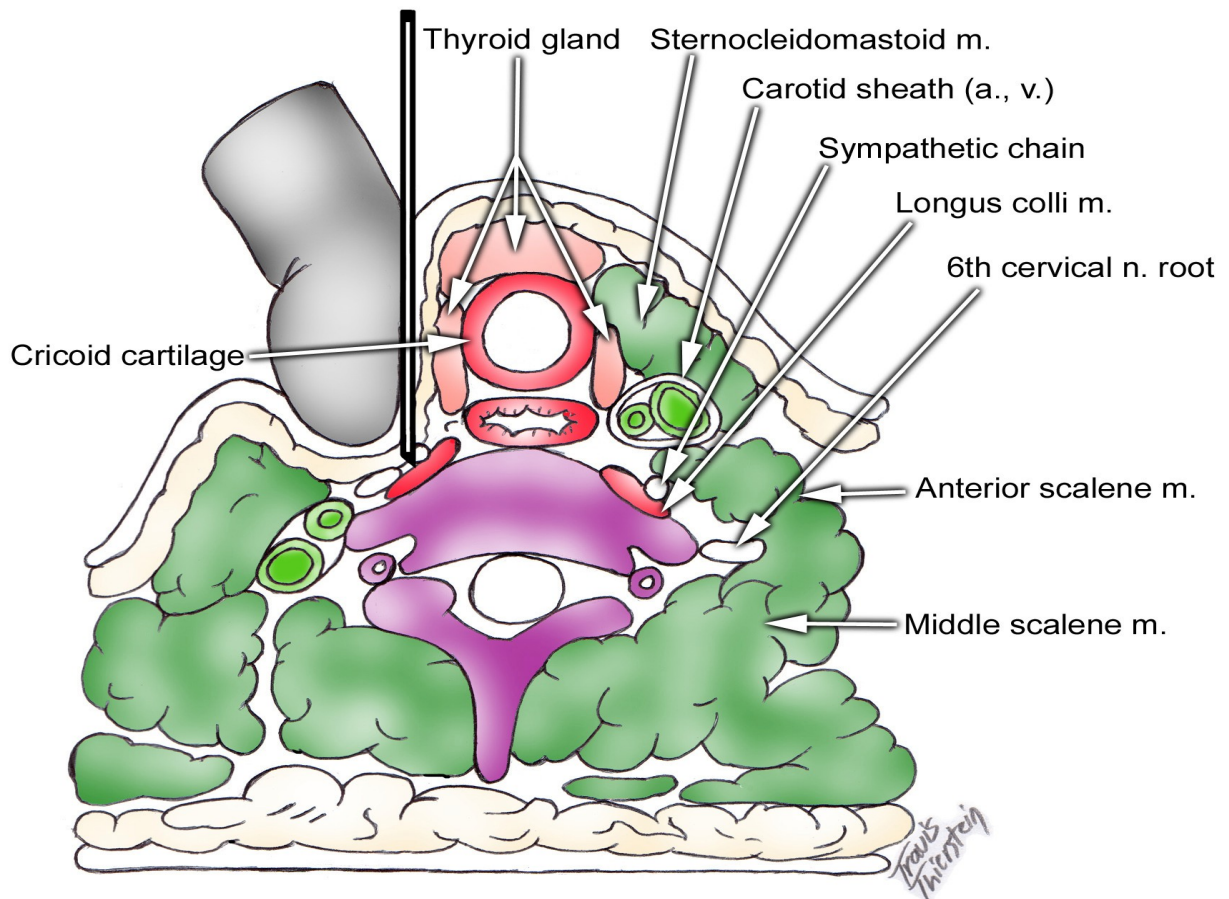


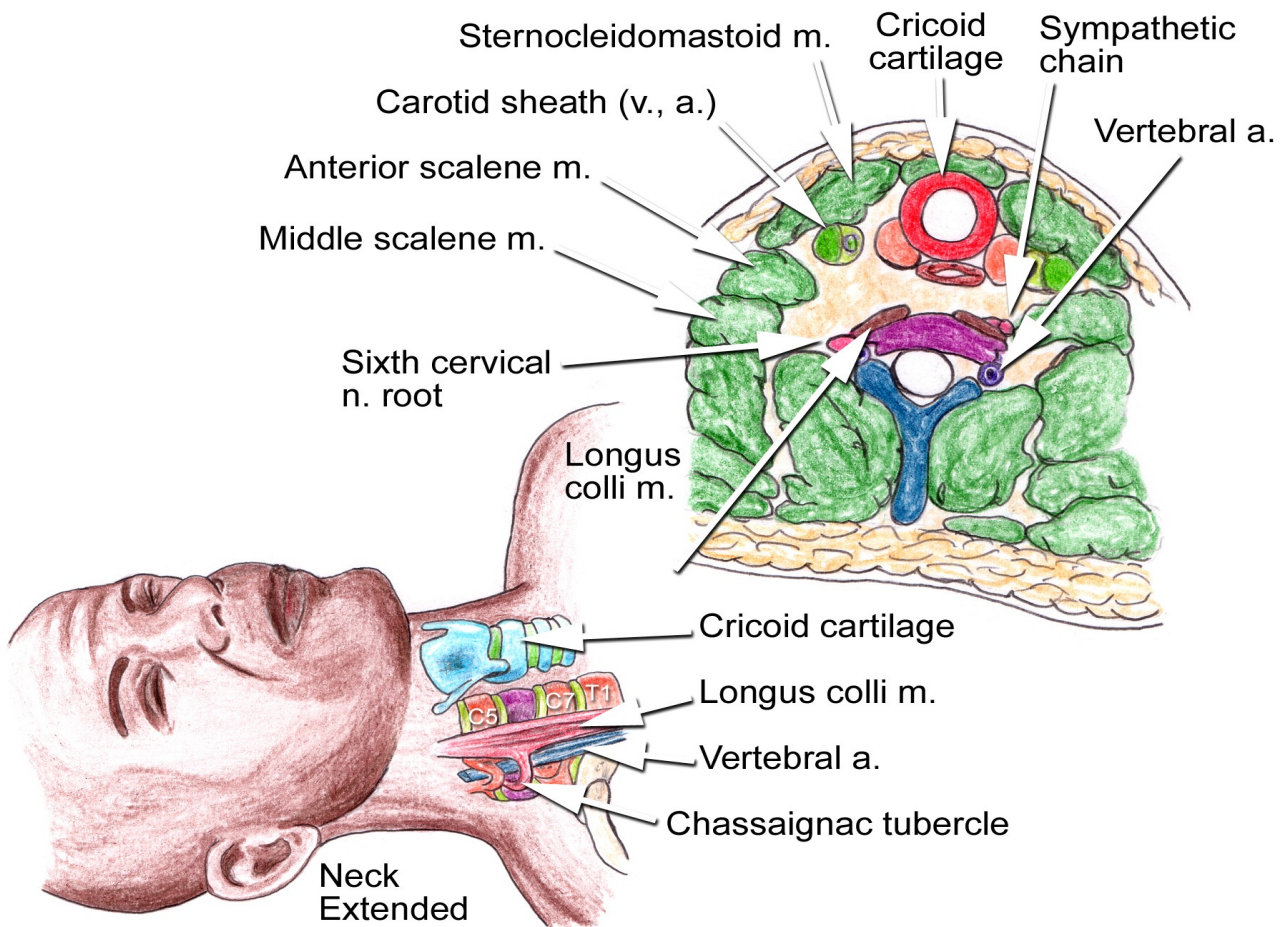
Schematic anatomical representations, sympathetic chain and stellate ganglion.



Cervical and superior thoracic anatomy (anterolateral view).

Stellate block, important anatomical landmarks (surface and cross-sectional views).

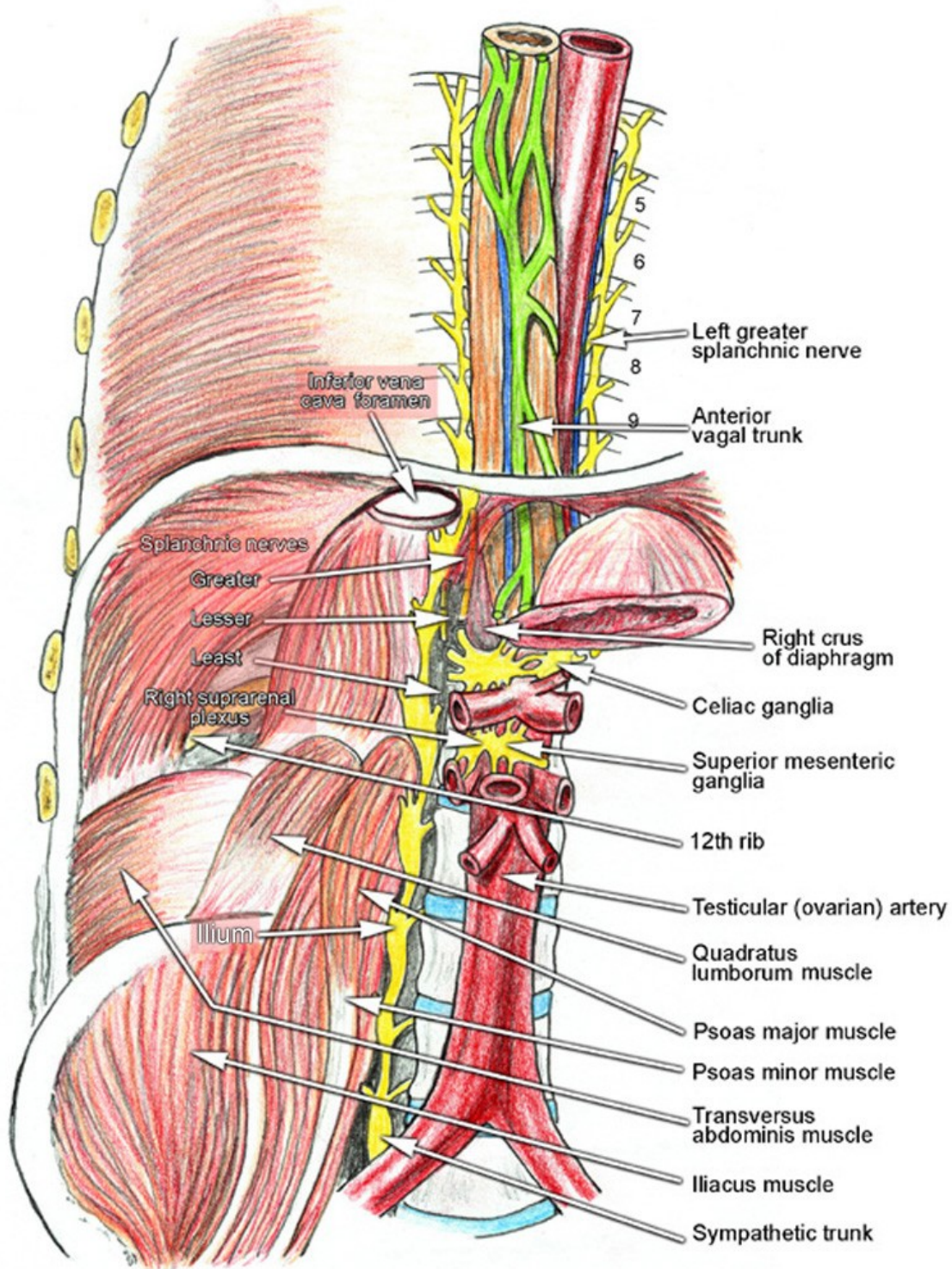




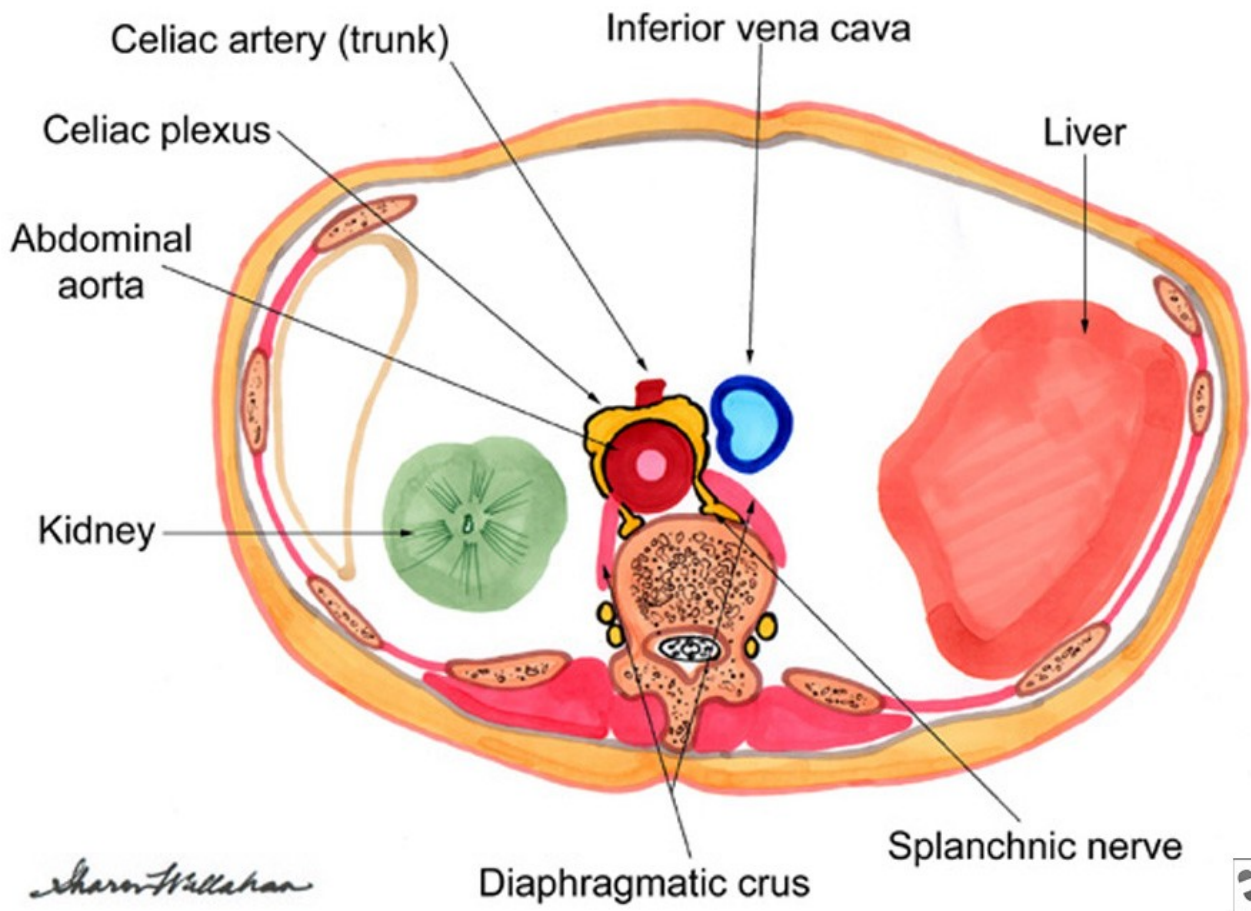
Stellate block technique and anatomy. See text for details.

Blockade of the thoracic sympathetic chain is a useful diagnostic and therapeutic procedure for identifying segmental nociceptive pathways, which may be causing pain due to inflammatory, infectious (herpes zoster), or structural pathology. Celiac plexus block should be performed by a skilled anesthesiologist for relieving severe pain caused by an acute visceral disease. Using the technique described by Brown, the patient is placed in prone position over a pillow placed beneath the abdomen to reduce lumbar lordosis.

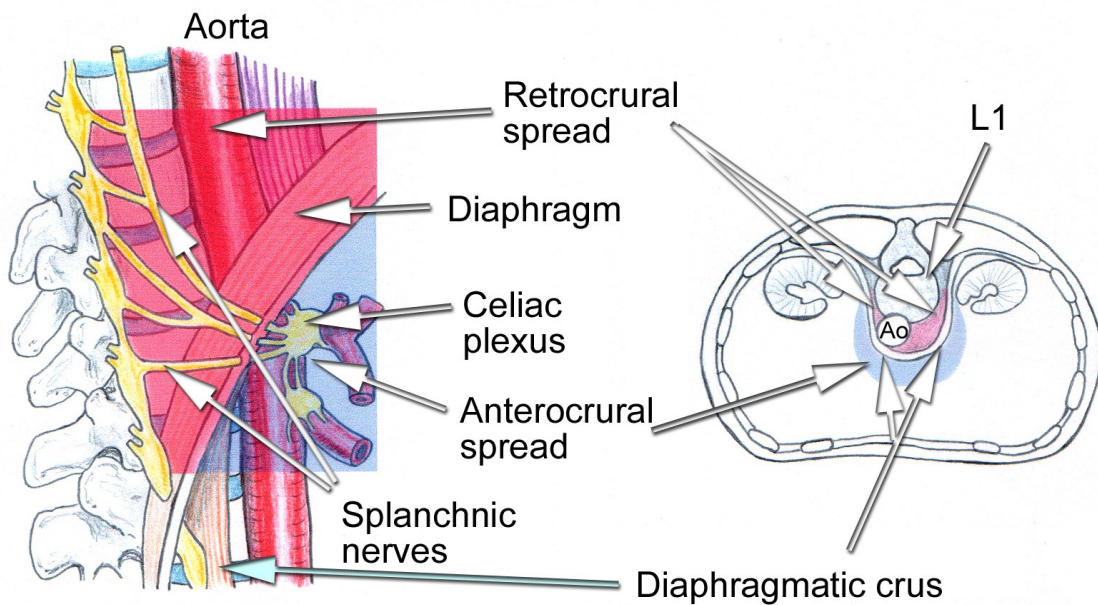
The lumbar and twelfth thoracic vertebral spines are identified and marked, and parallel lines to the vertical axis of the spine are drawn 7-8 cm from the axial midline. Then the tip of the twelfth rib is palpated and marked. Another mark is placed in the midline between the twelfth thoracic and first lumbar vertebral spines. Connecting lines between these 3 marks produce a flat isosceles triangle. Skin wheals are placed over the marks immediately below the twelfth rib, and a 12-15 cm, 20-gauge needle (without the syringe) is inserted.



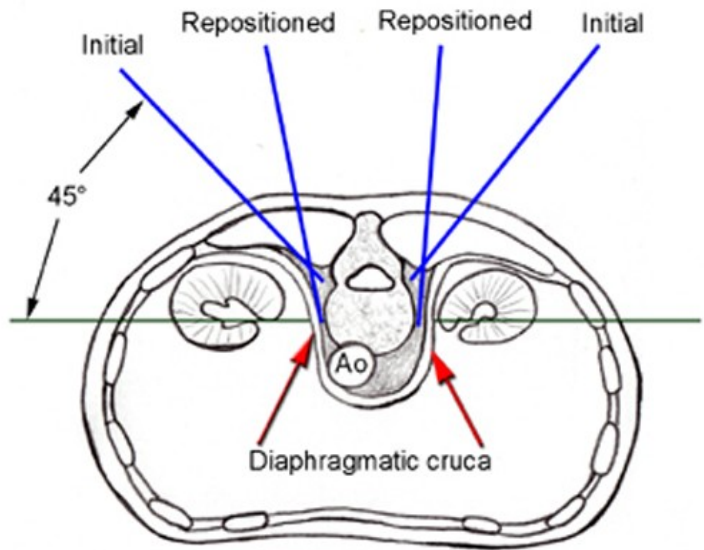
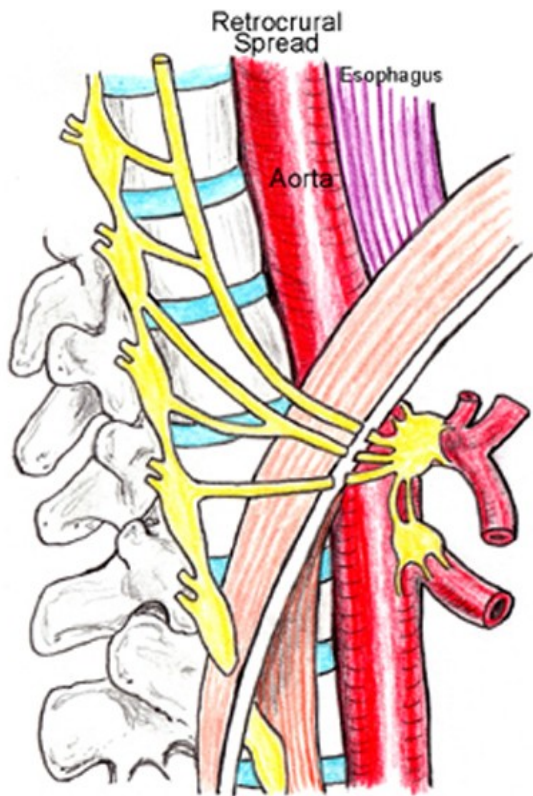
Pertinent anatomy for celiac plexus block.



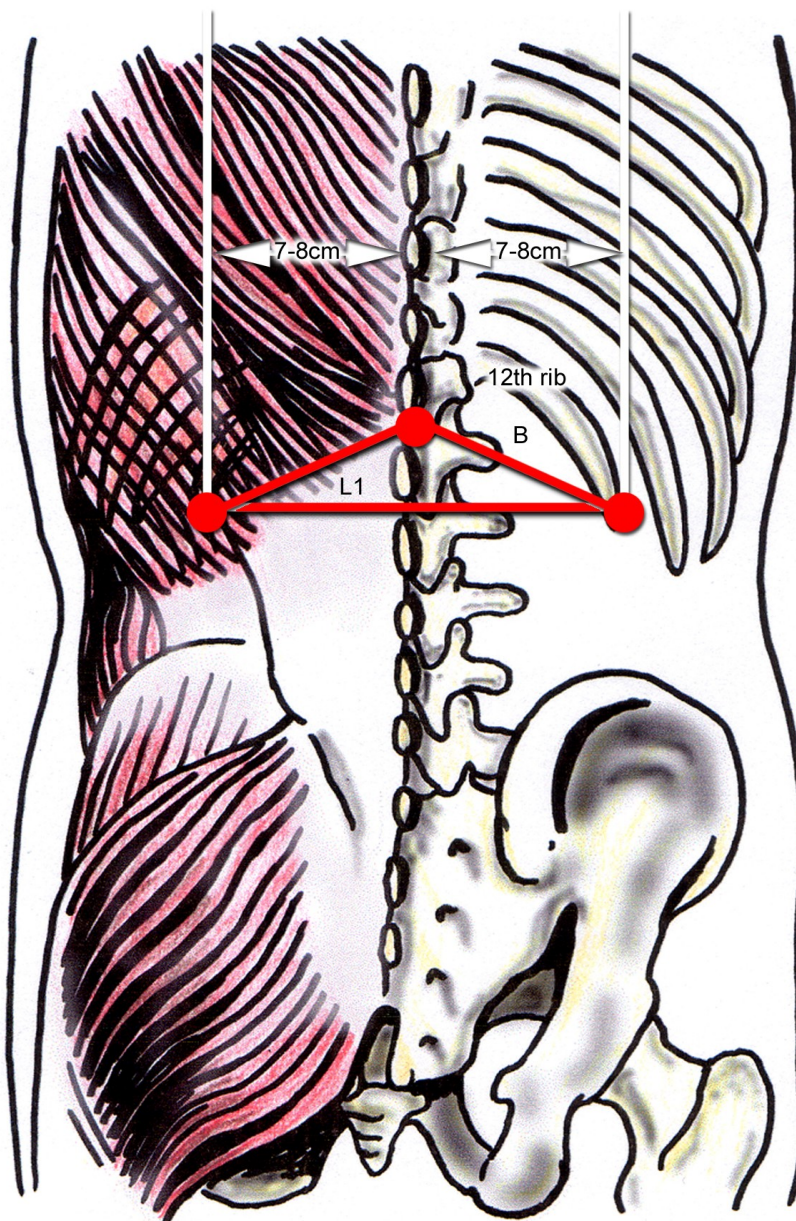
Pertinent anatomy for celiac plexus block (cross-sectional view).



Retrocrural and anterocrural relationships (celiac plexus block).



Celiac plexus block, retrocrural (deep splanchnic) technique. See text for details.



Surface anatomy and markings for celiac plexus block.

The needle is inserted between the T12 and L1 vertebral spines in a plane that is 45° to the horizontal table. This placement allows contact with the L1 vertebral body at a depth of 7-9 cm. More superficial bony contact is usually caused by needle impingement upon a vertebral transverse process. C-arm fluoroscopy is helpful for guiding the direction and depth of the needle. After the vertebral body is identified clearly, the needle is withdrawn to subcutaneous level and the angle changed to allow the tip to slip past the lateral border of the vertebral body.

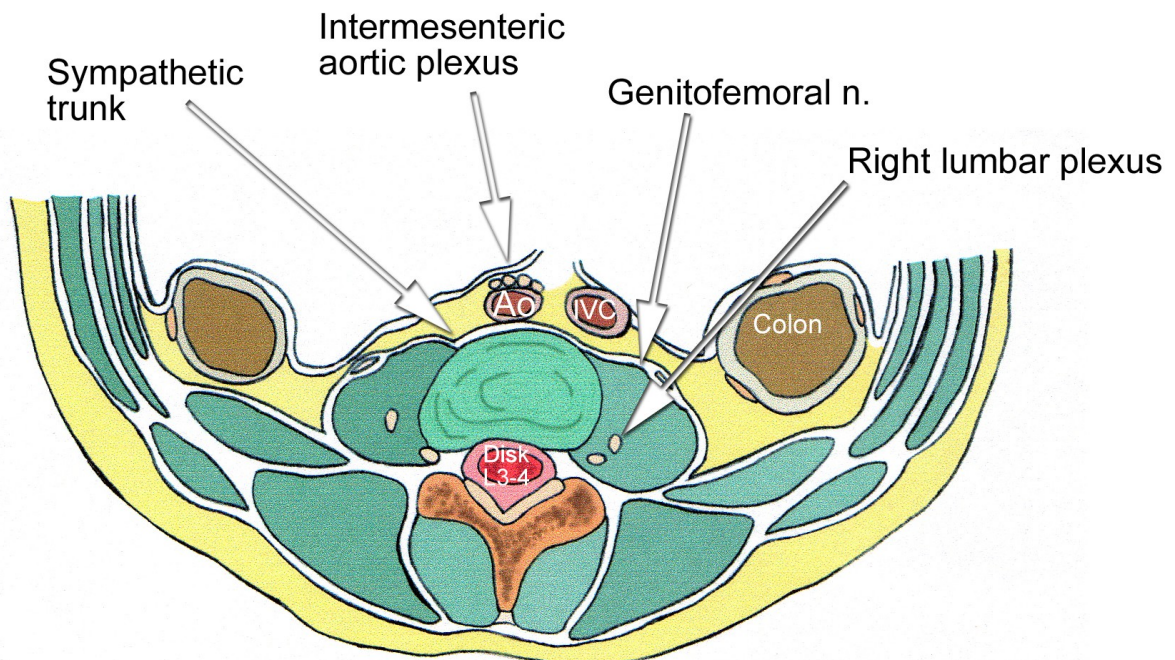
After the needle tip passes by the vertebral body, it should be inserted an additional 1.5-2 cm or until it approaches the aortic wall, which can be recognized by transmission of pulsations from this vascular structure through the needle.

On the right, the needle insertion can be placed deeper, approximately 2-3 cm beyond the vertebral body. Aspiration after needle placement is critical prior to the injection of LA or a neurolytic agent. Besides blood, faulty needle puncture may yield urine or CSF.

Lumbar sympathetic blockade also should be performed by an experienced anesthesiologist, preferably using C-arm fluoroscopic guidance. Using the technique described by Brown, the patient is placed in prone position. The second or third lumbar vertebral spines are identified, and a mark is placed on the skin 7-9 cm lateral to the midline. A skin wheal is raised using a 15-cm, 20- or 22-gauge needle, which then is inserted through the skin at an angle of 30-45° from the vertical plane ascribed to the patient's midline. The needle is advanced until it contacts the lateral aspect of the L2 vertebral body.

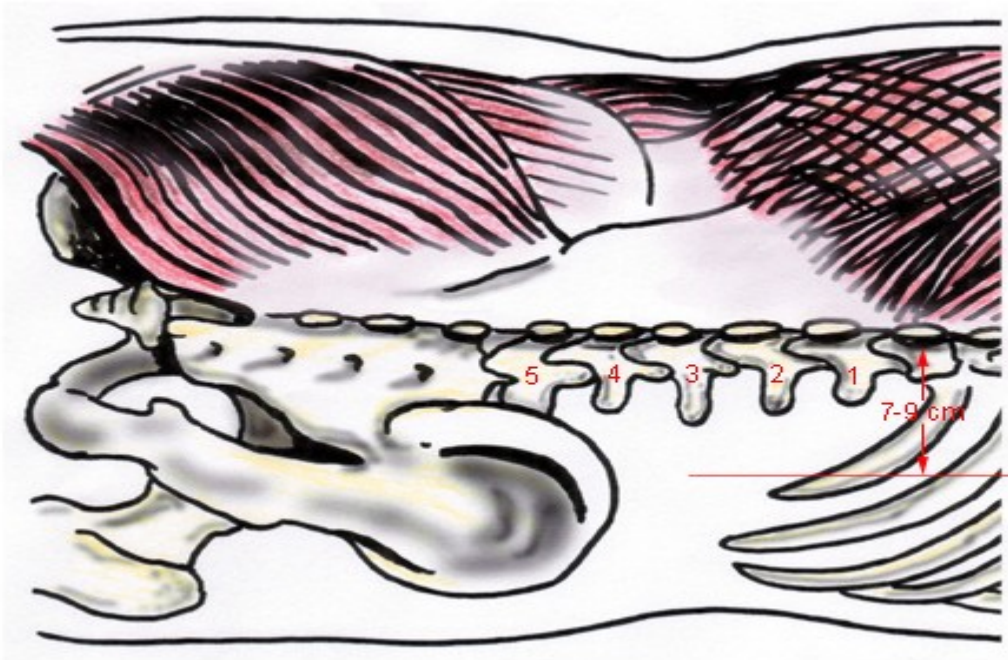
Superficial contact usually is caused by encroachment upon the transverse process. The needle is repositioned and redirected in a cephalad or caudal manner to avoid the transverse process. The target position for the needle is the anterolateral surface of L2. When the needle is in position, and after aspiration, 15-20 mL of LA solution, usually 0.5% lidocaine or 0.125-0.25% bupivacaine, is injected.

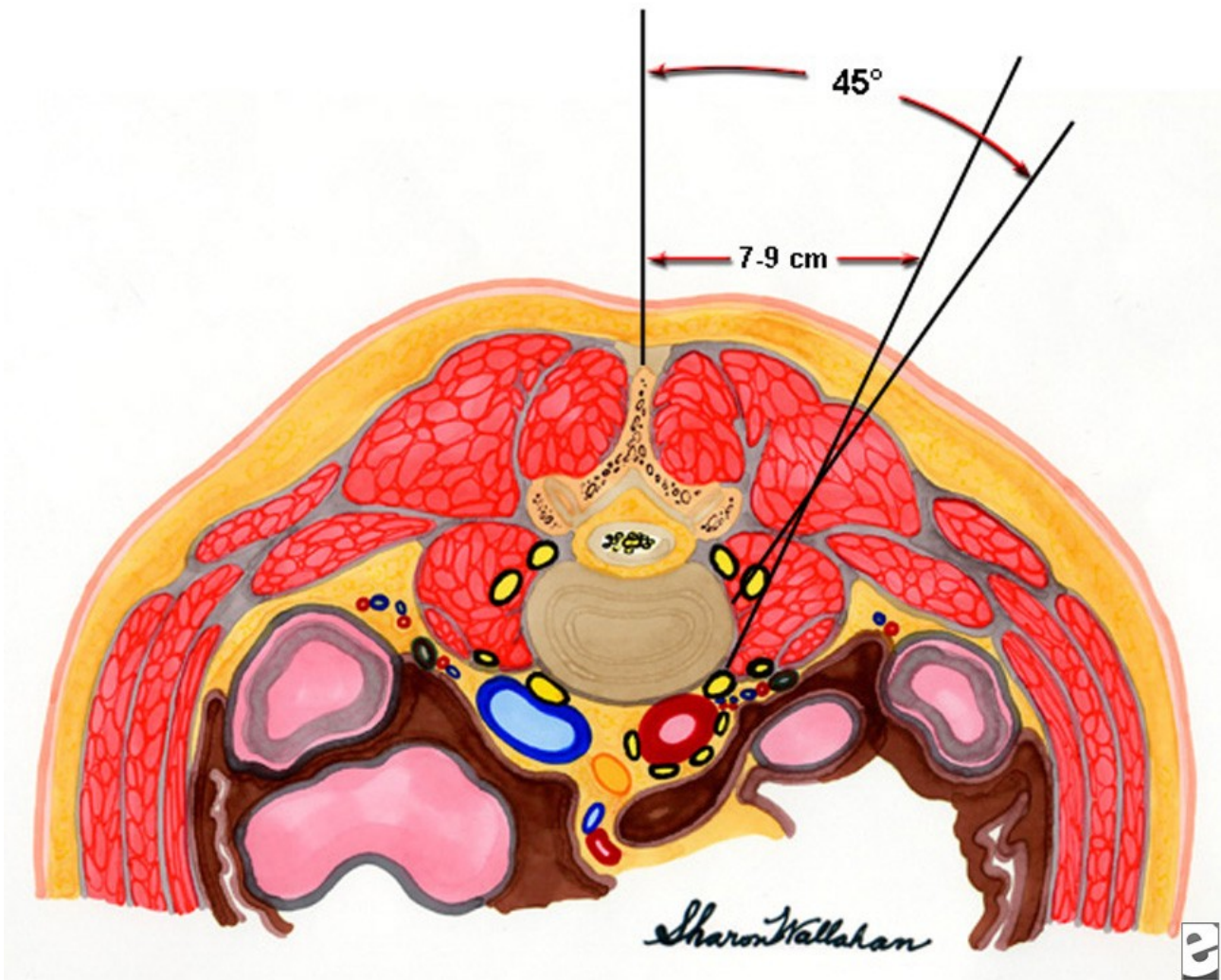
Complications are rare but can occur, including accidental injection into the inferior vena cava on the right or the aorta on the left, damage to lumbar vessels, and unintentional needle penetration or anesthesia to neighboring somatic nerves. Sympathetic nervous system monitoring (which has not been discussed in detail) determines the presence and extent of sympathetic blockade.



Pertinent anatomy for lumbar sympathetic block (cross-sectional view).

Surface technique of lumbar sympathetic block. See text for details.





Lumbar sympathetic block, cross-sectional technique. See text for details.

Intravenous regional sympathetic blockade entails injection of an antiadrenergic agent into the venous system of a limb with CRPS after the circulation is occluded temporarily with a tourniquet. An experienced interventionist, preferably an anesthesiologist, should perform this procedure. This procedure was originally developed using guanethidine, which can induce a prolonged, unselective sympathetic blockade by displacing NE from presynaptic vesicles and preventing NE uptake.

Guanethidine causes an initial release of NE, followed by NE depletion, which results in long-lasting interruption of adrenergic activity. Blockade may last for hours, days, and occasionally, weeks because of the high affinity of guanethidine for binding to sympathetic nerve endings, and also because guanethidine is eliminated slowly. Unfortunately, parenteral guanethidine is no longer available, since the drug is no longer used for the treatment of hypertension by the IV route.

Other possible candidates for alpha-adrenergic blockade include reserpine, which causes NE storage vesicle depletion and blocks NE reuptake; however, this drug has proved relatively ineffective and produces many adverse effects. Blockade of presynaptic (alpha2) and postsynaptic (alpha1) receptors can be performed with phentolamine, which is reversible, usually with duration of effect of less than 24 hours.

Blockade of postsynaptic (alpha1) receptors can be induced by prazosin; however, a parenteral form of this drug has not yet been approved, and investigation for this indication is insufficient to date. IV sympathetic blockade is particularly useful for patients in need of sympathetic blockade who are taking anticoagulant medications. Patients who are sensitive or experience excessive toxic systemic reactions to LA may be candidates for IV blockade.

Problems with the use of guanethidine include the initial effect of the drug, which causes an increase in NE and consequent cutaneous vasoconstriction, piloerection, and burning dysesthesia. Some of the guanethidine that escapes

occlusion of the circulation by tourniquet may produce side effects including tachycardia and dizziness, as well as other signs and symptoms of sympathetic blockade. Cardiac, blood pressure, and other vital signs should be monitored closely, and appropriate resuscitative measures and equipment should be available at the bedside.

Patient anxiety may increase with the transient rise in NE levels secondary to the guanethidine effect. Prior to the procedure, 100 mg of thiopental, 5-10 mg of IV diazepam, or 3-5 mg of IV morphine should be considered to minimize the pain and discomfort inherent to the procedure. Pain resolution associated with regional infusion of guanethidine can be considered sympathetically mediated from a diagnostic standpoint. Repeating this procedure on an outpatient basis may be necessary; patients with severe CRPS may require re-treatment every 3 days, whereas patients with milder CRPS may require therapeutic intervention only at 3-week intervals. Usually, treatment is limited to 2-3 sessions.

IV neural blockade is performed using a technique similar to that described for IV sympathetic blockade. The patient is prepared for IV infusion in the affected limb. The limb is elevated, and the tourniquet is inflated to a pressure above the patient's systolic blood pressure. Using a 50-mL disposable syringe, 30-40 mL of 0.5% lidocaine or procaine, without epinephrine or other vasoconstrictors, is injected slowly. The skin often becomes mottled as the injection proceeds, and analgesia develops rapidly. Adequate analgesia often develops within 5-10 minutes.

Tourniquet discomfort may require adjunctive systemic injections of sedatives or narcotics; however, concomitant opioid use can confound diagnostic interpretations. Usual indications for this procedure include CRPS syndromes, neuralgia, and deafferentation pain. IV lidocaine in doses of 1-1.5 mg per kilogram of body weight, or in a 0.1% solution administered over 35 minutes to 200 mg, are alternative technical approaches for this procedure.

Successful neural blockade by continuous infusion may allow the pain medicine specialist to consider oral lidocainelike derivatives such as mexiletine. To this point, neural blockade and therapeutic procedures that have been described entail the use of LAs. However, LA blockade of certain painful conditions may lead treating practitioners to consider a neurolytic agent for more permanent pain relief.

Neurolytic blockade is an important tool because it offers the potential for long-term relief from severe pain caused by conditions such as advanced cancer, certain neuralgias, and incurable conditions such as occlusive vascular disease. Neurolytic cranial nerve blocks, subarachnoid block, celiac plexus block, and lumbar sympathetic block, when properly executed, result in a high degree of success with acceptably low instances of adverse effects in patients who have not obtained satisfactory relief by other methods.

Practitioners with extensive experience, skill, and knowledge of the pharmacology and application of neurolytic agents should perform these procedures. Informed consent including potential outcomes and adverse effects should be expressed clearly through practitioner-patient communication.

Examples of neurolytic agents include alcohol in concentrations of 35-100%. Alcohol produces nerve fiber destruction, which results in wallerian axonal degeneration. If cell bodies at the level of the dorsal root ganglia are destroyed, no regeneration takes place, whereas if they are destroyed only partially, regeneration may occur.

Phenol often is used to induce prolonged sympathetic, somatic nerve, subarachnoid, and epidural blockade. Phenol is similar to alcohol in regards to its potency and nonselective damage to the nervous system. Injection concentrations of phenol usually vary between 5% and 8%. Concentrations above 5%, when applied to peripheral nerve, cause protein coagulation and necrosis with axonal degeneration and subsequent wallerian degeneration.

Injection of glycerol into the trigeminal ganglion has been popularized for the treatment of neuralgia because of its capacity to relieve pain without causing significant sensory deficits. Topical application of a 50% glycerol solution to nerves causes localized subperineurial damage, whereas intraneural injection is more damaging and causes axonolysis.

Cryotherapy, laser, and radiofrequency lesions are currently under investigation and are advocated as being effective for neurolytic procedures when performed by trained and experienced interventionists. Further clinical research is needed to develop methods that preferentially block nociceptive pathways (ie, strict neurolytic blockade that spares large myelinated sensory fibers).

Neuraxial neurolytic blocks are advocated to alleviate severe intractable pain caused primarily by advanced terminal cancer. The use of these techniques for chronic, nonmalignant pain should be discouraged. Agents used for this purpose include ethyl alcohol, phenol in glycerin, chlorocresol in glycerin, aqueous phenol, hypertonic saline solution, and ammonium compounds.

Subarachnoid neurolytic block is used to relieve severe pain resulting from continuous nociceptive impulses from skin, subcutaneous tissue, deep somatic structures, and viscera. Neurolytic agents are aimed by positioning the patient depending on whether the destructive agent is hyperbaric or hypobaric, so that the axons of the posterior rootlets are destroyed upon contact, thereby affecting neural input from the dorsal root ganglion to the spinal cord.

Subarachnoid neurolysis also can be used effectively for managing patients with spasticity. Intrathecal neurolysis is not

associated with significant pain and causes few serious complications; therefore, it can be performed on patients who are in poor physical condition and on elderly patients. Only a brief hospital stay may be necessary; therefore, it is more available to patients than other techniques because it requires no special costly or highly sophisticated equipment or facilities. Neurolytic injections can be repeated or extended if pain spreads or persists. Subarachnoid neurolytic block can delay or avoid neurosurgical procedures, and the duration of pain relief is usually sufficient to afford adequate comfort for patients with terminal cancer.

Intrathecal neurolysis for the management of cancer pain is not, however, devoid of problems and disadvantages. Inadequate pain relief may result from failure of the injection to interrupt all nociceptive pathways completely or from spread of the pain beyond the anesthetized region following the injection. Although the block initially may interrupt nerves to the painful region and afford relief of the pain, aggressive neoplasms often spread beyond the confines of induced analgesia to cause additional symptoms. Fortunately, the block can be repeated several times without further taxing the patient's already overburdened physiological status.

Complications may occur during or following the procedure, such as muscle weakness affecting the limbs or rectal and bladder sphincters. Contraindications for subarachnoid neurolysis include pain that is diffuse or poorly localized; tumor infiltration with involvement of the spinal cord or vertebral column at the level of injection; and inadequate pain relief despite repeated LA test blocks. Epidural and subdural neurolytic blockade also can be used with similar techniques as mentioned above and for similar indications.

Chemodenervation with botulinum toxin

Musculoskeletal pain often is attributed to myofascial pain syndrome (MPS). Of patients with pain presenting to a variety of specialists, the prevalence of MPS has been reported to vary from 30-90% depending on the subspecialty practice and setting. MPS is characterized by painful muscles with increased tone and stiffness containing trigger points, which are tender, firm nodules, or taut bands, usually 3-6 mm in diameter. Palpation produces aching pain in localized reference zones.

Mechanical stimulation of the taut band by needling or brisk transverse pressure produces a localized muscle twitch. Trigger point palpation often elicits a "jump sign"—an involuntary reflexlike recoil or flinching of the pain—that is disproportionate to the pressure applied. Multiple treatments including trigger point injections have long been advocated; however, reports conflict as to whether any therapeutic substance injected into a muscle provides more benefit than dry needling alone.

The pathogenesis of myofascial trigger points is unknown; however, Simons postulates that abnormally increased motor endplate activity caused by excessive release of acetylcholine at the neuromuscular junction results in spontaneous electrical activity and extrafusal muscle contraction in the immediate vicinity of the extrafusal muscle end plates, thus forming the taut band and trigger point.

Botulinum toxin is a potent neurotoxin with 7 serotypes that is produced by the bacterium *Clostridium botulinum*; it acts by blocking acetylcholine release at neuromuscular junctions. Botulinum toxin has been under investigation since 1968 and is used widely by practitioners for the treatment of focal muscle overactivity present for more than 15 years. Countless studies have demonstrated this neurotoxin to be safe and efficacious for the treatment of a large number of clinical syndromes associated with undesirable muscle contraction, spasm, or overactivity. The therapeutic benefit from botulinum toxin A injected into humans generally lasts 3-4 months, though responses may range from 2-6 months or more.

Botulinum toxin has been approved by the US Food and Drug Administration (FDA) for the treatment of strabismus, blepharospasm, and disorders of CN VII in patients aged 12 years or older; however, clinical investigation supports the treatment of many focal dystonic and nondystonic disorders of muscle spasm for which the agent has not yet received FDA approval. This neurotoxin also has been shown to be an effective treatment for pain caused by conditions of muscle overactivity.

Patients with cervical dystonia frequently complain of pain. Multiple studies have demonstrated that botulinum toxin is clinically effective in reducing painful muscle spasm and the abnormal head posture of cervical dystonia, as well as eliciting a dramatic reduction in the degree of pain, which is appreciated throughout the duration of the neurotoxin's expected effect.

Botulinum toxin also has been demonstrated to reduce the pain that results from muscle spasticity, and more recently botulinum toxin has been applied to treat more common painful disorders such as headache, neck pain, and back pain. The beneficial effect attributed to botulinum toxin is thought to be due to its capacity to reduce painful muscle contracture or spasm; therefore, research is underway to determine whether it can be used effectively as an intervention for MPS, which has been studied extensively throughout the literature but remains controversial and poorly understood.

Future directions

Solutions to pain disorders will stem from continued human and animal studies, which further define the biochemical and neurophysiological factors that influence these disorders. Of all medical specialists, neurologists may be best suited to analyze the complex physical and nonphysical components of chronic pain. Biochemical solutions in the chronic illness model also may make the neurologist best suited to adapt new treatments; however, further training in psychology and human behavior may be required in neurology residency programs if the specialty is to establish a prominent role in the management of these disorders. The future of pain management and research resides in multidisciplinary departments dedicated to the cross-section of knowledge and treatment disciplines required to manage pain disorders. As in all cases of human disease, the treatment of pain disorders will become more effective on the basis of results of future clinical research and vigorous outcome studies.

REFERENCES

- Selander D, Dhuner KG, Lundborg G. Peripheral nerve injury due to injection needles used for regional anesthesia. An experimental study of the acute effects of needle point trauma. *Acta Anaesthesiol Scand.* 1977;21(3):182-8. [Medline].
- Czervionke LF, Fenton DS. Basic needle manipulation techniques. In: Czervionke LF, Fenton DS, eds. *Image-Guided Spine Intervention.* 1-8. Philadelphia: Saunders; 2003.
- Mayo NA, Fadale PD. The role of injectable corticosteroids in orthopedics. *Orthop.* 2001;24:400-5.
- Sitzman BT. Pharmacology for the spine injectionist. In: Fenton DS, Czervionke LF, eds. *Image-guided spine intervention.* Philadelphia: Saunders; 2003:287-92.
- Schimmer BP, Parker KL. Adrenocortical steroids. In: Hardman JG, Gilman AG, Limbird LE. *Goodman and Gilman's The pharmacological basis of therapeutics.* 9th ed. New York: McGraw-Hill; 1996:1465-81.
- Wiggins ME, Fadale PD, Barrach H, Ehrlich MG, Walsh WR. Healing characteristics of a type I collagenous structure treated with corticosteroids. *Am J Sports Med.* Mar-Apr 1994;22(2):279-88. [Medline].
- Cohen IK, Diegelmann RF, Johnson ML. Effect of corticosteroids on collagen synthesis. *Surgery.* Jul 1977;15-20:82(1). [Medline].
- Bogduk N. Complications associated with transforaminal injections. In: Neal JM, Rathmell JP. *Complications in Regional Anesthesia and Pain Medicine.* Philadelphia, Pa: Saunders; 2007:259-265.
- Dreyer SJ. Commonly used medications in pain procedures. In: Lennard JA, ed. *Pain procedures in clinical practice.* Philadelphia, Pa: Hanley & Belfus; 2000:1-9.
- Manchikanti L, Staats PS, Singh V, Schultz DM, Vilims BD, Jasper JF, et al. Evidence-based practice guidelines for interventional techniques in the management of chronic spinal pain. *Pain Physician.* Jan 2003;6(1):3-81. [Medline].
- Benzon HT. *Fluoroscopy and Radiation Safety, Essentials of Pain Medicine and Regional Anesthesia.* Philadelphia: Elsevier Churchill Livingstone; 2005:516-24.
- Machikanti L, Bakhit CE, Fellowes B. Fluoroscopy is medically necessary for the performance of epidural steroids. *Anesthes Analg.* 1999;89:1330-31.
- Fishman SM, Smith H, Meleger A, Seibert JA. Radiation safety in pain medicine. *Reg Anesth Pain Med.* May-Jun 2002;27(3):296-305. [Medline].
- Broadman L. Radiation Safety. In: Raj PP, Abram BM, Benzin HT. *Practical Management of Pain.* 2. St Louis: Mosby; 2000:834-837.
- Gruber RD, Botwin KP, Chunila SP. Radiation safety for the physician. In: Leonard TA, ed. *Pain Procedures in Clinical Practice.* 2nd ed. Hanley & Belfast; 2000:25-32.
- Acquadro MA, Borodic GE. Treatment of myofascial pain with botulinum A toxin. *Anesthesiology.* Mar 1994;80(3):705-6. [Medline].
- Applegate WV. Abdominal cutaneous nerve entrapment syndrome. *Surgery.* Jan 1972;71(1):118-24. [Medline].
- Aprill C, Bogduk N. The prevalence of cervical zygapophyseal joint pain. A first approximation. *Spine.* Jul 1992;17(7):744-7. [Medline].
- Aprill C, Dwyer A, Bogduk N. Cervical zygapophyseal joint pain patterns. II: A clinical evaluation. *Spine.* Jun 1990;15(6):458-61. [Medline].

- Auld AW, Maki-Jokela A, Murdoch DM. Intraspinal narcotic analgesia in the treatment of chronic pain. *Spine*. Oct 1985;10(8):777-81. [Medline].
- Badgley CE. The articular facets in relation to low back pain and sciatic radiation. *Br J Bone Joint Surg*. 1941;23:481-496.
- Beliveau P. A comparison between epidural anaesthesia with and without corticosteroid in the treatment of sciatica. *Rheumatol Phys Med*. Feb 1971;11(1):40-3. [Medline].
- Berry FR. Analgesia in patients with fractured shaft of femur. *Anaesthesia*. Jun 1977;32(6):576-7. [Medline].
- Bogduk N. Zygapophysial blocks and epidural steroids. In: *Neural Blockade in Clinical Anaesthesia and Management of Pain*. 1988:935.
- Bogduk N, Aprill C, Derby R. Diagnostic blocks of spinal synovial joints. In: *Spine Care: Diagnosis and Conservative Treatment*. 1995:298-321.
- Bogduk N, Aprill C, Derby R. Epidural steroid injections. In: *Spine Care: Diagnosis and Conservative Treatment*. 1995:332-42.
- Bogduk N, Christophidis N, Cherry D. Epidural use of steroids in the management of back pain and sciatica of spinal origin. Canberra Australia. Report of the Working Party on Epidural Use of Steroids in the Management of Back. 1993.
- Bogduk N, Long DM. Percutaneous lumbar medial branch neurotomy: a modification of facet denervation. *Spine*. Mar-Apr 1980;5(2):193-200. [Medline].
- Bogduk N, Marsland. On the concept of third occipital headache. *J Neurol Neurosurg Psychiatry*. Jul 1986;49(7):775-80. [Medline].
- Bogduk N, Twomey LT. *Clinical anatomy of the lumbar spine*. 1991.
- Bogduk N, Valencia F. Innervation and pain patterns of the thoracic spine. In: *Physical Therapy of the Neck and Thoracic Spine*. 1998:27.
- Bohr T. Problems with myofascial pain syndrome and fibromyalgia syndrome. *Neurology*. Mar 1996;46(3):593-7. [Medline].
- Bonica J, Buckley C, Monica G, Murphy TM. Neurolytic blockade and hypophysectomy. In: Bonica JJ, ed. *The Management of Pain*. Philadelphia: Lea Febiger; 1990:1980-2039.
- Bonica JJ. Causalgia and other reflex sympathetic dystrophies. In: Bonica JJ, ed. *The Management of Pain*. Philadelphia: Lea & Febiger; 1990:220-43.
- Bonica TT, Buckley FO. Regional anesthesia with local anesthetics. In: *The Management of Pain*. 1990:1883-1966.
- Brands E, Callanan VI. Continuous lumbar plexus block--analgesia for femoral neck fractures. *Anaesth Intensive Care*. Aug 1978;(3):256-8. [Medline].
- Brevik H, Hesla PE, Molnar I, et al. Treatment of chronic low back pain and sciatica: comparison of caudal epidural injections of bupivacaine and methylprednisolone with bupivacaine followed by saline. In: Bonica JJ, Albe-Fessard DL. *Advances in Pain Research and Therapy*. 1. 1976:927.
- Bridenbaugh PO, DuPen SL, Moore DC, et al. Postoperative intercostal nerve block analgesia versus narcotic analgesia. *Anesth Analg*. Jan-Feb 1973;52(1):81-5. [Medline].
- Brown DL. *Atlas of Regional Anesthesia*. 1992.
- Buckley FP, Duval Neto G, Fink BR. Acid and alkaline solutions of local anesthetics: duration of nerve block and tissue pH. *Anesth Analg*. May 1985;64(5):477-82. [Medline].
- Burn JM, Langdon L. Duration of action of epidural methyl prednisolone. A study in patients with the lumbosciatic syndrome. *Am J Phys Med*. Feb 1974;53(1):29-34. [Medline].
- Bush K, Hillier S. A controlled study of caudal epidural injections of triamcinolone plus procaine for the management of intractable sciatica. *Spine*. May 1991;16(5):572-5. [Medline].

Carette S, Marcoux S, Truchon R. A controlled trial of corticosteroid injections into facet joints for chronic low back pain. *N Engl J Med.* Oct 3 1991;325(14):1002-7. [Medline].

Carrera GF. Lumbar facet joint injection in low back pain and sciatica: preliminary results. *Radiology.* Dec 1980;137(3):665-7. [Medline].

Catchlove RF, Braha R. The use of cervical epidural nerve blocks in the management of chronic head and neck pain. *Can Anaesth Soc J.* Mar 1984;31(2):188-91. [Medline].

Chan ST, Leung S. Spinal epidural abscess following steroid injection for sciatica. Case report. *Spine.* Jan 1989;14(1):106-8. [Medline].

Chutorian A, Root L. Management of spasticity in with botulinum-A toxin. *Int Pediatr.* 1994;9:1:35-43.

Cicala RS, Westbrook L, Angel JJ. Side effects and complications of cervical epidural steroid injections. *J Pain Symptom Manage.* Jun 1989;4(2):64-6. [Medline].

Colding A. The effect of regional sympathetic blocks in the treatment of herpes zoster. *Acta Anaesthesiol.* 1969;13:133.

Covino B. Neural blockade in clinical anesthesia and management of pain. In: *Clinical Pharmacology of Local Anesthetic Agents.* 2nd ed. 1988:111-144.

Crawford ED, Skinner DG. Intercostal nerve block with thoracoabdominal and flank incisions. *Urology.* Jan 1982;19(1):25-8. [Medline].

Cuckler JM, Bernini PA, Wiesel SW, et al. The use of epidural steroids in the treatment of lumbar radicular pain. A prospective, randomized, double-blind study. *J Bone Joint Surg Am.* Jan 1985;67(1):63-6. [Medline].

Czarski Z. [Treatment of sciatica with hydrocortisone and novocaine injections into the sacral hiatus]. *Przegl Lek.* 1965;21(7):511-3. [Medline].

Dan K, Higa K, Tanaka K. Herpetic pain and cellular immunity. *Current Topics in Pain Research and Therapy.* 1983;293-305.

Devor M, Govrin-Lippmann R, Raber P. Corticosteroids suppress ectopic neural discharge originating in experimental neuromas. *Pain.* Jun 1985;22(2):127-37. [Medline].

Dilke TF, Burry HC, Grahame R. Extradural corticosteroid injection in management of lumbar nerve root compression. *Br Med J.* Jun 16 1973;2(867):635-7. [Medline].

Doherty M. Introduction to examination. In: *Rheumatology Examination and Injection Techniques.* 1993:1-19.

Dory MA. Arthrography of the cervical facet joints. *Radiology.* Aug 1983;148(2):379-82. [Medline].

Dougherty JH Jr, Fraser RA. Complications following intraspinal injections of steroids. Report of two cases. *J Neurosurg.* Jun 1978;48(6):1023-5. [Medline].

Drewes A, Jennum P. Epidemiology of myofascial pain, low back pain, morning stiffness and sleep related complaints in the general population. *J Musculoske Pain.* 1995;3 (supp 1):68.

Dussault RG, Nicolet VM. Cervical facet joint arthrography. *J Can Assoc Radiol.* Mar 1985;36(1):79-80. [Medline].

Dwyer A, Aprill C, Bogduk N. Cervical zygapophyseal joint pain patterns. I: A study in normal volunteers. *Spine.* Jun 1990;15(6):453-7. [Medline].

Engberg G. Single-dose intercostal nerve blocks with etidocaine for pain relief after upper abdominal surgery. Preliminary communication. *Acta Anaesthesiol Scand Suppl.* 1975;60:43-9. [Medline].

Fishbain DA, Goldberg M, Meagher BR, et al. Male and female chronic pain patients categorized by DSM-III psychiatric diagnostic criteria. *Pain.* Aug 1986;26(2):181-97. [Medline].

Fisher RH. Correspondence. *J Bone Joint Surg.* 1986;68A:789.

Fricton JR, Kroening R, Haley D, Siegert R. Myofascial pain syndrome of the head and neck: a review of clinical characteristics of 164 patients. *Oral Surg Oral Med Oral Pathol.* Dec 1985;60(6):615-23. [Medline].

Galindo A. pH-adjusted local anesthetics: clinical experience. *Reg Anesth.* 1983;8:35.

Gardner WJ, Goebert HW, Sehgal AD. Intraspinal corticosteroids in the treatment of sciatica. *Trans Am Neurol Assoc.* 1961;86:214-5. [Medline].

Garvey TA, Marks MR, Wiesel SW. A prospective, randomized, double-blind evaluation of trigger-point injection therapy for low-back pain. *Sep 1989;14(9):962-4.* [Medline].

Gerwin RD. A study of 96 subjects examined both for fibromyalgia and myofascial pain. *J Musculoskeletal Pain.* 1995;3(suppl 1):121.

Ghormley RK. Low back pain with special reference to articular facets with presentation of an operative procedure. *JAMA.* 1993;101:1773-1777.

Gloucke CR, Graziotti P. Extradural abscess following local anaesthetic and steroid injection for chronic low back pain. *Br J Anesth.* 1990;35:515.

Goebert HW, Jallo JS, Gardner WJ, et al. Painful radiculopathy treated with epidural injections of procaine and hydrocortisone acetate: results in 113 patients. *Anesth Analg.* 1961;140:130.

Goldenberg DL, Burckhardt C, Crofford L. Management of fibromyalgia syndrome. *JAMA.* Nov 17 2004;292(19):2388-95. [Medline].

Goldwait JE. The lumbosacral articulation: an explanation of many cases of lumbago, sciatica and paraplegia. *Botson Med Surg J.* 1911;164:365-372.

Hickey RF, Tregonning GD. Denervation of spinal facet joints for treatment of chronic low back pain. *N Z Med J.* Feb 9 1977;85(581):96-9. [Medline].

Hobson DE, Gladish DF. Botulinum toxin injection for cervicogenic headache. *Headache.* Apr 1997;37(4):253-5. [Medline].

Hove B, Gyldensted C. Cervical analgesic facet joint arthrography. *Neuroradiology.* 1990;32(6):456-9. [Medline].

Humphreys CF, Day M. The control of postoperative pain with the use of bupivacaine injections. *J Urol.* 1976;122:506.

Jackson RP. The facet syndrome. Myth or reality?. *Clin Orthop.* Jun 1992;(279):110-21. [Medline].

Jackson RP, Jacobs RR, Montesano PX. 1988 Volvo award in clinical sciences. Facet joint injection in low-back pain. A prospective statistical study. *Spine.* Sep 1988;13(9):966-71. [Medline].

Jacobs S, Pullan PT, Potter JM. Adrenal suppression following extradural steroids. *Anaesthesia.* Oct 1983;38(10):953-6. [Medline].

Jankovic J, Brin MF. Therapeutic uses of botulinum toxin. *N Engl J Med.* Apr 25 1991;324(17):1186-94. [Medline].

Jankovic J, Schwartz K. Botulinum toxin injections for cervical dystonia. *Neurology.* Feb 1990;40(2):277-80. [Medline].

Jankovic J, Schwartz K, Donovan DT. Botulinum toxin treatment of cranial-cervical dystonia, spasmodic dysphonia, other focal dystonias and hemifacial spasm. *J Neurol Neurosurg Psychiatry.* Aug 1990;53(8):633-9. [Medline].

Johansson A, Hao J, Sjolund B. Local corticosteroid application blocks transmission in normal nociceptive C-fibres. *Acta Anaesthesiol Scand.* Jul 1990;34(5):335-8. [Medline].

Kirkpatrick AF. The use of epidural steroids in the treatment of lumbar radicular pain [letter]. *J Bone Joint Surg [Am].* Jul 1990;72(6):948-9. [Medline].

Kirno K, Lindell K. Intercostal nerve blockade. *Br J Anaesth.* Feb 1986;58(2):246. [Medline].

Klenerman L, Greenwood R, Davenport HT. Lumbar epidural injections in the treatment of sciatica. *Br J Rheumatol.* Feb 1984;23(1):35-8. [Medline].

Knusel B, DeGryse R, Grant M, et al. Intramuscular injection of botulinum toxin type A (Botox) in chronic low back pain associated with muscle spasm [poster abstract]. San Diego, Calif. American Pain Society. Nov 1998.

Korbon GA, Rowlingson JC, Carron H. Correspondence. *J Bone Joint Surg.* 1986;68A:788.

- Lau LS, Littlejohn GO, Miller MH. Clinical evaluation of intra-articular injections for lumbar facet joint pain. *Med J Aust.* Dec 9-23 1985;143(12-13):563-5. [Medline].
- Lewinnek GE, Warfield CA. Facet joint degeneration as a cause of low back pain. *Clin Orthop.* Dec 1986;(213):216-22. [Medline].
- Lilius G, Laasonen EM, Myllynen P. Lumbar facet joint syndrome. A randomised clinical trial. *J Bone Joint Surg [Br].* Aug 1989;71(4):681-4. [Medline].
- Lindholm R, Saleinius P. Caudal, epidural administration of anaesthetics and corticosteroids in the treatment of low back pain. *Acta Orthop Scand.* 1964;1:114.
- Lippitt AB. The facet joint and its role in spine pain. Management with facet joint injections. *Spine.* Oct 1984;9(7):746-50. [Medline].
- Lofstrom B, Wennberg A, Wien L. Late disturbances in nerve function after block with local anaesthetic agents. An electroneurographic study. *Acta Anaesthesiol Scand.* 1966;10(2):111-22. [Medline].
- Lofstrom JB, Cousins MJ. Sympathetic neural blockade of upper and lower extremity. In: Cousins MJ, Bridenbaugh PO, eds. *Neural Blockade in Clinical Anesthesia and Management of Pain.* Philadelphia: 1988:461-502.
- Lord SM, Barnsley L, Wallis BJ. Percutaneous radio-frequency neurotomy for chronic cervical zygapophyseal-joint pain. *N Engl J Med.* Dec 5 1996;335(23):1721-6. [Medline].
- Lynch MC, Taylor JF. Facet joint injection for low back pain. A clinical study. *J Bone Joint Surg [Br].* Jan 1986;68(1):138-41. [Medline].
- Mandl F. Aqueous solution of phenol as a substitute for alcohol in sympathetic block. *J Int Col Surg.* 1950;13:566.
- Mathews JA, Mills SB, Jenkins VM, et al. Back pain and sciatica: controlled trials of manipulation, traction, sclerosant and epidural injections. *Br J Rheumatol.* Dec 1987;26(6):416-23. [Medline].
- McCarron RF, Wimpee MW, Hudkins PG. The inflammatory effect of nucleus pulposus. A possible element in the pathogenesis of low-back pain. *Spine.* Oct 1987;12(8):760-4. [Medline].
- Meyers RR, Katz J. Neural pathology of neurolytic and semidestructive agents. In: Cousins MJ, Bridenbaugh PO, eds. *Neural Blockade in Clinical Anesthesia and Management of Pain.* 2nd ed. Philadelphia: 1988:1031-51.
- Mooney V, Robertson J. The facet syndrome. *Clin Orthop.* Mar-Apr 1976;(115):149-56. [Medline].
- Moran R, O'Connell D, Walsh MG. The diagnostic value of facet joint injections. *Spine.* 13(12):1407-10. [Medline].
- Mount HT. Hydrocortisone in the treatment of intervertebral disc protrusion. *Can Med Assoc J.* Dec 18 1971;105(12):1279-80. [Medline].
- Mount HTR. Epidural injection of hydrocortisone in the treatment of intervertebral disc protrusion. In: *Current Controversies in Neurosurgery.* 1976:67.
- Murphy DF. Intercostal nerve blockade for fractured ribs and postoperative analgesia. Description of a new technique. *Reg Anesth.* 1983;8:151.
- Murtagh FR. Computed tomography and fluoroscopy guided anesthesia and steroid injection in facet syndrome. *Spine.* Jun 1988;13(6):686-9. [Medline].
- O'Brien CF. Clinical applications of botulinum toxin: implications for pain management. *Pain Digest.* 1998;8:342-5.
- O'Kelly E, Garry B. Continuous pain relief for multiple fractured ribs. *Br J Anaesth.* Sep 1981;53(9):989-91. [Medline].
- Racz GB, Heavner JE, Singleton W, et al. Hypertonic saline and corticosteroid injected epidurally for pain control. In: Racz GB, ed. *Techniques of Neurolysis.* Boston: Kluwer Academic Publishers; 1989:73-86.
- Raymond J, Dumas JM. Intraarticular facet block: diagnostic test or therapeutic procedure?. *Radiology.* May 1984;151(2):333-6. [Medline].
- Relja M. Treatment of tension-type headache by local injection of botulinum toxin. *Eur J Neurol.* 1997;4 (suppl 2):S71-S73.

Rengachary SS, Watanabe IS, Singer P. Effect of glycerol on peripheral nerve: an experimental study. *Neurosurgery*. Dec 1983;13(6):681-8. [Medline].

Restelli L, Movilia P, Bossi L. Management of pain after thoracotomy: a technique of multiple intercostal nerve blocks [letter]. *Anesthesiology*. Sep 1984;61(3):353-4. [Medline].

Ridley MG, Kingsley GH, Gibson T. Outpatient lumbar epidural corticosteroid injection in the management of sciatica. *Br J Rheumatol*. Aug 1988;27(4):295-9. [Medline].

Robechhi A, Capra R. Lidrocortisone (composto F): prime esperienze cliniche in campo reumatologico. *Min Med*. 1952;98:1259.

Ryan MD, Taylor TKF. Management of lumbar nerve-root pain. *Med J Aust*. 1981;2:432.

Rydevik D, Holm S, Brown MD, et al. Nutrition of spinal nerve roots: the role of diffusion from the cerebral spinal fluid. *Transactions of the 30th Orthopedic Research Society Meeting*. 1984;9:276.

Saal JS, Franson RC, Dobrow R. High levels of inflammatory phospholipase A2 activity in lumbar disc herniations. *Spine*. Jul 1990;15(7):674-8. [Medline].

Schultz D at ASSIP, Memphis TN. December 1-2.

Sharrock NE. Correspondence. *J Bone Joint Surg*. 1985;67A:981.

Shealy CN. Dangers of spinal injections without proper diagnosis. *JAMA*. Sep 26 1966;197(13):1104-6. [Medline].

Shulman M. Treatment of neck pain with cervical epidural steroid injection. *Regional Anesth*. 1986;11:92.

Silvers HR. Lumbar percutaneous facet rhizotomy. *Spine*. Jan 1990;15(1):36-40. [Medline].

Simons DG. Clinical and etiological update of myofascial pain from trigger points. In: *Clinical Overview and Pathogenesis of Fibromyalgia Syndrome, Myofascial Pain*. 1996:93-122.

Sluijter M. Percutaneous thermal lesions in the treatment of back and neck pain. In: *Radionics Procedure Technique Series*. Burlington, Ma: 1981.

Sluijter ME, McMillin JN, Emery ST et al. Intradiscal steroids. A prospective double-blind clinical trial. *Spine*. 1992;17S:172-75.

Snoek W, Weber H, Jorgensen B. Double blind evaluation of extradural methyl prednisolone for herniated lumbar discs. *Acta Orthop Scand*. 1977;48(6):635-41. [Medline].

Sweet WH, Poletti CE, Macon JB. Treatment of trigeminal neuralgia and other facial pains by retrogasserian injection of glycerol. *Neurosurgery*. Dec 1981;9(6):647-53. [Medline].

Travell J, Simons D. Myofascial pain and dysfunction. In: *The Trigger Point Manual*. Baltimore: Williams and Wilkins; 1983.

Tsui JK, Eisen A, Stoessl AJ. Double-blind study of botulinum toxin in spasmodic torticollis. *Lancet*. Aug 2 1986;2(8501):245-7. [Medline].

Tucker GT, Mather LE. *Properties, Absorption, and Disposition of Local Anesthetic Agents*. 2nd ed. 1988:47-110.

Warfield CA. Correspondence. *J Bone Joint Surg*. 1985;67A:980.

Wasner G, Backonja MM, Baron R. Traumatic neuralgias: complex regional pain syndromes (reflex sympathetic dystrophy and causalgia): clinical characteristics, pathophysiological mechanisms and therapy. *Neurol Clin*. Nov 1998;16(4):851-68. [Medline].

Watkin H. Back pain--an integrated approach in primary care. *Acupunct Med*. Dec 2004;22(4):203-6. [Medline].

Wedel DJ, Wilson PR. Cervical facet arthrography. *Reg Anesth*. 1985;10:7.

Wheeler AH. Botulinum toxin A, adjunctive therapy for refractory headaches associated with pericranial muscle tension. *Headache*. Jun 1998;38(6):468-71. [Medline].

Wheeler AH. Therapeutic uses of botulinum toxin. *Am Fam Physician*. Feb 1 1997;55(2):541-5. [Medline].

Wheeler AH, Goolkasian P, Gretz SS. A randomized, double-blind, prospective pilot study of botulinum toxin injection for refractory, unilateral, cervicothoracic, paraspinal, myofascial pain syndrome. *Spine*. Aug 1 1998;23(15):1662-6; discussion 1667. [Medline].

Wheeler AH, Hanley EN Jr. Nonoperative treatment for low back pain. Rest to restoration. *Spine*. Feb 1 1995;20(3):375-8. [Medline].

Yates DW. A comparison of the types of epidural injection commonly used in the treatment of low back pain and sciatica. *Rheumatol Rehabil*. Aug 1978;17(3):181-6. [Medline].

Yue SK. Initial experience in the use of botulinum toxin A for the treatment of myofascial related muscle dysfunction. *J Musculoskeletal Pain*. 1995;3 (suppl 1):22.

Zwart JA, Bovim G, Sand T. Tension headache: botulinum toxin paralysis of temporal muscles. *Headache*. Sep 1994;34(8):458-62. [Medline].