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 non-noxious 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 CPRS 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 disturbances.

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.
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.
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.
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.
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...
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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 lidocaine-like derivatives such as mexiletine. To this point, neural blockade and therapeutic procedures that have been described entail the use of LA. 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 is often 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 axinolysis.

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.

Subarachnoid and epidural spinal 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 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 is rarely 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-gauge or 26-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 non-nociceptive 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.
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