Lumbar Sympathetic Block

57

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Introduction

The lumbar sympathetic block targets the sympathetic chain into the lower extremities with the objective of disrupting the nerve supply. This has been useful for the treatment of sympathetically mediated pain pathologies such as complex regional pain syndrome, peripheral vascular disease, vasospastic syndrome, frostbite, phantom limb pain, hyperhidrosis, and postherpetic neuralgia.

Background and Historical Perspective

While the anatomy of the sympathetic nervous system was delineated as early as the sixteenth century by Renaissance physician Vesalius, the functions of the sympathetic nervous system were not explored until the end of the nineteenth century with a series of surgical sympathectomies. In 1917, Rene Leriche, a military surgeon, performed the first periarterial sympathectomy on a patient with chronic hand pain and numbness after a gunshot wound to the axilla. After noting complete resolution of his patient's pain, Leriche successfully replicated his surgical technique for other syndromes with marked vasomotor dysregulation and conceived of the term "sympathetic neuritis"—a concept that emphasized the crucial role of the sympathetic nervous system in pathophysiology of neuropathic pain. With the goal of targeting the sympathetic ganglia, Royle and Hunter performed the world's first lumbar sympathectomy in 1923 on a young World War I veteran with spastic paralysis. They noted not only the anticipated result of reduced spasticity but also increased temperature and bright color of the affected limb—a seminal milestone that was quickly followed by Brown and Adson in 1925 who demonstrated the efficacy of lumbar sympathetic blockade for Raynaud's and obliterative arterial disease.

In 1924, Brunn and Mandl in Vienna described the first percutaneous lumbar sympathetic block, for which they initially used local anesthetic and later 70% alcohol. The original description outlined a paravertebral approach: (1) the spinous processes of the second through fourth lumbar vertebra were palpated, and three needles were placed 5–6 cm laterally to the spinous processes with a needle at each level, and (2) the needles were then advanced in a medial direction until contact with the anterolateral aspect of the vertebral body. In 1949, Haxon applied the same paravertebral technique in 220 patients with 1 important modification: he used phenol for sympathetic neurolysis rather than alcohol. The result was significant pain reduction for patients with hyperhidrosis, frostbite, and peripheral vascular disease; one patient even experienced 2 years of sympathetic denervation. Overall, the complications were noted to be minimal compared to the mortality and morbidity associated with open sympathectomy.

During World War II, lumbar sympathetic blockade rose in popularity as a treatment option for patients with lower extremity neuropathic pain and has continued to evolve over the past century as a commonly used interventional pain procedure. With the advent of fluoroscopy, CT, and ultrasound for visualization, lumbar sympathetic block has developed an improved safety profile, while the options for disrupting sympathetic innervations have expanded to include not only surgical ligation and local anesthetic infiltration but also various forms of chemical neurolysis and radiofrequency lesioning.

Uses and Indications

Lumbar sympathetic blocks are indicated for a multitude of sympathetically mediated pain disorder. These include the following (for full list, see Table 57.1):

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Table 57.1 Potential indications for lumbar sympathetic block

Complex regional pain syndrome I and II
Chronic occlusive arterial disease (includes critical limb ischemia)
Diabetic neuropathy
Phantom limb pain
Vasospastic disorders (Raynaud's phenomenon, acrocyanosis, livedo reticularis)
Thromboangiitis obliterans
Erythromelalgia
Cancer pain
Radiation neuritis
Frostbite
Acute herpes zoster, postherpetic neuralgia
Renal colic
Hyperhidrosis

I. *Complex regional pain syndrome (CRPS)* is a regional pain of complex pathophysiology involving disturbance of both central and autonomic nervous system. CRPS I is formerly known as reflex sympathetic dystrophy and entails presence of initiating noxious event or a cause of immobilization, while CRPS II is formerly known as causalgia and entails the presence of continuing pain after nerve injury. Patient typically endorses symptoms and exhibits signs of allodynia, hyperalgesia, vasomotor dysfunction, edema, and trophic changes of the affected limb.

   The optimal management of CRPS involves early diagnosis and time-dependent, interdisciplinary treatment, which focuses on rehabilitation, pain management, and psychological therapy. Any delay can adversely affect response to treatment. Therefore, a minimally invasive interventional pain procedure, such as lumbar sympathetic block, should be considered earlier in the treatment algorithm, especially when a patient has reached a plateau in progress after intensive rehabilitation and aggressive pharmacotherapy. In providing an updated algorithm of the management of CRPS, a panel of experts meeting in 2002 recommended minimally invasive procedures such as sympathetic nerve block in conjunction with mainstay therapy within the first 12–16 weeks—after which, failure to progress should prompt consideration of more advanced techniques such as spinal cord stimulation and DRG stimulation. Positive response to sympathetic nerve block may also bode well for spinal cord stimulation; a study in 2003 showed that patients with a good response to sympathetic blocks were more likely to have pain relief during SCS trial and long-term pain relief after placement of permanent device.

   While the sympathetic nervous system is traditionally implicated in the origin of CRPS, the terms sympathetically maintained pain and sympathetically independent pain have been employed to account for the inconsistent response to sympathetic blocking procedures.

II. *Chronic obstructive arterial disease (COAD)* is the reduction in blood flow in the arterial beds of the lower extremities, often manifesting as intermittent claudication when the obstruction of the vessel is greater than 50% and progressing onto severe persistent pain at rest when the collateral flow becomes insufficient, such as when COAD developed into critical limb ischemia (CLI). Because CLI is often associated with ulcers and gangrene, interventions such as bypass, endarterectomy, and stenting have to be undertaken. Lumbar sympathetic blockade is a viable treatment option for patients with painful COAD and CLI, for whom revascularization is not feasible, and should be considered prior to proceeding with amputation. Some vascular surgeons may even consider lumbar sympathetic neurolysis as a treatment for ulcers or as a bridge to revascularization. Lumbar sympathetic chemical neurolysis, a fairly safe and minimally invasive procedure, has similar short-term success rates compared to surgical sympathectomy and may be an effective strategy in avoiding or delaying surgical trauma.

   The mechanism of pain control after sympathetic blockade is likely related to the transient abolishment of basal and reflex constriction of arterioles and precapillary sphincters leading to increased flow through the occluded vessel as well as increased perfusion through collateral circulation. The improvement in blood flow bears nutritive value for the capillary beds and may alleviate further tissue damage. Furthermore, the sympathetic blockade may directly interrupt or attenuate nociceptive transmission directly via decreasing tissue norepinephrine levels, which would account for analgesic relief in the patients with severe multilevel arterial occlusions who are still able to experience pain relief without improvement in distal perfusion. Clinically, patients will typically demonstrate significant increase in warmth of the affected extremity due to shunting through cutaneous arteriovenous anastomosis as well as increased filling of the veins and increase in arterial pulsations.

III. *Vasospastic disorders*, including Raynaud's phenomenon, acrocyanosis, and livedo reticularis, present with the hallmark symptom of episodic cyanosis of the extremities. Excessive spasm of the peripheral blood vessels in the lower extremities results in numbness as well as pain. Conservative treatment includes lifestyle modification and calcium channel blockers, which may be effective in only two thirds of the patients; refractory cases of vasospasm can lead to unremitting pain as well as digital ulceration. While there is a paucity of data on efficacy of lumbar sympathetic blockade in vasospastic disorders, there exist case reports of successful bilateral lumbar sympathetic blockade performed on patients

with refractory Raynaud's phenomenon. The benefit of lumbar sympathectomy is likely due to its reduction of vasoconstrictor tone and improvement of circulation to the ischemic region.

IV. *Diabetic neuropathy* is a relatively common condition of microvascular dysfunction secondary to chronic hyperglycemia. Neuropathic pain can often occur as the presenting symptom, and patients may experience gradual onset of paresthesia, allodynia, and hyperalgesia. The pathophysiology of painful diabetic neuropathy is thought to involve increased cross talk between sympathetic fibers and peripheral sensory nerves, elevated circulation of norepinephrine and inflammatory mediators, and heightened dysregulation of sympathetic-mediated local circulation. When a newly diagnosed diabetic patient presented to Cleveland Clinic in 2012, Cheng recommended a series of lumbar sympathetic block over the course of 2 years. Each block resulted in 1–2 months of significant analgesic relief of the patient's painful diabetic neuropathy. The lumbar sympathetic block was hypothesized to decrease the patient's pain via reducing sympathetic outflow and improving circulation.

V. *Phantom limb pain* is the painful sensation of the amputated limb. Patients may often experience intermittent recurrence of the pre-amputation pain. Multiple mechanisms have been proposed for phantom pain, including peripheral consequences of amputation leading to the development of neuroma, loss of inhibitory control in the dorsal root ganglion, increased excitability of spinal cord neurons, as well as inappropriate central reorganization of the thalamus and cortex. Sympathetically maintained pain is hypothesized to occur when sympathetic nerve fibers secrete not only sympathetic neurotransmitters, such as norepinephrine, but also inflammatory mediators, which can directly stimulate and sensitize the peripheral nociceptive fibers. The sensitized peripheral fibers then release more neurotransmitters to sensitize other pain fibers and contribute to the overall inflammatory state. In amplifying the general inflammation with vasodilation, edema, and activation of white blood cells and other inflammatory cells, the sympathetic nervous system plays a key role in maintaining and exacerbating the chronic pain state. Successful lumbar sympathetic blockade for the treatment of phantom limb pain has been described in multiple case reports; however, the results are not consistent as there may be a subset of phantom limb pain which is sympathetically independent.

VI. *Plantar hyperhidrosis* is a functional disorder of excessive secretion of the sweat glands of the feet that is associated with an overacting sympathetic nervous system. Severe cases of plantar hyperhidrosis can be disabling and personally and professionally limiting. In patients who do not respond to conventional treatment, such as topical agents and botulinum toxin injections, percutaneous lumbar sympathetic neurolysis and radiofrequency ablation have been attempted with varying success. Since 2008, an endoscopic approach to lumbar sympathectomy, entailing clamping of the sympathetic chain at L3 or L4, has been gaining traction among surgeons as an outpatient procedure. Multiple prospective studies demonstrate at least a 95% success rate in achieving anhidrosis.

Anatomy

The sympathetic trunk, also known as the paravertebral ganglia for it lies alongside the vertebral column, is composed of bilateral sympathetic chain ganglia, extending from the cervical spine down to the coccyx. There are 3 pairs of ganglia in the cervical region, 11 pairs in the thoracic region, 4 pairs in the lumbar region, and 4 to 5 pairs in the sacral region. The lumbar portion of the sympathetic trunk lies along the anterolateral aspect of the first through fourth lumbar vertebra. Anterior to the chain on the right is the IVC; anterior to the chain on the left lies the abdominal aorta. Posterior and medial to the chain are the intervertebral foramina. Posterior and lateral to the chain is the psoas muscle, which separates the lumbar sympathetic chain from the somatic plexus and minimizes spread of local anesthetic or chemical neurolysis (see Fig. 57.1). The preganglionic neurons of the lumbar sympathetic trunk arise from the intermediolateral nucleus of the lateral gray column of the spinal cord at levels T10 to L3 and emerge via the white rami of the ventral roots of spinal nerves L1–L4 before synapsing at the paravertebral ganglia. The axons of the postganglionic neurons then extend distally and innervate specific sites in the pelvis, lower abdomen, and lower extremities.

Despite variability in anatomic locations, most cadaveric studies suggest lumbar sympathetic plexus is most dense at the level of the mid-body of the third lumbar vertebra and the discs above and below. Specifically, the ganglia is most frequently located 0–0.5 cm posterior the anterior body of the third lumbar vertebral and 1.8–3.0 cm laterally from the center of the vertebra. Studies of radiofrequency ablation of L3 ganglia are consistent with anatomic studies in showing that adequate block can often occur from blocking L3 ganglia alone, while studies of ganglionectomy show that resection at L2 and L3 is usually sufficient. Therefore, the best approach is to advance the needle paravertebrally to lower third of L2 or upper third to midpoint of the body of L3 (see Fig. 57.2).

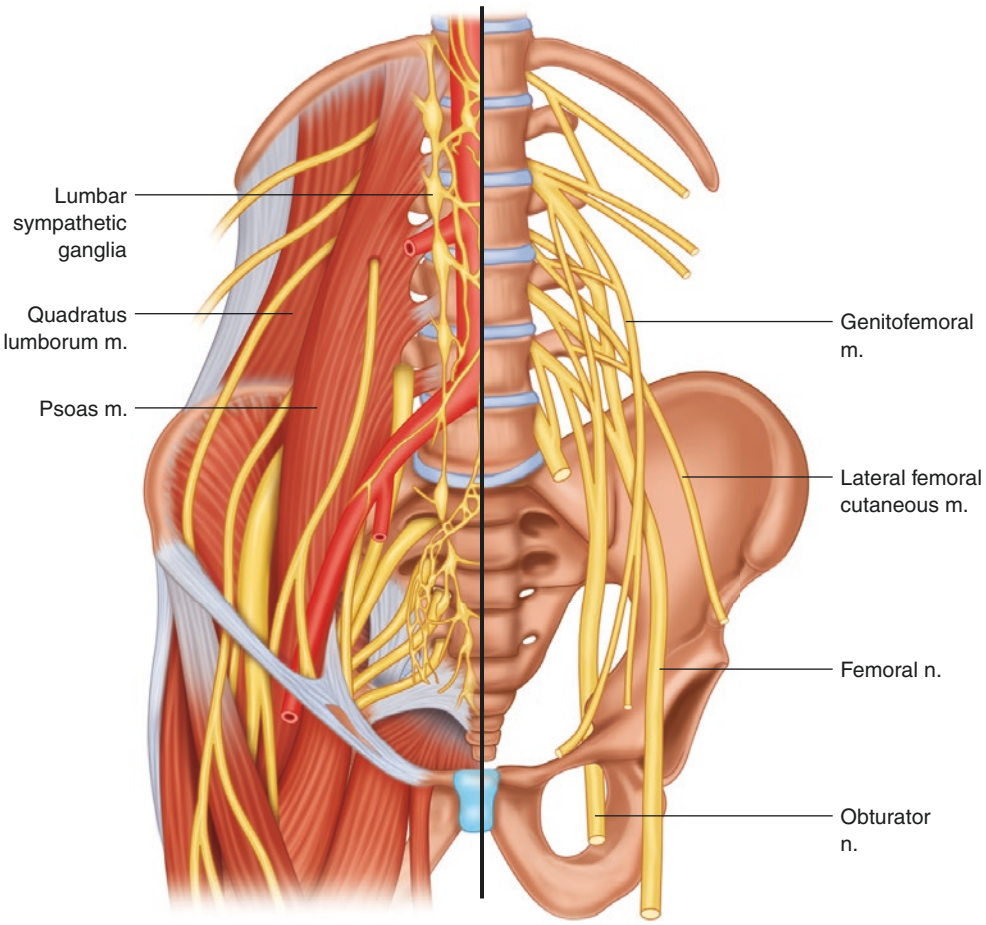

Fig. 57.1 Schematic of lumbar sympathetic ganglia located anterior to the psoas muscle, which separates the ganglia from the lumbar plexus

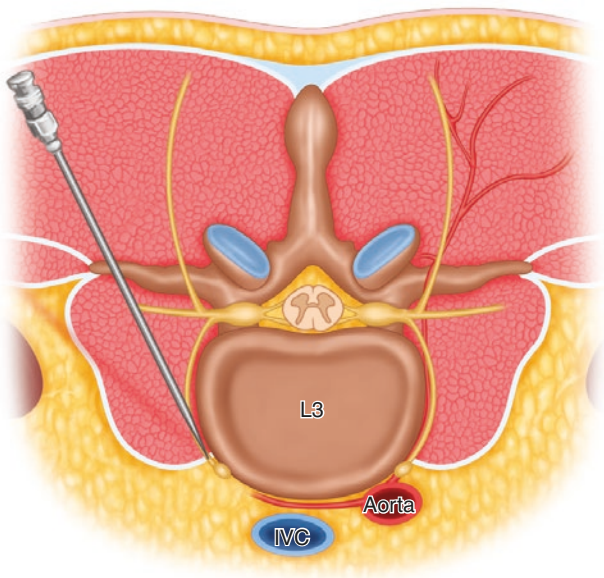

Fig. 57.2 Axial schematic of needle trajectory using paravertebral approach

Technique

Prior to positioning, the baseline temperature of bilateral lower extremities should be established and recorded. Continuous EKG, oxygen saturation, and intermittent blood pressure monitoring along with intravenous access are recommended. The patient is positioned prone with a cushion placed under the abdomen to reduce the lumbar lordosis. Fluoroscopic beam is adjusted until the superior end plates of L2–L3 are aligned. From there, the C-arm is rotated ipsilaterally until the tip of ipsilateral L2 or L3 transverse process is superimposed with the lateral border of the L2 or L3 vertebral body. The desired entry point is typically along the inferior third of the anterolateral border of the L2 vertebral body or superior to mid-third of the anterolateral border of the L3 vertebral body. In this oblique view, the needle is advanced until the needle tip contacts the anterolateral border of the vertebral body.

Lateral imaging is obtained to confirm that the needle tip is at the anterior vertebral line. An AP image is taken to

verify that the needle tip remains within the medial border of the ipsilateral pedicle. 1–5 ml of contrast is injected. Appropriate spread of contrast typically appears "cloudy" and extends superiorly and inferiorly in front of the ipsilateral lumbar vertebra rather than laterally; ideally, there is sufficient spread from L1 to L3, and if not, a multi-needle technique should be considered. The contrast spread should not be "streaky" and extend caudally and laterally in outlining the psoas muscle. If the needle tip is still within the psoas muscle, the needle should be advanced slightly until repeat injection of contrast shows appropriate spread; failure to advance past the psoas muscle may result in blockage of the anterior rami of the upper lumbar region.

Digital subtraction angiography (DSA) can be used to confirm lack of vascular pattern as it is more sensitive than continuous fluoroscopy. Once appropriate contrast spread is demonstrated on fluoroscopy, 5–15 ml of local anesthetic, usually 1% lidocaine or 0.25% bupivacaine or 1:1 mixture, is injected in 1–2 ml increments after verifying negative aspiration for heme and CSF. If chemical neurolysis is desired, 2–3 ml of phenol (3%–6%) or alcohol (50%–100%) is injected. Failure to obtain appropriate contrast spread should prompt consideration of aborting the procedure.

A technically successful block is supported by post-procedural temperature rise of at least 3 °C in the ipsilateral lower extremity. Following the procedure, patient's pain is re-evaluated for any resolution or reduction of pain. EKG changes and any mental status changes should be closely observed. Of note, some protocols recommend maintaining patients in the prone position afterward with the needles in situ for 20–30 minutes to minimize posterior spread of the solutions into the psoas muscle, but this is usually considered when phenol is used rather than local anesthetic. Regardless, the patient should be monitored for at least 20 minutes post-procedure.

With minimal modification to the technique, conventional radiofrequency ablation could be undertaken. A 20 gauge 150-mm-long radiofrequency needle with a 10 mm non-insulated tip should be utilized. Two levels, i.e., L2 and L3, could be considered though one level has been shown to be adequate in many cases. After confirming appropriate position via contrast, electrical stimulation can be carried out using 50 Hz and 5 Hz to identify any proximity to sensory and motor nerves and to decrease the risk of ablating a segmental nerve root. 1 ml of 2% lidocaine is injected prior to lesioning at 80 ° for 1 minute. Pulsed radiofrequency ablation can similarly be performed over 3 cycles and 120 seconds/cycle per level at a temperature of 42 °C. The generator is usually set at a target voltage of 45 Volts with a pulse width of 20 ms and a pulse rate of 2 Hz though these parameters are usually automatically changed to maintain the temperature.

Efficacy

Evidence for efficacy of lumbar sympathetic blocks in the treatment of sympathetically mediated pain disorders comprise primarily of case studies and single-center randomized trials. There is a paucity of high-quality randomized control trials comparing treatment with placebo.

Evidence for Efficacy in Complex Regional Pain Syndrome

Lumbar Sympathetic Block Versus Placebo

Price in 1998 evaluated seven patients with complex regional pain syndrome in a double-blind, placebo-controlled crossover study. Four patients underwent stellate ganglion block, while three underwent lumbar sympathetic block. Magnitude and duration of pain relief were monitored. There was no significant difference in the initial peak reduction of pain intensity, as measured by mechanical allodynia 30 minutes after the block, between patients who received saline and local anesthetic. However, there was a significant difference in duration of local anesthetic (90 hours) as compared to saline (19 hours) in all patients.

Cochrane review in 2016 concluded that there was insufficient evidence to support or refute the efficacy of lumbar sympathetic block for the treatment of CRPS. To date, the most rigorous trial of note is a double-blind, placebo, crossover trial in 23 patients, age 10–18 years of age, with CRPS. Half of the patients were randomized to lumbar sympathetic blockade with lidocaine and an IV saline infusion, while the other half were randomized to a lumbar sympathetic blockade with saline and an IV lidocaine infusion. Patients who received lumbar sympathetic blockade with lidocaine demonstrated significant pain reduction and improvement in sensory dysfunction compared with placebo—this provided evidence that a portion of pain secondary to CRPS was mediated by abnormal sympathetic outflow.

Radiofrequency of Lumbar Plexus Versus Chemical Neurolysis

In a comparison study of radiofrequency denervation and phenol sympathetic blocks, 17 patients in 1991 underwent either lumbar sympathetic neurolysis with phenol or radiofrequency of the lumbar sympathetic plexus. Eighty-nine percent of patients in the phenol group were shown to have sympathetic blockade after 8 weeks whereas only 12% in the radiofrequency ablation group ($P < 0.05$). The radiofrequency ablation group was noted to have lower incidence of post-sympathetic neuralgia. A similar study was undertaken in 2008 with refinement of the radiofrequency ablation technique. In the ten patients randomized to conventional radiofrequency, the radiofrequency cannula was introduced

lateral to L2, L3, and L4 with two sites lesioned at each level, whereas in the ten patients randomized to neurolysis, the cannula was similarly placed, but this was followed by 3 ml of 7% phenol at every level. The conclusion was that there was no statistical significant difference in efficacy in the days to 4 months following the procedure. Freitas in 2014 randomized 40 patients with CRPS Type I to pulsed radiofrequency ablation or sympathetic neurolysis. Pain scores and quality of life were recorded in follow-up of 1 day, a week, 2–4 months, and 6 months; based on these measures, pulsed radiofrequency was found to have comparable results to phenol neurolysis.

Lumbar Sympathetic Block with Botox Versus Conventional Lumbar Sympathetic Block

A preliminary study in patients with refractory complex regional pain syndrome (CRPS) by Carroll in 2009 showed the addition of botulinum toxin A to bupivacaine, a standard local anesthetic in lumbar sympathetic blocks, can significantly prolong the median analgesic period from 10 days to 71 days. One proposed mechanism of analgesia is the inhibition of release of acetylcholine from the cholinergic sympathetic ganglia.

Evidence for Efficacy in Peripheral Vascular Disease

Cohort Studies

The evidence for efficacy in peripheral vascular disease comprises of both cohort studies and early randomized trials. A retrospective study of 153 patients who underwent surgical lumbar sympathectomy showed that after 5 years, 67% of patients with claudication and 54% of patients with rest pain were able to avoid further surgery. A study of 385 patients after surgical lumbar sympathectomy by Matarazarro in 2002 reported improvement in pain, recovery of trophic lesions, and rise in skin temperature in 63% after 1 year.

In 1995, Mashiah described a prospective study of 373 patients with peripheral vascular disease who underwent a phenol lumbar neurolysis; over 1–10-year follow-up, it was found that 58% experienced pain relief and healing of gangrenous ulcers. The results were most notable in diabetic patients with rest pain and in nondiabetic patients with digital gangrene or ulcers, suggesting patient selection is key.

Chemical Neurolysis Versus Placebo

In 1975, Fyfe randomized 25 patients with claudication pain to phenol sympathectomy and local anesthetic control and found that there was no subjective improvement in pain or objective evidence in improvement with treadmill testing. In 1985, 41 patients with intermittent claudication were randomized to either phenol lumbar neurolysis or a placebo bupivacaine injection; 83% of patients in the treatment group reported decreased pain compared to the 23% of patients in the placebo group at 6 months. Updated Cochrane review in 2016 concluded that there was no high-quality randomized controlled trials that compared surgical or percutaneous lumbar sympathectomy with no treatment in patients with critical lower limb ischemia as there were only four randomized trials described in the literature to date and patient selection was not based on sufficiently objective criteria.

Pearls and Pitfalls

Lumbar sympathetic block is a minimally invasive and relatively safe pain reduction procedure. The most common complications include bleeding, swelling, and soreness at the injection site. However, the complications can span a full spectrum of categories, including neurologic, renal and ureteric, and vascular sequelae (see Table 57.2 for comprehensive list of pitfalls and pearls).

To minimize procedural complications, one must focus on both careful patient selection and precise needle advancement. Pre-procedure discussion with the patient should involve a discussion of anticoagulation; specifically, patient must hold anticoagulation for the same specified number of days as he or she would for a neuraxial procedure. To highlight the incompatibility of concurrent anticoagulation and lumbar sympathetic block, Hohf in 1953 presented a series of cases, which ultimately all resulted in death from massive retroperitoneal hematoma.

CT scans of cadavers suggest that the paravertebral distance of insertion of needles should not exceed 6, 7, and 10 cm lateral to the spinous processes of L2, L3, and L4, respectively, so as to avoid puncturing internal organs. Placement at the superior third of L2 is potentially challenging as the needle tip might approach the vertebral insertion of the diaphragm. The correct final needle position should be 0.5–0.8 cm dorsal to the anterior vertebral border, for if it were a right-sided lumbar sympathetic block, advancement toward the vertebral border might led to inadvertent IVC puncture in 20% of the cases. The optimal approach is to advance the needle paravertebrally through the fascia of the psoas muscle slightly cephalad to the midpoint of the body of L3; excessive anterior advancement of the needle will likely result in intravascular puncture, whereas insufficient advancement of the needle past the psoas muscle will likely result in partial block of the lumbar plexus, resulting in a temporary ipsilateral weakness post-procedurally.

Table 57.2 Complications following lumbar sympathetic block and tips

Complication	Description	Tips
Neuralgia	The most common neuralgia is that of genitofemoral nerve (incidence varies from 6 to 40% after chemical neurolysis); patient will typically present with dysesthesia in the anteromedial upper thigh, approximately 2–6 weeks in duration Depending on the extravasation to the psoas muscle, neurolysis may also spill over to the lateral cutaneous nerve of the thigh, resulting in transient meralgia paresthetica	Do not inject if the contrast spread suggest the needle tip is in the psoas muscle Use smaller volumes of neurolytic agent at levels above L4 to minimize spread to genitofemoral nerve Symptoms can be relieved with transcutaneous electric nerve stimulator and intravenous lidocaine infusion
Somatic nerve block	Patients may exhibit reduced strength in the myotomes of L1–L4, suggesting posterior spread of local anesthetic along tendinous arches which bridges the concave sides of lumbar vertebra, or reduced strength in the distribution of the femoral nerve, suggesting inadvertent injection of local anesthetic into the psoas muscle	Do not inject if the contrast spread suggest the needle tip is in the psoas muscle Use smaller volumes of local anesthetic to minimize extravasation
Subarachnoid injection	Death can result following lumbar sympathetic blockade, following inadvertent subarachnoid injection, leading to rapid motor block and hypotensive collapse seizures	Pre-procedural intravenous access and monitoring of vitals are recommended as safeguards All practitioners should have ready access to resuscitation equipment and be ready to administer cardiopulmonary resuscitation
Post-dural puncture headache (PDPH)	There have been cases of PDPH without signs of subarachnoid injection. Entry point for inadvertent dura puncture may be a dural root sleeve or other anatomical variants	Epidural blood patch may not be effective if there was puncture of anatomical variant
Paraplegia	While the risk for paraplegia is higher with thoracic paravertebral alcohol injection or celiac plexus neurolysis, there is one documented of a T10 myelopathy. The mechanism of injury is unclear and may be either due to an anterior spinal artery ischemia or a direct spinal cord injection. Inadvertent intrathecal injection of neurolysis may also cause paralysis but is more likely to be transient	Alcohol likely provides more intense neural destruction than phenol and diffuse more rapidly through tissue. While phenol provides a shorter duration compared to alcohol, phenol may be a safer choice though there is no controlled study comparing the two for lumbar sympathetic block
Aseptic meningitis	There have been 2 cases of self-limiting aseptic meningitis without any obvious signs of an intrathecal injection	
Epidural injection	Retrograde spread via communicating sympathetic rami into the epidural space can result depending on the volume of injectate. In one study, 5/6 patients were shown to have epidural spread when 5 ml of injectate was used at L2, whereas in another study, 3/5 patients were shown to have epidural spread extending to T10 when 10 ml of injectate was used	Use smaller volumes of injectate to minimize posterior extravasation Maintaining the patient in the prone position for 20–30 more minutes following the procedure may limit posterior spread
Renal and ureteric injuries	Patient may experience hematuria, which resolves spontaneously after 2–3 days, suggesting puncture injury to the kidney or ureter. Sequelae of renal puncture also include transient hematuria, renal colic, dysuria, and massive subcapsular hematoma	Use paravertebral technique with fluoroscopy or CT. The needle entry should not be too lateral Anatomical study using CT showed that insertion of needles in L2, L3, and L4 should not exceed 6, 7, and 10 cm from midline, respectively
Vascular complications	Multiple reports of retroperitoneal hematomas in patients who were concurrently on anticoagulation were described in the 1950s	Patients on anticoagulation should not undergo lumbar sympathetic blocks The pain practitioner should abide by the American Society of Regional Anesthesia (ASRA) guidelines for holding anticoagulation for neuraxial procedures
Hypotension	Hypotension may result immediately following the procedure due to vasodilation following sympathetic blockade and may also develop in the hours to days following the procedure due to an occult bleed	Limit injectate volume and perform unilateral lumbar sympathectomy rather than bilateral If large volumes of local anesthetic are desired, consider administering IV fluids prior to the procedure in healthy patients who can tolerate increased preload

(continued)

Table 57.2 (continued)

Complication	Description	Tips
Local anesthetic toxicity	Provided that the injection is not intravascular, there is low risk for local anesthetic toxicity as the lumbar sympathetic ganglion is not localized to a highly vascular area. The local anesthetic absorption is notably slow. In one study, patients who received lumbar sympathetic blocks with 20 ml of 0.25% bupivacaine did not demonstrate peak levels above the toxic threshold; the duration to peak plasma concentration was approximately 24–60 minutes later	Injectate volume of 20 ml of local anesthetic can be considered Consider test dose of lidocaine with 1:200 K epinephrine with pulse monitoring to assess if there's any intravascular spread Be ready to administer intralipid in an emergent situation. May require extracorporeal membrane oxygenation if bupivacaine is injected intravascularly.
Pneumothorax	Rare complications following lumbar sympathetic block include pneumothorax	Avoid placement of needle at the superior third of L2 as the needle tip will approach the vertebral insertion of the diaphragm If abnormal anatomy is anticipated, consider CT-guided lumbar sympathetic block or obtaining a CT prior to the procedure for planning the safest needle trajectory
Discitis	Discogenic pain may result from inadvertent intra-discal injection	Place needle tip at the level of mid-body of vertebrae
Sexual dysfunction	Retrograde ejaculation is a known risk following bilateral lumbar sympathetic block. Diabetes is a risk factor for retrograde ejaculation	Avoid bilateral lumbar sympathetic block

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