Lumbar Sympathectomy

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Abstract

Lumbar sympathectomy is a procedure that has been used in the past century for various disease processes. Its role has evolved as treatment modalities for the aforementioned pathologies have advanced. It is currently reserved for patients with complex regional pain syndromes, hyperhidrosis, and vascular disease not amenable to revascularization or other medical therapies.

Introduction

The topic discussed in this chapter is not a specific disease process that is in need of treatment but a procedure in need of a disease process. Lumbar sympathectomy has been performed for almost a century for various indications. Its main indication before the birth of peripheral vascular operations was to relieve ischemic rest pain. Many of the current indications have to do with pain control in the setting of complex regional pain syndrome (CRPS), hyperhidrosis, and recalcitrant vascular disease.

Pain is a primitive protection mechanism that has been evolutionarily selected to protect the organism from noxious stimuli from outside world. As a secondary benefit, pain informs the organism of internal problems. As a clinician, it is our role to tease out the subtleties of what this internal pain translates into. It is often within this framework that we try to alleviate the pain by addressing the causing factor. The fibers that

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deliver the pain message can also be damaged or erroneously stimulated in the absence of internal and external noxious stimuli resulting in a sensation of pain that is pathologic.

It is our goal in this chapter to delineate one of the basic methods of pain modulation that has been well known and practiced for many years. We will explore the role of the sympathetic nervous system in pain. In addition the chapter will explore the physiologic role of the sympathetic nervous system, how it regulates blood flow to organs, and how it can be manipulated in an attempt to improve blood flow to end organs. It will also discuss the sympathetic mechanisms involved in hyperhidrosis.

Anatomy and Physiology of Autonomic Nervous System

The autonomic nervous system (ANS) consists of a primitive automated system of nerves that are not under the direct conscious control of the organism. The system has evolved to respond to outside and sometimes inside stimuli without the need for direct higher cortical function. It has classically been divided into sympathetic nervous system (SNS) and parasympathetic nervous system (PSNS). The modern descriptions also include a third component, the enteric nervous system (Langley 1903). The ANS coordinates bodily functions to ensure homeostasis and responses to stress.

Anatomically, the SNS consists of a central component which includes the cerebral cortex (insular and medial prefrontal region), amygdala, stria terminalis, hypothalamus, brain stem, and diencephalon (Benarroch et al. 1993). In the periphery, there are preganglionic and postganglionic neurons. The information from the central part of the SNS travels to the preganglionic neurons in the anteromedial column of the thoracolumbar cord. These originate from the level of T1 to the level of L2. The axons from these preganglionic neurons travel to the paravertebral sympathetic ganglia. In these ganglia, the preganglionic neurons synapse onto the postganglionic neurons. The postganglionic neurons then have axons that travel up and down the chain of ganglions before exiting and innervating the target organ (Shields 1993).

The SNS controls what is known as fright, fight, and flight response of the body. The sympathetic fibers innervate the cutaneous and subcutaneous arterioles. Here, the sympathetic response causes constriction of these vessels, decreasing flow to the skin and its underlying structures. In a similar fashion, the sympathetic response constricts the blood supply to all body system not involved with fight or flight. This serves to reserve the flow for muscles when in a fight or flight situation as well as preventing heat loss when the body is cold. The same system also increases the blood flow to the muscles of the extremities in order to prepare the organism for activity as well as shivering in cold conditions. The exocrine glands of the skin are stimulated by the sympathetic nervous system to produce sweat. In addition, the system innervates the erector pili muscles on the skin and causes piloerection.

Historical Perspective

The sympathetic nervous system was described by Galen (130–200 AD) and then further depicted by Vesalius (1514–1564) (Galen 1968) and Eustachius (1514–1574) (Tissot 1778). Its name comes from Jacques Benigne Winslow (1669–1760) who believed these nerves control the "sympathies" of the body (Winslow 1732). It took until the later part of the nineteenth century to link its physiologic role to vasoconstriction. Langley, a Cambridge University physiologist, coined the term autonomic nervous system in 1898 and described its three components (Langley 1903).

The first sympathectomy performed was by Alexander in 1889. It was a cervical sympathectomy performed for the treatment of epilepsy. In 1913 Leriche introduced periarterial sympathectomy for ischemic lesions caused by vasospasm (Leriche 1943). The first lumbar sympathectomy was performed in 1923 by Royle on a patient with spastic paralysis of the lower extremity (Royle 1924). The minimally invasive retroperitoneal

approach using a scope was first described by Wittmoser in 1973 (Wittmoser 1973) and thoracoscopic sympathectomy was first introduced by Kux in 1977 (Leseche et al. 1995).

In 1924, Felix Mandl first described lumbar sympathetic block. James White used 95 % alcohol to perform chemical sympathectomy for patients with hyperhidrosis in 1935. In 1949, Haxton published the results of injection of the lumbar sympathetic chain with 10 % phenol in patients with peripheral arterial disease. Robert Boas who was an anesthesiologist introduced fluoroscopic-guided positioning of needles and injection of neurolytics as a way to perform sympathectomy in 1976 (Huang 2002).

Lumbar sympathectomy was popularized in the first half of the twentieth century for treatment of peripheral arterial disease. It was used in the 1930s–1950s because it was the only alternative to amputation. With the advent of open revascularization in the 1950s, endarterectomies and bypass procedures surpassed sympathectomy as the procedure of choice for arterial occlusive disease. Sympathectomy was repopularized as a therapeutic choice for CRPS in the late twentieth century and now plays a significant role in recalcitrant CRPS. It has and remains a good treatment option for hyperhidrosis.

The Role of Sympathetic Nervous System in Pain

Although, there is no direct connection between the pain pathways and the SNS, it is well known that hyperactivity in the SNS can increase anxiety that may lead to amplified pain perception, effect pain behavior, and depress mood.

As a part of response to injury, the sympathetic pathways are activated. Under ordinary circumstances, these pathways shut down within minutes to hours after the noxious stimuli have stopped. In certain individuals, the sympathetic activity can remain high. The abnormally high sustained sympathetic activity can lead to blood vessel spasm, leading to more inflammation, swelling, and pain.

Stimulation of the SNS also results in the release of noradrenalin from the nerves as well as the adrenal gland along with adrenalin. The release of these into the blood stream will increase the heart rate and sweating, dilate the blood vessels to the muscles in anticipation of increased muscle activity, as well as constrict blood flow to other tissues nonessential to fight or flight.

In special circumstances, the sympathetic nerves that usually do not carry the pain signals can act to modulate the pain sensation through two different pathways. When nerve injury occurs, the sympathetic fibers can grow toward the injured pain nerve fibers and release noradrenalin onto the injured pain fibers. The noradrenalin potentiates the electrical excitability of the injured pain nerve fibers and increases the sensation of pain. The second mechanism involves activation of the SNS secondary to the pain sensation through central pathways that leads to "reflex" increase in sympathetic activity. This mechanism will then lead to skin color changes as well as temperature, increased sweating, and edema and inflammation in the area of injury (Sato and Perl 1991).

Role of Sympathetic System in Vascular Disease

The blood vessels at the level of arterioles are controlled by the sympathetic nervous system. It is well known and experimentally demonstrated that sympathetic denervation increases blood flow to an extremity with normal physiologic flow and no arteriopathy. What is less clear is the impact of the procedure on limbs with significant arterial occlusive disease. It is believed that there are four mechanisms by which there is improvement in patients with physiologically significant peripheral arterial disease.

The first is increased blood flow. With no sympathetic tone, the arterioles and the precapillary sphincters will maximally dilate allowing the maximal amount of flow to reach the tissues (Barcroft and Swan 1953/1954; Cronenwett and Lindenauer 1977; May et al. 1968). It can also contribute to wound healing of superficial ulcerations by increasing the flow through cutaneous arteriovenous anastomoses.

Another mechanism is the effect of collateral circulation is increased. The smaller arteries that carry the blood around the blockage will maximally dilate after sympathectomy. This dilatation has been shown in studies to increase the blood flow by 11 % (Ludbrook 1966). The improved flow may however be short lived, and the local distal tissues that are ischemic usually produce locally released factors that maximally dilate the collateral circulation around a major arterial blockage as well (Cronenwett et al. 1983).

There is a controversy about whether the nutritive value of blood flow is increased to the skin and the subcutaneous structures in order to improve wound healing. The increased flow through the skin with sympathetic stimuli is through these arteriovenous shunts that bypass the capillaries. It is debated whether this increase in flow is providing the increased nutritional requirements to heal superficial wounds (Cronenwett and Lindenauer 1980; Welch and Leiberman 1985).

Finally, there is alteration of the pain impulse transmission after sympathectomy. Studies on the feline hind limb noxious stimuli model have shown that sympathectomy enhances tolerance of noxious stimuli to that limb (Petten et al. 1983). It is theorized that the perception of painful stimuli is dampened by decreased norepinephrine in the tissues and decrease in the transmission of the painful stimuli that are transmitted to the brain.

Clinical Indications for Sympathectomy

Complex Regional Pain Syndrome (CRPS)

Complex regional pain syndrome presents as a constellation of symptoms generally related to a traumatic injury. This clinical entity has been known by many names, including causalgia, reflex sympathetic dystrophy, and Sudeck's atrophy. Successful surgical treatment for CRPS with sympathectomy may be optimized by first exhausting noninvasive medical options. Patient education, oral analgesic trials, physical therapy, and psychosocial evaluations are essential. Additionally, a favorable response to lumbar sympathetic nerve block confers a favorable response to sympathectomy (Bandyk et al. 2002).

Raynaud's Disease/Vasospastic Disorders

Symptoms of Raynaud's are best avoided by maintaining digital normothermia in an effort to prevent peripheral arterial vasospasm. Temperature biofeedback has been shown to decrease incidence of vasospasm. It is not until symptoms disrupt activities of daily living and conservative measures fail that pharmacologic therapy is indicated. The mainstay of medical therapy includes drugs from the following medication classes: calcium channel blockers, angiotensin-converting enzyme inhibitors, serotonin reuptake inhibitors, and prostaglandins. Those patients with symptoms refractory to medical therapy qualify for treatment by interruption of the lumbar sympathetic ganglia. The procedure is typically offered to patients with tissue loss secondary to vasospasm (Janoff et al. 1985).

Plantar Hyperhidrosis

There are a multitude of medical treatments for hyperhidrosis. First-line therapy includes the topical application of aluminum chloride hexahydrate and Drysol. The use of anticholinergic agents such as propantheline bromide, glycopyrrolate, and oxybutynin may be limited by their systemic effects including ametropia, xerostomia, constipation, and difficulty with micturition. Botulinum toxin injections produce a temporary, though well-tolerated and effective, relief of symptoms. Lumbar sympathectomy is reserved as the treatment of refractory hyperhidrosis (Rieger et al. 2009; Rieger and Pedevilla 2007).

Thromboangiitis Obliterans (TAO)

The most effective intervention in the treatment of TAO is cessation of tobacco smoking. Anti-inflammatory medications have failed to improve

distal limb perfusion. When compared to aspirin, the prostaglandin analogue, Iloprost, demonstrated improved ulcer healing and ischemic pain in addition to decreasing the need for amputation (Fiessinger and Schäfer 1990). Attempts at distal arterial revascularization have resulted in high incidence of graft failure (Sasajima et al. 1997). Lumbar sympathectomy may be considered in patients who quit smoking and continue to suffer from rest pain and nonhealing skin ulceration.

Lower Extremity Rest Pain/Ulceration Without Possibility of Revascularization

For those patients with advanced peripheral arterial occlusive disease who cannot undergo bypass – either due to high medical risk of surgery or lack of distal target vessels – therapeutic options remain few. Due to decreased local tissue perfusion, local wound debridement has limited success and may ultimately lead to lower extremity amputation. Lumbar sympathectomy by chemical ablation or open surgical resection has been performed in order to provide limb salvage with alleviation of rest pain and healing of some superficial ulcers (Nesargikar et al. 2009; Holiday et al. 1999). Patients with severe PAD with ABI of less than 0.3 have poor outcomes (Yao and Bergan 1973). Diabetics with autonomic neuropathy should also be excluded as these patients have poorly functioning sympathetic system.

Diagnostic Testing

Although diagnostic testing of the sympathetic nervous system is not a necessary part of the work up of non-hyperhidrosis vascular patients, it should be mentioned here. Most of the testing involves measuring the activity of sweat glands and therefore is an indirect test of the sympathetic function. The tests described here are expensive and require specialized equipment and the expertise to perform and interpret them.

Silastic imprint test, quantitative direct and indirect axon reflex, and direct sweat tests are sophisticated tests to evaluate postganglionic sympathetic cholinergic pseudomotor function by evaluating sweating in response to stimulation by iontophoresis of acetylcholine, pilocarpine, or methacholine. The level of sweating is then measured with different techniques depending on the test. These tests have limitations as to the level of their sensitivity (Sandroni 1998). Room temperature, humidity, anxiety level, hydration status, and even coffee intake can affect the results of these tests.

The sympathetic skin response test measures the electrodermal activity as a surrogate marker for sympathetic activity. A stimulus induces a change in the skin potential which can be recorded. The stimulus could be electric, acoustic, or even deep breathing. The sympathetic skin response is then recorded as present or absent (Sandroni 1998).

A more effective and important test for the practitioner who intends to evaluate the patient for potential sympathectomy is sympathetic nerve block. Bupivacaine is injected near the sympathetic ganglion, and it temporarily stops the function of the sympathetic system. The patient is then asked if the symptom of concern be it pain or hyperhidrosis has gone away. If by temporarily disabling the sympathetic system the patient has relief of symptoms, then lumbar sympathectomy would most likely produce the desired effect on the patient (Jeong et al. 2013).

Techniques for Sympathectomy

Classically, lumbar sympathectomy was performed as an open surgical technique. In the current era of minimally invasive procedures, there are various techniques that can be used to achieve similar results but each fraught with its own set of complications. In addition to chemical sympathectomy, percutaneous radiofrequency techniques, long-term blocks with botulinum toxin, and implantation of stimulators on the sympathetic ganglion chain have been described.

Open: The classic open technique involves placing the patient in the lateral decubitus position in the same manner as performing retroperitoneal

aortic repair or vena cava repair. A much smaller incision is made extending from the tip of the 12th rib down to the rectus muscle at the level or below the level of umbilicus. The three layers of abdominal wall muscles are divided. The retroperitoneal space is entered and dissected bluntly forward. The ureter and the gonadal vessels should be mobilized anteriorly with care not to injure them. On the left side, the space between the aorta and the psoas muscle should be explored. The lumbar portion of sympathetic chain lies on the anterolateral bodies of the lumbar vertebrae. It can be palpated as a vertical cord that is firm secondary to tethering to rami communicantes. On the right, the space between the psoas and the inferior vena cava is explored. The sympathetic chain is typically in this position. Once clearly identified, the second to the fourth or fifth ganglia are removed. Clips are applied to the ends. The specimen is then sent to pathology for confirmation. A transperitoneal approach can also be performed but is unpopular secondary to need for visceral rotation.

Laparoscopic/Endoscopic: The minimally invasive techniques can be performed through the peritoneum. The abdominal cavity is entered by trocars and insufflated with carbon dioxide. In this case, the right or left colon is mobilized as well as the kidney and the sympathetic chain is accessed in the retroperitoneum. The alternative more popular technique is to access the retroperitoneal space directly with balloon dissection in the retroperitoneum and then insufflation with carbon dioxide to create a pneumoretroperitoneum. The remainder of the dissection is then completed under visualization with a scope and the sympathetic ganglia are resected.

Chemical: Chemical sympathetic blockade requires accurate percutaneous needle placement adjacent to the sympathetic chain at the levels of the second through fourth lumbar vertebrae. The patient is placed in the lateral decubitus position, and the external anatomic landmarks of L_1 and intercristal line are used to triangulate the needle tip. Phenol and alcohol have been used for chemical sympathectomy. The technique has been described with the use of physical landmarks, under fluoroscopic guidance as well as CT guidance. It is obvious that precise placement of caustic chemical is paramount to prevent unwanted complications.

Results and Discussion

In reviewing the effectiveness of sympathectomy, we rely mainly on case series as there are no randomized studies. The indications for performing the procedure have changed over the last century. As medical therapy has improved and other surgical procedures that can address the primary cause of pain become popular, referrals for lumbar sympathectomy have decreased to the point that the only clear indications today are CRPS that cannot be medically controlled and severe hyperhidrosis. There is some benefit seen for patients with vasospastic disorders, non-bypassable atherosclerotic occlusion with rest pain and limited superficial ulceration, thromboangiitis obliterans, and vasospastic angiopathy.

There are three mechanisms by which sympathectomy can be effective. These are increased blood flow, alteration of pain impulse transmission, and decrease stimulation of sweat glands.

It is well recognized that under normal physiologic conditions, the lumbar sympathetic trunk provides resting vasomotor tone in the lower extremity arterioles. Lee et al. demonstrated 33–83 % increases in blood flow to lower limb skeletal muscle following lumbar sympathectomy in a canine model (Lee et al. 1987). Interruption of the lumbar sympathetic chain resulted in up to a threefold increase in capillary blood flow in pretibial skin in experiments by Moore and Hall (1973). What remains an ongoing controversy is the applicability of this physiologic response in the pathophysiologic state. Johnson et al. studied foot TcPO2 response to lumbar sympathectomy when focal ischemic necrosis is present (Johnson et al. 1998). They had 10 patients in the study of which only five healed their wounds. The patients who healed their wounds tended to be the ones with higher ABI but also had a sustained increase in TcPO2 10 days after the operation. The five patients whose wounds did not heal had a return of

TcPO2 to preoperative levels after 10 days. It is well known that when tissue is ischemic, it releases local mediators to dilate the arterioles locally and result in improved flow. In severe ischemia, these hormones should be acting to maximize blood flow to the limb, and it is less well understood if the addition of sympathectomy in a pathologic state will improve the blood flow even more than the local phenomenon.

On the clinical front, there are a multitude of studies that support the use of lumbar sympathectomy in patients with vascular disease. Baker and Lamerton analyzed a series of patients during a 5-year period with severe peripheral vascular disease who were not suitable for revascularization. They performed 132 lumbar sympathectomies on 118 patients. 45 % of those patients went on to have an amputation. The study shows resolution of rest pain in 86 % of patients and recovery from trophic changes in 64 % (Baker and Lamerton 1994).

Since there are no randomized studies and the resulting improvement after sympathectomy does not seem to be high, there has been some skepticism in regard to its effectiveness. The placebo effect is a well-known phenomenon, and there are arguments that the modest improvements seen are secondary to this. The reported relief from pain and superficial ulcer healing ranges from 28 % to 73 % (Collins et al. 1981; Persson et al. 1985). The literature supports the use of lumbar sympathectomy on patients with ABI greater than 0.3 and superficial ulcerations. These same patients are likely to have relief from their pain and healing of the ulceration with proper wound care in that time period. Randomized treatment with medication therapy has shown that these ulcerations will heal and the rest pain will resolve in 50 % of the patients getting placebo (Cronenwett et al. 1986).

Alteration of pain impulse or perception of such pain impulse can be seen after lumbar sympathectomy. Multiple studies show that there is significant improvement of pain with patients who have sympathetic mediated CRPS. Most of these retrospective trials put the early effectiveness of sympathectomy at 60–90 %. Bandyk et al. looked at 73 patients with CRPS who underwent sympathectomy. They showed that at 3 months, 90 % of patients had some level of pain relief. At 1 year, 25 % of patients had significant pain relief and another 50 % showed reduced levels of pain. The overall patient satisfaction at 1 year was 77 % (Bandyk et al. 2002). AbuRahma et al. looked at their 12-year experience with patients with CRPS. They found the patients' satisfactory outcome of 82 % early and 71 % late (>6 months) after sympathectomy (AbuRahma et al. 1994). Olcott and colleagues looked at 35 patients with CRPS who underwent sympathectomy and good results were obtained in 74 % of patients (Olcott et al. 1991). Hassantash et al. did a meta-analysis of 110 published reports totaling 1,528 cases of CRPS. Sympathetic blockade was noted in 88 % of these patients with a good response in 94 % of patients (Hassantash et al. 2003). Of note is the importance of evaluating these patients by injection of anesthetic in the sympathetic chain and showing improved symptoms. The patients who have a good response to the block have sympathetic mediated pain and should be offered the more radical operation.

Sympathectomy is often performed for palmar and upper extremity hyperhidrosis. There are many reports demonstrating good results after this procedure. They put the effectiveness of this procedure at 95–100 %. Lumbar sympathectomy can be performed for intolerable plantar and lower extremity hyperhidrosis. In certain patients, the excessive sweating of the foot results in maceration of the skin of the plantar aspect of the foot resulting in wounds. Rieger et al. performed a series of lumbar sympathectomies for lower extremity hyperhidrosis. Of the 90 patients who had this procedure performed, 87 had relief of their symptoms (97 %) (Rieger et al. 2009).

Complications

The procedure is fraught with a number of complications. The technical complications that can occur involve inadvertent injury to the ureter, genitofemoral nerve, lumbar veins and arteries, aorta, or vena cava. The ureter and genitofemoral veins are structures that travel vertically along the same direction as the sympathetic chain, and care must be taken not to confuse them with the

sympathetic chain. Lumbar veins and arteries cross over the sympathetic chain and should be carefully ligated if they are in the way. Special care should be taken not to compress a severely atherosclerotic aorta which may result in complete occlusion or embolization. It is important to pay special attention to the anatomic detail and also treat the vessels that are in the way with care to prevent injury or bleeding.

Post-sympathectomy neuralgia is a common complication of the procedure affecting up to 50 % of patients within a month after sympathectomy. This consists of pain in the anterolateral thigh that is not related to motion or activity. It is generally a dull aching that is amenable to treatment with medications. Although the pain remits in up to 12 weeks after the operation, the patients should be warned of this potential issue before the surgery (Kramis et al. 1996).

Sexual dysfunction in men can result if the L1 sympathetic ganglion is removed bilaterally. This is mainly in the form of retrograde ejaculation and affects up to 50 % of males with bilateral sympathectomy where L1 ganglion is removed (Quayle 1980).

The described mortality of the procedure in older series ranges from 3 % to 6 % (Palumbro and Lulu 1963; Taylor 1973); however, the more recent series report no deaths (Mockus et al. 1987; Persson et al. 1985). It should however be noted that the patient population who will get the procedure for arterial occlusive indications will have significant comorbidities. Evaluation of the cardiac and pulmonary system should be undertaken prior to this operation when deemed necessary.

Chemical sympathectomy shares many of the complications with open sympathectomy and has its own complications as well. It can result in injury to the ureter from phenol injection. The injections are not as precise in their placement. This will also lead to incomplete sympathectomy if the injection is placed away from the ganglion.

For patients who have lumbar sympathectomy for hyperhidrosis, compensatory hyperhidrosis can occur in other regions of the body. This complication can occur in up to 52 % of patients with sympathectomy but only about a quarter of those patients will have it to the point of dissatisfaction (Shelley and Florence 1960).

Conclusion

Lumbar sympathectomy has limited utility from a vascular surgical standpoint. The vascular surgeon should attempt to address the primary cause of ischemia and pain with medical therapy or revascularization if possible. There is evidence in the form of case series to indicate that lumbar sympathectomy helps with patients with arterial occlusive disease with rest pain or superficial ulcerations, thromboangiitis obliterans, and Raynaud's disease. The core of this therapy currently is directed at patients with CRPS and hyperhidrosis. It should however be kept in the armamentarium of a vascular surgeon for those intractable cases where revascularization and amputation are not good options.

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