CLINICAL APPLICATIONS

Spinal Cord Stimulation in Axial Low Back Pain: Solving the Dilemma

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ABSTRACT

Objective. To present the problem of producing stimulation paresthesia effects in the lower back dermatomes and to present some of the relevant outcome data using newer stimulation techniques, which suggest that this technique is efficacious in treating lower back pain.

Design. Review of clinical, physiological, and anatomical arguments regarding the use of spinal cord stimulation in lower back pain.

Conclusion. Use of modern stimulation techniques allows reliable production of paresthesia stimulation effects in the lower back dermatomes. Clinical studies have suggested that improvement of pain can be attained in about 50% of patients and held stable for up to 2 years.

Key Words. Stimulation; Spinal Cord; Low Back; Pain

Perhaps the “Holy Grail” of spinal cord stimulation (SCS) for the relief of pain is consistent and time-stable relief of axial low back pain. That this relief has been difficult to obtain has been associated with a number of anatomical and technical issues [1–7]. We are entering a time when reliable and predictable production of paresthesias in the region of the axial low back has begun to show that good to excellent relief of pain can be achieved in about half of the patients treated [8,9]. This success is due to the development of programmable, multicontact stimulation devices, and a growing use of patient interaction with the stimulation systems using computer interfaces [3,10–13]. The recent introduction of independent contact control allowing continuous electrical field adjustment and the shaping of the stimulation field has made the production of paresthesias overlapping the lower back even more predictable [14].

This article proposes to present the arguments about how we produce lower back paresthesia with SCS and why it has been so difficult to attain this goal. A brief review of the outcomes reported in the use of SCS in axial low back pain will also be presented.

Anatomy

In order to understand how we have gotten to the point of producing axial low back paresthesias, it is necessary to begin with some basic functional neuroanatomy and build the argument about how to apply SCS.

Recall that dermatomes are regions of the body where a person might feel sensation. These areas are related to the spinal column (the bony structures) and the spinal cord (the neural structures) (Figure 1). The spinal nerves carry nerve fibers mediating sensation of all types from receptors in their specific areas of the body. For example, the L5 dermatome is innervated by the L5 spinal nerve. Each spinal nerve is composed of a dorsal and a ventral root. The dorsal root (DR) mediates predominantly sensory information and the ventral...
Spinal Cord Stimulation in Back Pain

Figure 1 A representative dermatomal chart.

root motor functions. When exiting the spine, these roots merge to form the mixed (motor and sensory) spinal nerve.

The question for the physician who desires to use SCS becomes “what structure should I stimulate to produce a paresthesia in the area of pain?” Of course, the classical answer has been the dorsal columns (DCs) of the spinal cord. This has been the target because the DC has primarily sensory fibers and when electrically activated, the patient will feel predominantly sensory effects, tingling or paresthesias, not motor or autonomic effects. The DC contains a representation of all the body parts/dermatomes below the level of the lead [15]. These may be stimulated from a lead at that position. A not uncommon observation is a high cervical lead producing paresthesias in the legs and feet when activated.

However, the DRs that enter the DCs carry many types of sensory fibers, not only proprioception and vibration, but also pain and uncomfortably sharp paresthesias. High amplitudes of stimulation can evoke motor responses. DR stimulation itself will produce paresthesias only in the distribution of a single dermatome at the level stimulated. More diffuse paresthesias require interaction with the DC. Hence, if a lead is placed off the midline of the spinal cord at the T₉ vertebral level, a narrow band of paresthesia will be felt in the chest wall.

From the dermatomal map, the dermatomes we would like to cover with paresthesias for the axial low back seem to be predominantly L₂₋₅ (Figure 2). The most common teaching is to place the lead at the T₉ vertebral level if we want to produce lower back paresthesias. Why?

There are a number of important reasons why this should be so. Anatomically, the spinal cord is shorter than the vertebral column. The conus medullaris or tip of the spinal cord commonly terminates at the T₁₂ to L₁ vertebral level with the cauda equina of nerve roots extending caudad from that point. Also, throughout its length the spinal cord is not at a constant distance from the dorsal epidural space, where the lead is positioned. This is related to a number of factors. The normal curvature of the spine known as the lumbar and cervical lordosis and the thoracic kyphosis means the spinal cord will be closer to or farther away from the dorsal epidural space depending upon the vertebral level. One can visualize this by seeing the spine as curved but the spinal cord as straight hanging off the brain. The spinal cord itself does not have a constant diameter. There is a lumbar enlargement that is located in the lower thoracic vertebral level and a cervical enlargement in the neck. The diameter of the spinal cord is at its smallest in the thoracic vertebral areas above about T₇₋₈. These effects result in the dorsal cerebrospinal fluid space (dCSF) being larger relative to the position and diameter of the spinal cord in the mid-thoracic levels.

Where to put the lead to produce axial low back paresthesias is determined by the dermatomes to cover, the spinal segment to be stimulated and its relation to the bony vertebral segment, and the accessibility of the spinal cord to stimulation at a specific vertebral level. This latter problem is related to the size of the dCSF, and to the location of the nerve fibers to be stimulated within the DCs.

Functional Neuroanatomy

Dermatomes are the region of skin innervated by a given spinal nerve. The actual body location of a dermatome varies slightly from person to person (Figure 1). The low back dermatomes are in back of the trunk and in front of the legs. In general, low back is the rear trunk portion of L₂₋₅. For purposes of electrode placement, it is important to note that the spinal segment representing the lower back will be found approximately at the T₉ vertebral level due to the termination of the spinal cord at the T₁₂–L₁ level. So the vertebral level of the lead will not match the dermatomes that are desirable to stimulate. The spinal nerves enter the vertebral column at their vertebral level, and then travel up the vertebral column central canal to
their spinal cord segment. Figure 2 shows the spinal segment L₂ enters the cord around vertebral level T₁₁,₁₂.

As noted from our dermatome discussion, the low back dermatomes have a lot of anterior leg coverage. This is a key point in placing the initial lead in the operating room at T₉. At this level, the DC mapping or somatotopography allows access from the L₂ dermatome downward and avoids high resistance due to a large dCSF, while preferentially stimulating the DCs instead of the DRs. The DRs have a lower threshold for stimulation as they sit within the excellent electrical conductor of cerebrospinal fluid space, while the DCs have conductivity similar to fat.

At any spinal segment, fibers from dermatomes below that segment proceed through the DCs. The fibers enter the spinal cord at their spinal segment. The sensory fibers then move into the DCs from the side. As they ascend in a cephalad direction, they travel toward the midline. It is as though they are pushed toward the center by the newer fibers entering (Figure 3). So, when do L₂

Figure 2 Note the dermatomal representation of the lower back L₂–₅. Also note the entry point of the spinal nerves into the spine at their associated vertebral level but entry into the spinal cord at a much higher vertebral level.

Figure 3 The location of the nerve fibers representing sacral, lumbar, and thoracic areas. Shading indicates the location of the L₂ dermatomal representation.
fibers become most accessible for stimulation? Clearly at the L1 level, the fibers are located too deep within the DC and too lateral. At T5, the dCSF to spinal cord volume ratio is about as large as it will get resulting in predominantly DR, anterior chest stimulation. So to stimulate the L2 dermatome, the compromise level would be on the average the T9 vertebral segment. At this level, there is good access to the fibers from a midline lead position, and the dCSF is reasonable at 3–4 mm, so DC stimulation can be achieved without DR stimulation.

Why do DR fibers have a lower stimulation threshold than DC fibers? The DR fibers have a larger diameter, are located in the cerebrospinal fluid space where most of the stimulation current is found, and are curved, which makes them easier to stimulate. These facts force us to place our lead(s) as close to the midline as possible. That way, the contacts are closer to the DC fibers than the DR fibers and we get more stimulation where we need it.

Another anatomical problem in producing desired low back stimulation effects is the composition of the DCs themselves. The DCs are made up of white matter, which has very poor electrical conductivity, similar to fat. In order to obtain much penetration of current into the DC, the amplitude must be turned way up. In doing so, most of the current runs through the excellently conducting cerebrospinal fluid space off to the sides around the DR. By the time the top 0.5 mm of the DC has been recruited, the nearby DR fibers have been completely stimulated. Clinically, the patient feels perhaps back and anterior leg stimulation (L2), but now the abdomen starts cramping (T9 dermatome).

So what about stimulating the low back? There are several hypothetical reasons for why it is a challenging target. The representation of the lower back in the brain is small as represented by the known sensory homunculus. The lower back is a small body area relative to the legs and, finally, there is an even distribution of fibers in the DCs.

In the sensory cortex of the brain, the amount of nervous tissue dedicated to processing sensory information from a particular body region is represented from medial to lateral (Figure 4). It is a functional map. In general, the more important the function is to survival of the organism, the more nervous tissue will be dedicated to it. So how much is dedicated to the leg? How much is dedicated to the back? The back is a lot less than the leg. It seems reasonable that there will be fewer fibers innervating the low back. Additionally, the L2–5 dermatomes are made up of anterior legs and back. From a percent body surface standpoint, the anterior legs make up 80% of the area represented by these dermatomes. If the receptive fields per nerve are equal, then there would be at least four times fewer fibers for back coverage than leg coverage. Finally, within the “slice” of DC that the L2 dermatome represents, it makes sense to think that the leg fibers and back fibers would be evenly spread out in the slice. That is, a low back fiber is just as likely to be on top of the DCs as at the bottom (Figure 5). So low back fibers are not especially important from a functional standpoint and there are few of them. Low back fibers do not cover much of the body surface area relative to the legs with which they share dermatomes, so there

Figure 4 The sensory homunculus. Note the small amount of area devoted to the low back when compared with the legs.

Figure 5 The representation of the L2 leg and low back dorsal column fibers.
are fewer low back fibers. Low back fibers are not especially available for stimulation in the DCs. That is, it may be hard to reach enough of them to get good coverage.

In order to stimulate low back fibers, we have to penetrate the stimulation field deeper in the DC to reach the rare low back fibers without overly stimulating the DRs. With precise placement of a single lead on the physiologic midline of the spinal cord, or with dual leads placed closely to the right and left of the midline, often using multicontact programming, these fibers have now become accessible [9]. Axial paresthesia production has been made even more reliable with the development of independent current control on every lead contact. This keeps the electrical field best confined away from the DR. Utilizing wide pulse width, which recruit more within the stimulation field, allows an increase in the field reach. The ability to stimulate with the cathodal or negative field anywhere along the lead length allows to find the optimum stimulation location more precisely.

Results

The ability to produce paresthesia overlap in the lower back begs the question of pain control. Does SCS produce relief of axial low back pain?

Two retrospective case studies supported the application of SCS in low back pain. Van Buyten et al. reported 17 patients with failed back surgery syndrome having back and leg pain treated with 2 × 4 contact leads had significantly improved global back and leg pain as well as reduced medication usage [16]. Ohmeiss and Rashbaum reported outcomes in 41 patients with failed back surgery syndrome and found that 60% were improved and 75% would undergo the treatment again for the same result [17].

Barolat et al. reported a prospective study using a large 16-contact paddle style lead to treat 44 patients with predominantly axial low back pain [8]. To enter the study, patients needed a greater than 50% back pain component unresponsive to conventional treatments for the prior 12 months. Conventional treatments included physical therapy, epidural steroid injections, anesthetic injections, narcotics, psychological counseling, non-narcotic medications, biofeedback, and relaxation techniques. In this study, leg pain relief exceeded back pain relief at 6 and 12 months. The trend at 12 months was for leg pain relief to be more stable than for axial back pain.

North et al. recently reported a prospective controlled trial comparing dual with single percutaneous leads in patients with axial low back pain in failed back surgery syndrome [9]. At 2-year follow-up, 53% of the 20 patients in the study were considered a clinical success. In their hands, single leads provided significant advantages in paresthesia coverage, and amplitude requirements while maintaining a stable low back paresthesia over time. Dual lead systems, however, were also successful in a majority of patients with low back pain.

In both of the prospective studies, the relief of the neuropathic leg pain component of the pain remained the most stable over time. In the North et al. study, axial low back pain relief decreased from 50% to 46% of patients at 2 years [9]. Radicular lower extremity pain actually improved from 88% to 92% of patients. In the Barolat et al. study, back pain relief decreased from 82.7% to 68.8% over 1 year while leg pain relief declined from 91.6 to 88.2% in the same time period [8].

With the limited number of prospective trials and only one randomized prospective trial of SCS for the treatment of the failed back surgery syndrome, more validation of the technique is required. At present, there is a multicenter trial in the United States being conducted to study predominantly low back pain relief with SCS, but it is too preliminary to report outcomes. Hopefully, the PROCESS study in Europe and Australia will have some data concerning relief of lower back pain although the primary inclusion criterion is neuropathic leg pain [18].

References