

Theoretical Aspects of Pain: Bizarre Pain Phenomena During Low Spinal Anesthesia

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A KNOWLEDGE of peripheral and spinal sensory pathways is necessary for an appreciation of the sensory changes observed during spinal anesthesia. With this basic information, a rational explanation usually can be found for the successes as well as the failures of spinal anesthesia. At times, however, certain bizarre pain phenomena of the lower extremity are encountered during low spinal block (to a dermatomal level of T-10 or lower) which cannot be satisfactorily explained on the basis of current concepts of anatomical pathways of pain.

From a correlation of scattered physiological, anatomical and clinical observations, the authors present the thesis that pain arising from deeper structures of the lower extremity is transmitted along amyelinated nerve fibers, which first pass through the sympathetic trunks before they terminate in the dorsal root ganglia. Furthermore, such peripheral sympathetic afferent fibers may traverse the sympathetic trunks for quite a distance, and ultimately terminate in the spinal cord cephalad to the level of a low spinal block.

Based on this assumption a meaningful explanation is provided for the occurrence of tourniquet pain, pain on transection of the sciatic nerve and recurrence of phantom limb pain or causalgic pain during an otherwise adequate low spinal block.

Relation of Nerve Fiber Size and Conduction Velocity to Function

Improved electronic instrumentation led to the famous and still current classification by Erlanger and Gasser in 1924¹ of nerve fibers, according to their conduction velocity, into three groups, A, B and C. A-fibers are myelinated somatic fibers of 22-1 μ diameter with conduction speeds of 120-5 meters/second.

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They are further subdivided into α , β , γ and δ groups according to decreasing conduction velocity and diameter.² It was established that the conduction velocity of myelinated fibers, in meters per second is approximately equal to six times the diameter of the axon in micra.³ By this means the size of a fiber is easily calculated from its conduction velocity *in vivo*, without the need for cumbersome histological studies.

B-fibers are thinly myelinated preganglionic autonomic fibers and are not involved in the problems under discussion. C-fibers are amyelinated, small (1.5-1 μ diameter) and slow conducting (2.5-0.5 meter/second) fibers.

Stimulating a nerve, while simultaneously measuring the conduction velocities of its fiber components and observing the patient's interpretation of the stimulation, allows correlation between the size of a nerve fiber and sensory modality.⁴ By means of this technique it has been found that pain is mediated along two different groups of fibers⁵⁻¹³: (a) myelinated A- δ fibers of 6-3 μ diameter, conducting at 40-15 meters/second, found in cutaneous nerves ("fast pain") and (b) amyelinated C-fibers of 1-0.5 μ diameter, conducting at 2.5-0.5 meter/second, terminating in free nerve endings ("slow pain").

Striking physiological and anatomical differences exist between these two groups, which have given rise to the concept of duality of pain.¹¹ The difference in conduction velocities, for example, causes impulses in the A- δ fibers to arrive at the spinal cord first.⁹ When the C impulses arrive, the cord neurons are already in a refractory state, leading in effect to a partial block of slower impulses by earlier arriving fast impulses.¹⁴ Slow pain from the extremities therefore is not clearly differentiated.* When the fast pain component is differentially blocked, however, as can be done with an extremity tourniquet^{4, 15} the late arriving, slow

* When fast pain fibers are simultaneously activated.

impulses will pass through the spinal cord without interruption and are then consciously manifested.

Pain Fibers in the Sympathetic Trunks

Although familiar with the sometimes dramatic relief of pain following a sympathetic block, physicians still hesitate to accept the concept that pain fibers are present in the sympathetic trunks. Nevertheless, isolated bits of information and an occasional case of unusual pain distribution provide good evidence that some pain sensation must be transmitted along the sympathetic trunks. Bonica¹⁶ states (in discussing causalgia) that the presence of afferent sympathetic fibers would explain the pain and its relief by sympathectomy, “. . . but unfortunately no positive proof has been brought forth to demonstrate their existence . . .”

Efferent preganglionic sympathetic fibers of the thoracolumbar outflow arise in the intermedio-lateral columns of the spinal cord from the first thoracic through the second lumbar segments, to innervate the body literally from head to toes. Because of their limited origin and widespread distribution they traverse the sympathetic trunks diffusely and often for a considerable distance. Some synapse in the trunks, others merely pass through it.¹⁷ Sympathetic afferent fibers innervating the viscera are also well known. They give rise to visceral sensations and autonomic reflexes. Besides these visceral sympathetic afferent fibers, there are also other small fibers (as yet little studied) whose cell bodies are located in the spinal dorsal root ganglia. Their termination is less apparent, but increasing evidence supports the view that they are peripheral fibers.¹⁸⁻²³ They leave a spinal nerve along its gray ramus to the sympathetic trunk, thence travel to a dorsal root ganglion but not necessarily at the same level. Every spinal nerve is connected to the sympathetic trunk via a gray ramus; a peripheral sympathetic sensory fiber therefore may traverse the trunk for some distance before it enters the cord at a higher level, rather than enter the cord directly with the peripheral somatic nerve of the corresponding segment.²⁴ It is likely that these peripheral sympathetic afferent fibers are C-fibers, which carry some of the unpleasant

sensations associated with causalgia, phantom-limb pain, tourniquet pain, direct nerve manipulation, and others.^{17, 23-27} Because of their diffuse spread, the associated pain is poorly localized. Furthermore, in the normal person, impulses conducted along these slow fibers are partially inhibited by earlier arriving impulses from faster conducting myelinated pain fibers.¹⁴ Only when these latter inhibiting influences are decreased or removed, as for example by spinal block, are impulses from peripheral sympathetic afferents transmitted freely to the central nervous system.

Kuntz²¹ showed in the cat, and also later in man, that peripheral afferent fibers *did* traverse the sympathetic trunks, to enter the spinal cord via white communicating rami. These fibers could be demonstrated arising from the upper as well as lower limbs. Their distribution is very probably in relation to blood vessels or other deep structures, rather than to cutaneous pain fibers.^{20, 21, 23, 24, 27, 28}

Kuntz¹⁷ found in man that stimulation of the lumbar sympathetic chain caused severe pain, even after section of the corresponding white ramus. This was also observed by Kiaer,²⁰ who found that stimulation of the first lumbar ganglion could cause pain referred to the groin and back, and that sympathetic block would relieve pain arising from stimulation of the head of the femur. Kuntz²⁴ additionally stressed the fact that patients with complete transection of the lower cord were paralyzed and had cutaneous analgesia of the legs, but that deep, burning, causalgic pain was often still present, which could be relieved only by sympathectomy.

For the lower extremity, spinal ganglia from probably the mid-thoracic to mid-lumbar level send fibers through corresponding white rami to the sympathetic trunk.^{21, 24} These fibers descend, apparently without synapsing, through the trunk, to lower levels and join the spinal nerves to the lower extremity via the corresponding gray rami (sympathetic roots). Although afferent fibers in gray rami are relatively sparse, they are of sufficient number to be functionally significant. They obviously do not belong to the usual peripheral afferent conductors, but are probably related to vessels or other deep structures.

Threadgill²³ was impressed by the fact that

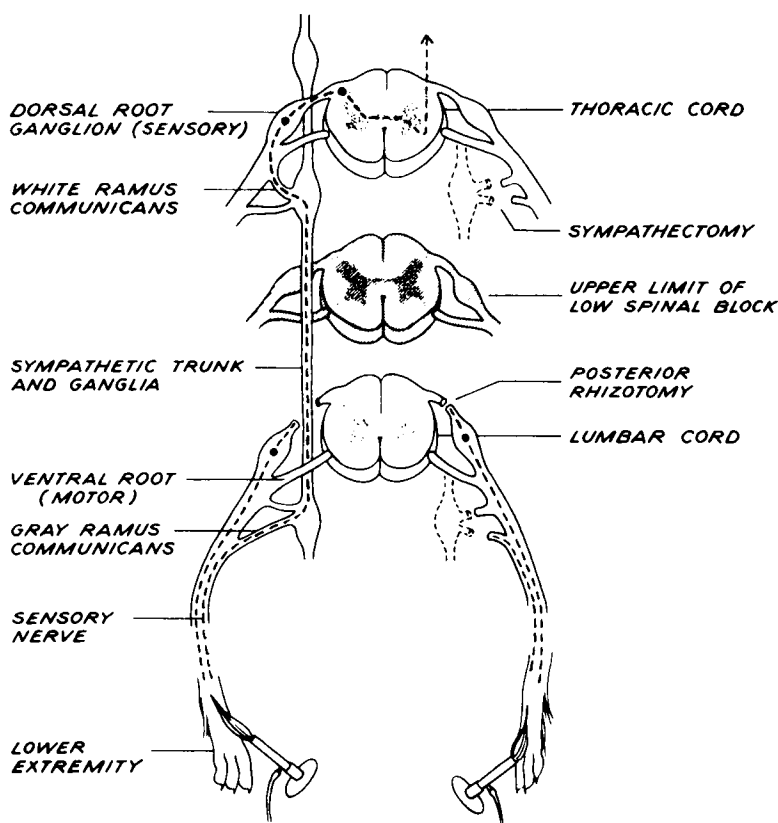


FIG. 1. Ascending pain pathways in the sympathetic trunk. Following bilateral dorsal rhizotomy of the lumbar dorsal root to the foot, sharp pain was no longer perceived. A response to severe pain (burning) was still present, however, on the non-sympathectomized side (left). Following lumbar sympathectomy the foot became totally analgesic (right).

Note that paraspinal pain pathways may enter the cord *above* the level of sensory block and thus are not blocked by low spinal analgesia.

Modified by publisher's permission from Threadgill, F. D.: Afferent conduction via the sympathetic ganglia innervating the extremities, *Surgery* 21:570, 1947.

in patients with causalgia, sympathectomy relieved the pain, but did not increase the peripheral blood flow. To prove his thesis that peripheral *afferent* sympathetic fibers exist, he performed a bilateral posterior root section of L-5. Although the animal became analgesic to pin-prick, it still responded to a severe stimulus such as burning. Following unilateral sympathectomy, this response to severe stimulation was abolished on the operated side (see figure 1). Freeman *et al.*¹⁹ in analogous experiments using plethysmographic indices of vascular activity, also drew the conclusion that peripheral *afferent* fibers, related to vessels, were present in the sympathetic trunks. From an observation that the painful sensation to cold is greatly reduced following sympathectomy, Hyndman²⁶ also postulated the presence of peripheral sympathetic *afferents* for pain. He substantiated his theory by observations on patients with complete spinal cord transection at the second lumbar segment who were completely paralyzed and analgesic to cutaneous

stimuli of the leg. Nevertheless, inflation of a leg tourniquet, crushing the foot or immersing the foot in ice water gave rise to a stinging or burning sensation, which slowly disappeared. Since the sweating mechanism was still functioning, indicating an intact sympathetic supply, it seemed apparent that the vague, poorly localized burning pain was transmitted by peripheral afferent sympathetic fibers.

Noordenbos,¹⁴ a neurosurgeon with considerable experience in the use of chordotomy for the relief of intractable pain, was impressed by the high incidence of incomplete pain relief following this operation and also attributed the inadequacy of chordotomy, in treatment of phantom pain and causalgia, to peripheral sympathetic afferent fibers.

The above presented theoretical considerations of duality of pain fibers and the presence of myelinated peripheral afferent fibers in the sympathetic trunks enable us to explain the occurrence of bizarre pain phenomena during low spinal block.

Tourniquet Pain

Tourniquet pain may be encountered during spinal anesthesia for lower extremity surgery, but it has always been difficult to explain satisfactorily when adequate cutaneous analgesia is present. After a pneumatic tourniquet has been applied on the thigh for 45 to 60 minutes the patient may complain of a vague dull ache in the leg and become restless. The cutaneous level of analgesia at the time is above the level of the tourniquet. Release of the tourniquet is followed by immediate and complete relief. If the tourniquet cannot be released, analgesics or general anesthesia or both are often required to keep the patient comfortable.*

The incidence of tourniquet pain is inadequately documented. Egbert and Deas²⁹ observed 35 of 55 patients under spinal anesthesia (63.6 per cent) with tourniquet pain, but reduced the incidence to 33.3 per cent (17 of 51 patients) by increasing the dose of spinal anesthetic drug. An incidence of tourniquet pain in even one out of three patients is, however, higher than we have seen, and would certainly have been commented on before by others. The fact that no other statistics are available probably attests to its relative rarity. Greene³⁰ and Cole³¹ mention its occurrence but give no figures. Egbert and Deas²⁹ believe that tourniquet pressure-pain is carried by fibers larger than those for cutaneous pain sensation (the A- δ fibers). This seems difficult to accept in light of the above discussed relation of fiber size to sensory modality.

Limb compression by the tourniquet will block nerve conduction in such a manner that larger sensory fibers cease to conduct impulses before smaller ones. After 30 minutes of compression, conduction in large afferent fibers within the compressed area has ceased completely.^{8, 15, 32, 33} Even if large fibers were responsible for tourniquet pain, they would already have been effectively blocked by compression at the very time that tourniquet pain usually has its onset (*e.g.*, after 45–60 minutes).

* This syndrome may also be observed during general anesthesia, where prolonged tourniquet inflation occasionally has given rise to increased sympathetic activity, manifested by a gradual rise in blood pressure and pulse, cardiac irregularities, sweating and vasoconstriction.

Cole³¹ noted the sympathetic aspects of tourniquet pain and believed it due to compression or ischemia of the sciatic nerve and of sufficient intensity to "penetrate" the spinal block. Greene³⁰ states ". . . the phenomenon of pain due to a tourniquet in an otherwise satisfactory spinal anesthesia remains to be completely explained. . . ." Dr. Louis W. Lewis³⁴ cites an unusual case. A patient for bilateral triple arthrodesis of the ankle received a unilateral lumbar sympathetic phenol block preoperatively. Spinal anesthesia was given to a satisfactory level and bilateral thigh tourniquets were inflated simultaneously. The patient experienced tourniquet pain after about one hour, but *only* on the unblocked side!

It is interesting to note the similarity in sensation and in time of onset between tourniquet pain under spinal anesthesia and tourniquet pain in the unanesthetized experimental subject. During compression, nerve fibers are blocked according to size. The largest fibers are rapidly blocked, smaller ones later, in order of decreasing diameter.^{2, 4, 6, 8, 15, 32, 33, 35} Decrease in sensation occurs within a few minutes after compression; numbness is noted after 15–20 minutes. Then suddenly, after about 30 minutes, the numbness changes to a very unpleasant "sickening" sensation, at a time when the A- δ fibers ("fast" pain fibers) are just blocked. This implicates myelinated C-fibers ("slow" pain fibers) as being responsible for tourniquet pain. After release of compression, sensation rapidly returns to normal. Bishop²¹ confirmed that in man after 25–40 minutes of tourniquet compression of the arm, touch and pricking pain are lost distal to the tourniquet.

Why the progressive and differential block of nerve fibers occurs during tourniquet compression has not yet been settled. Magladery and his group³⁵ give convincing evidence that the effects are due to anoxia of the nerve; large fibers are more sensitive to oxygen lack than smaller fibers. Weddell³⁶ on the other hand believes the effect due to direct compression of the nerve. Certainly prolonged compression of a nerve may lead to temporary or even permanent damage to the portion of nerve under the tourniquet, as evidenced by the "tourniquet paralysis syndrome."^{36, 37}

It may be postulated therefore that tourni-

quet pain during spinal anesthesia is due to impulses arising in C-fibers which traverse the sympathetic trunks, before entering the spinal cord above the level of analgesia.

Pain from Direct Stimulation of the Sciatic Nerve

We have noted on several occasions, during an otherwise satisfactory and solid spinal block for leg amputation, that clamping, cutting and ligation of the sciatic nerve was quite uncomfortable to the patient. Cessation of the stimulus led to immediate cessation of pain. Infiltration of the nerve with a local anesthetic abolished further discomfort. Other anesthetists have also noted this curious phenomenon, yet we have found no report of it in the literature. This effect seems to be associated particularly with trauma to the sciatic nerve. During amputation under low spinal block other nerves are also divided, but without apparent discomfort.

It again appears in this instance that certain types of pain are transmitted centrally around a completely blocked section of spinal cord, since it is unlikely that even severe pain can "break through" a block. Furthermore, as described above, fibers larger than A-delta size have never been shown to transmit pain. Only when the usual pain pathways are interrupted with a spinal block, chordotomy or transection of the cord, do these diffuse extra-spinal pathways come to our attention. It is clinically apparent that only an extremely severe stimulus is so manifested, however. The fact that thousands and thousands of satisfactory spinal anesthetics have been given for lower extremity surgery with only a few instances of bizarre pain on record, attests to its rarity.

Causalgia and Phantom Limb Pain

Causalgia is a painful state occasionally seen following peripheral nerve trauma, especially of the lower extremity. It presents an instructive example of "slow" pain, related to the sympathetic system, which becomes altered following spinal block. The vascular and trophic changes associated with causalgia are probably due to reflex sympathetic efferent activity. The pain of causalgia, at least in its early stages, is characteristically relieved by block of the corresponding sympathetic ganglia

or by sympathectomy.^{16,38,39} Gerard³⁸ pointed out that the pain of causalgia may still be present even in patients with complete transection of the lumbar spinal cord; and called attention to sporadically reported similar observations of return of severe causalgic pain during low spinal anesthesia. Interestingly, the pain disappeared again after the block had worn off or if the dermatomal level of the block was raised to mid-thoracic levels.⁴⁰ It has been observed that stimulation of the lumbar sympathetic chain gives rise to pain.^{22,23} Such pain is still perceived even after complete transection of the cord, and is only abolished following sympathectomy.³²

Even though sympathectomy for causalgia does not improve circulation of the affected limb, as indicated by measurement of skin temperature and plethysmography, the pain disappears.^{23,26} This finding again suggests the presence of certain peripheral pain fibers in the sympathetic trunks. Bonica¹⁶ points out that causalgia, phantom limb pain, and several other pain syndromes, are characterized by a triad of symptoms consisting of: pain, which is diffuse, "visceral" and difficult to treat, vasomotor disturbances, and sweating and trophic changes in the affected area. As a fourth characteristic, Bonica includes response to sympathetic nerve interruption.

Significant conclusions about sympathetic pain fibers may be drawn from reports following the operation of anterolateral chordotomy for intractable pain. Pain (and temperature) fibers cross over in the spinal cord to run cephalad in the spinothalamic tract. Interruption of this tract by a surgical incision a few segments above the affected area should therefore provide complete and permanent relief of pain distal to the incision. However, following unilateral chordotomy, a causalgic syndrome may remain which is sometimes more severe than the original pain,¹⁴ while at other times pain following deep stimulation is still perceived but is referred to other areas of the body.²²

Phantom limb, the sensation that an amputated limb is still present, is seen following most amputations (90-97 per cent).¹⁶ However, pain in the phantom limb is seen in only 10 per cent of amputees, accompanied by signs of sympathetic hyperactivity such as vasoconstriction and sweating. Sympathetic

trunk interruption, done early and properly, appears to be the treatment of choice for this condition also.¹⁶

Since sympathetic nerves, like the peripheral nerves, enter and leave the spinal cord, low spinal anesthesia should completely interrupt *all* pain impulses arising from the lower extremity including those from the sympathetics. It is disturbing that this simple explanation does not always appear to hold true. There are occasional reports that severe phantom limb pain may recur during spinal anesthesia, disappearing only when the sensory block has dissipated.^{16, 38, 40, 41, 42} Leatherdale⁴² reports one such case with a cutaneous analgesia level to T-10, and cites two other cases. Arrowood and Sarnoff⁴⁰ studied seven patients with intractable amputation stump pain or phantom limb pain. They differentially blocked pain and sympathetic fibers with a continuous subarachnoid drip of 0.2 per cent procaine, leaving motor, touch and proprioceptive fibers intact. Five of seven patients obtained relief. But one patient suffered a severe exacerbation of his pain with a level of analgesia at T-10. Not until high spinal anesthesia was achieved did he get relief of his pain. Bonica¹⁶ presents four cases of recurrence of phantom limb pain during spinal anesthesia. One of these is significant in that pain recurred even with a level to the eighth thoracic segment, and only disappeared following sympathetic block. Dr. Louis W. Lewis³⁴ reports a case with recurrence of phantom-limb pain during spinal anesthesia which was completely relieved following ipsilateral sympathetic block. One of us has observed a case where a previous amputee developed extremely severe pain in the phantom within two minutes following induction of spinal anesthesia to a cutaneous level at the eleventh thoracic segment. Tilting the operating table caused the anesthetic to rise, until with the level at the sixth thoracic, the pain disappeared.

From such observations it may be concluded again that not all impulses are carried directly to the spinal cord by spinal nerves. Instead some must first enter the sympathetic trunks and may travel several segments cephalad in the trunk before reaching the cord. Raising the level of a spinal block would interrupt the sympathetic afferents entering at the higher level. Fortunately, a cutaneous level of anal-

gesia to the tenth thoracic segment (umbilicus) provides adequate spinal anesthesia for the vast majority of lower extremity operations. Only very rarely does one get an inkling that some pain fibers ascend higher than the tenth thoracic along the sympathetic trunk and enter the cord cephalad to the block.

Discussion

The presence of causalgic type pain following apparently complete interruption of pain pathways by low spinal block, transection of the lumbar spinal cord or after surgical division of the spinothalamic tracts (anterolateral chordotomy) has been variously explained, but is far from solved. Observations of similar pain patterns occasionally seen during spinal anesthesia, which are relieved by lumbar sympathetic block, add to a mounting body of evidence that certain peripheral pain fibers may run in the sympathetic trunks (paraspinal pathways). Pain is difficult to study experimentally in animals, and even more difficult to extrapolate to man. Yet the anatomical, physiological and clinical evidence cited leaves little doubt that peripheral afferent fibers transmitting "slow" pain from the extremities travel along paraspinal pathways found in the sympathetic trunks.

Sympathetic afferent nerves reach the extremities via the mixed spinal nerves, along gray communicating rami arising from the sympathetic chain. They enter the spinal cord along white communicating rami of the thoraco-lumbar autonomic division. It is apparent from the clinical success of low spinal anesthesia (cutaneous level at or below the tenth thoracic segment) for lower extremity surgery that the great majority of peripheral sympathetic afferents must enter the cord below this segmental level of block. But apparently some afferent pain fibers from the lower extremity may enter the spinal cord along paraspinal pathways at as high as thoracic segmental levels (fig. 1).

Bizarre spinal pain is compatible with stimuli transmitted by small, myelinated, slow-conducting C-fibers. Impulses along this system are partially inhibited, or balanced, by earlier arriving "fast" pain impulses from myelinated A- δ fibers. Only when this balance is disturbed, either by the arrival of fewer "fast"

or more "slow" impulses, is "slow" or protopathic pain consciously manifested.

During spinal anesthesia, the slow pain impulses that do arrive above the level of block are ordinarily inhibited by fast cutaneous impulses entering at that level, so that the patient is unaware of their presence. During direct nerve manipulation, however, there is a tremendous increase in impulses from synchronously firing slow fibers, such that they outnumber the inhibition by fast fibers and are perceived consciously. During tourniquet compression, direct compression of nerve trunks and the added stimulus of nerve ischemia will cause increased firing of all nerves. Initially this increase will be in both fast and slow fibers, so that the inhibitory balance is not disturbed, *e.g.*, there is no change in pain patterns. But since large fibers are much more sensitive to compression and ischemia than small (pain-carrying) fibers they will be progressively blocked and fall out, leaving only slower fiber groups, which as seen are being increasingly stimulated. Because of the reduction in inhibition by fast fibers and increase in discharge from slow fibers, this slow pain may then reach consciousness.

Causalgia and phantom limb pain are often associated with incompletely regenerated nerves, stump neuromas, or artificial synapses.⁴³ In these areas a predominance of small over large fibers is found, leading to reduced fast fiber inhibition. After a low spinal block, this balance may be even more disturbed since the remaining fast pain fibers follow dermatomal segmental distribution and become blocked, leading to increased passage of slow impulses along the sympathetic paraspinal pathways to levels above the block and therefore perception of (causalgic) pain.

Summary

The perplexing observation that occasionally pain still may be perceived in an extremity that to all appearances is completely analgesic following solid spinal block, is discussed. Such pain may occur almost immediately following spinal block in patients suffering from causalgia or phantom limb. It may occur after a remarkably constant time interval of tourniquet compression. And finally it may be noted only when a large nerve trunk, such as the sciatic nerve is manipulated. Although of

seemingly different origin, the pain pattern is remarkably similar under all these circumstances. It is a deep, burning, poorly localized, diffuse and uncomfortable ache. This response characterizes the pain as being transmitted by small, slow, myelinated C-fibers. With an adequate cutaneous level of spinal analgesia, complete interruption of all pain-carrying fibers below this level might be expected. However, since pain under certain uncommon circumstances still can be perceived, it seems most probable that some pain fibers enter the cord above the level of block. Laboratory and clinical evidence strongly points to the presence of peripheral afferent myelinated pain-carrying fibers from the extremities in the sympathetic trunks. Such fibers use the trunks (the "paraspinal pathways") to gain access to the spinal cord at a more rostral segmental level via white communicating rami. These fibers are ordinarily blocked by impulses from faster fiber groups and are therefore not consciously perceived. Only when the faster fibers are blocked, as by tourniquet compression, or when all slow fibers are stimulated simultaneously, as by direct manipulation of the sciatic nerve, is their message able to gain access to the central nervous system.

Under these circumstances a lumbar sympathetic block or a raised spinal segmental level may provide complete relief of pain.

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