Autonomic Responses to Spinal Pain

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ABSTRACT: On a broad scale, relief from current pain is probably the single most important consideration for our patients. However, physical pain is not the only component of a patient’s suffering, and sometimes other aspects of a spinal pain syndrome are of equal or greater clinical importance. Pain in general, and perhaps spinal pain in particular, is capable of eliciting changes in visceral function which can be distressing and even dangerous. There might be various mechanisms through which such functional changes might occur, but the somatoautonomic reflex is probably the best understood. This paper will first discuss the mechanism of the somatoautonomic reflex and then examine whether evidence supports an important role for this phenomenon in spinal pain syndromes.

Key words: autonomic nervous system, pain, spinovisceral reflex

Observations from Basic Physiological Studies

Both everyday experience and basic scientific studies demonstrate that pain is capable of activating the autonomic nervous system and so bringing about changes in organ function. Early experimentation of this sort frequently used noxious stimulation (the better to elicit consistent results), applied to limb tissues (which were easily accessible), to elicit changes in heart rate and blood pressure (which were easily measured). Very few studies have examined the effects of stimulation of spinal tissues, especially the effects of innocuous stimulation. Most experimental models have utilized anesthetized animals in order to eliminate the influence of emotional factors. These aspects of experimental design have been essential to successful investigation of somatoautonomic phenomena, and led to the development of a model of autonomic response to noxious stimulation generally attributed to Walter Cannon and characterized as “fight or flight”. The essential elements of the model were that noxious stimulation applied to any tissue would elicit a generalized response mediated by the brain.

In early investigations, it had frequently been observed that transection of the cervical spinal cord eliminated somatosympathetic reflex discharges. Consequently it was assumed, by Cannon and others, that these reflexes were mediated at the supraspinal level. Later, however, Beacham and Perl were able to demonstrate somatosympathetic reflex discharges of spinal origin. Since then, many investigators have confirmed the existence of both spinal and supraspinal...
reflex centers. Kimura et al. 5) demonstrated that in CNS-intact anesthetized rats, noxious mechanical stimulation of the skin elicits significant responses in heart rate (Fig. 1). Pinching virtually anywhere produced some response. However, there was a segmental tendency, with the strongest responses coming approximately equally from stimulation of the hindpaws or forepaws. In spinalized animals the segmental tendency was altered but exaggerated. Thus, in spinalized animals hindpaw and forepaw stimulation still gave significant but relatively weak responses, while stimulation in the thoracolumbar region produced much-enhanced reflexes. Furthermore, and quite interestingly, stimulation on the right side gave a significantly greater response that stimulation on the left side. In contrast then to the Cannon model, there is clear evidence of spinal reflex centers which mediate segmentally organized responses.

In general, it has been found that natural stimulation of nociceptors or electrical stimulation sufficient to recruit unmyelinated C-fibers have been most effective in eliciting consistent somatoautonomic reflex responses6). Reflex effects have been demonstrated throughout the cardiovascular system, in the digestive system, urinary system, endocrine system and immune system1). In anesthetized animals, innocuous stimulation produces weak, inconsistent responses or no reflex at all. In particular, it has generally been shown that stimulation of group Ia fibers (from muscle spindles) or group Ib fibers (from Golgi tendon organs) has virtually no effect on autonomic nervous system activity or visceral function7). For example, in anesthetized cats, it has been shown that movement of the knee joint within its normal physiological range has no effect on blood pressure or heart rate8). However, forced movement beyond the normal physiological range produces significant increases in these parameters (Fig. 2). Furthermore, in the acutely inflamed joint, these responses are greatly exaggerated. In fact, in the inflamed joint, even movement within the normal range produces reflex increases in blood pressure and heart rate. Similar observations abound: noxious stimulation elicits clear and consistent autonomic responses; innocuous stimulation elicits weak and inconsistent responses or none at all.

Basic physiological studies involving stimulation of peripheral tissues in anesthetized animals therefore provide only partial support for the view that spinal dysfunction may impact autonomic function. Segmentally organized spinal reflexes have been demonstrated, but only consistently in response to noxious stimulation.

The modern physiological investigations of the impact of somatosensory input on autonomic functions have been reviewed in a very comprehensive monograph by Sato, Sato and Schmidt 1). Of the approximately 750 basic scientific
papers which they cited, however, only 3 made reference to spinal stimulation. In the past, therefore, we have been compelled to look at the effects of, for example, limb joint or skin stimulation, and extrapolate these to the spine.

It is not unreasonable to think that axial tissues may differ in innervation from more peripheral tissues, or that sensory input from axial tissues might elicit distinct reflex responses. A single study conducted by Drs. Sato and Swenson investigated the effects of mechanical stimulation of the spine on blood pressure, heart rate and renal sympathetic nerve activity (Fig. 3). The application of lateral stress to the lower lumbar or lower thoracic spine produced changes in the monitored parameters and these changes outlasted the length of stimulation. The results were clearly shown to be the result of activation of spinal afferents. However it is unclear whether the forces applied, from 0.5 to 3.0 kg, should be characterized as noxious or innocuous.

More recent studies have employed noxious chemical
stimulation of interspinous tissues in anesthetized rats. The virtue of this system is that the algesic used, capsaicin, causes a well-characterized response within a subset of polymodal nociceptors, so that mechanical stimulation is removed as a consideration. The stimulation is pure and relatively long-lasting pain, as might be encountered in clinical syndromes of spinal pain. Such stimulation has been shown to produce an immediate profound increase in systemic blood pressure and a matching increase in sciatic nerve blood flow. However, although blood pressure remained elevated for perhaps 20 minutes or more, sciatic nerve blood flow quickly dropped below pre-stimulus levels and remained there for approximately 20 minutes before normalizing. This suggests that noxious chemical stimulation of the interspinous tissues evokes two competing reflexes: (i) an increase in systemic blood pressure, which initially leads to a passive increase in sciatic nerve blood flow, and (ii) constriction of the sciatic vasa nervorum and so a decrease in sciatic nerve blood flow (Fig. 4). It would appear that with the long-lasting noxious spinal stimulation of capsaicin injection, the reflex constriction of the vasa nervorum becomes fully manifested and overpowers the effect of systemically increased blood pressure.

A related study has examined adrenal nerve activity and catecholamine secretion in response to capsaicin injection of thoracic and lumbar interspinous tissues. Both in CNS-intact and spinalized animals, noxious stimulation of the interspinous tissues normally leads to increases in adrenal sympathetic nerve activity and catecholamine secretion. It was possible to confirm both supraspinal and spinal reflex responses to stimulation of A and C fibers, and there was a relatively greater response to thoracic stimulation in the spinalized animal. In this regard, it should be noted that the bulk of preganglionic sympathetic neurons serving the adrenal gland in the rat are located between the T7 and T10 level of the cord.

Another recent study reported responses of bladder motility to noxious spinal stimulation. Previous studies had shown that the resting bladder could be stimulated to contract by noxious stimulation of the perineal skin. Noxious stimulation of other areas was ineffective. This suggests that the reflex depended upon stimulation within the territory of afferent fibers which enter the cord at the level of parasympathetic outflow to the bladder. However, the more recent study showed that stimulation at either the thoracic or lumbar level could produce a brisk response in bladder tone, this response was mediated at the supraspinal level, and the efferent limb of the reflex was within the pelvic nerves which provide parasympathetic innervation to the bladder. Here then we see that, in contrast to the adrenal studies, when the reflex is
mediated principally at the supraspinal level, there is not a clear segmental organization.

A study just completed has examined responses of gastric motility to capsaicin injection of thoracic and lumbar interspinous tissues. Noxious chemical stimulation of the interspinous tissues was associated with arrest of peristaltic movement and a sharp decline in gastric muscle tone. The decrease in gastric tone was significantly greater in response to thoracic as opposed to lumbar stimulation, was unaffected by bilateral vagotomy and was preserved in spinalized animals. This is the clearest demonstration to date of a segmentally organized, spinally mediated visceral response to noxious stimulation of spinal tissues.

To summarize the results of these animal studies, we may say that autonically mediated reflex responses to noxious stimulation of spinal tissues have been clearly demonstrated. Where parasympathetic influences dominate, a segmental organization has not been apparent. Where sympathetic mediation has been significant, it has been possible to demonstrate the existence of spinal reflex centres, and, to some degree at least, a measure of segmental organization.

Extrapolated to the patient, these results would predict that spinal pain syndromes might be accompanied by visceral symptomatology mediated by somatoautonomic reflexes, and that the nature of the visceral symptomatology might be predicted by the region of the spine experiencing pain. Are these predictions borne out by clinical observations?

**Observations from Clinical Studies**

Autonomic symptomatology may be an important sign of occult disease in spinal patients, patients with impaired nociception and patients who are unable to communicate pain. With the central nervous system intact, spinally-mediated reflexes tend to be dampened by supraspinal influences. However, when the influence of the brain is removed, as in patients with spinal cord injuries, the physiological importance of spinal reflex centers becomes obvious. This is apparent in the phenomenon of autonomic dysreflexia with high spinal cord injuries. Particularly with interruption of the spinal cord above the T6 level, any of a variety of stimuli to the gastrointestinal or urogenital tracts, or skin, muscle or bone below the level of injury, may result in severe paroxysmal hypertension, bradycardia and various other symptoms. The underlying pathophysiology of hypertension in autonomic dysreflexia would appear to be reflex vasoconstriction in the splanchnic bed, mediated by sympathetic preganglionic neurons in the lower thoracic cord. This is completely in
accord with animal studies cited earlier and demonstrates a segmentally organized, spinally-mediated reflex phenomenon.

The welfare of patients with spinal cord injuries is an important clinical priority. However, we also want to ask whether spinovisceral reflexes are of clinical importance in the larger population.

From observations of autonomic dysreflexia, and from animal studies, it appears that with the central nervous system intact spinally mediated reflexes are generally attenuated by descending supraspinal influences. Does this mean, therefore, that spinovisceral reflexes are of no importance in the population at large. Certainly not. Probably the classical example of the pathological spinovisceral reflex is paralytic ileus, the immobilization of the bowel which accompanies severe thoracolumbar pain, as for example in fracture of a vertebra. Mercifully, such injuries are uncommon. However, severe spinal pain is something which effects most individuals at some point in their lives. Most often it occurs in the absence of substantial tissue damage. Is it possible that such non-pathological pain can still produce clinically significant changes in visceral function, and if so, how widespread is this type of phenomenon?

The question which I have posed could be, and probably should be dealt with on purely scientific terms. However, it does have strong political overtones. Traditional western medicine, also called allopathic medicine, has tended to view the visceral and somatic systems in isolation. Indeed, we have taught the physiology and pathology of the autonomic and somatic nervous systems separately, as if the two systems did not communicate. Other health care systems, such as acupuncture, see no such separation, and frequently advocate stimulation of somatic structures to relieve visceral disease. Similarly, the western practice of chiropractic sees spinal pain as a potentially important factor in visceral disorders.

Observations from Physiological Studies in Humans

Physiological studies in humans support the observations made in animals. That is, somatic stimulation, including spinal stimulation, can significantly effect visceral function. For example, it has been shown that even innocuous mechanical stimulation of the cervical spine can induce statistically significant decreases in blood pressure\(^{14}\).

Observations from Surveys

In a recent study by the chiropractic profession in Sweden, a large number of patients were surveyed about relief of visceral symptoms after they had received treatment for spinal pain. This was an uncontrolled, retrospective study, which therefore provides only weak evidence. However, it was interesting that relief of visceral symptoms was not uncommon\(^{15}\).

A survey of clinical studies found reports of various visceral symptoms being relieved by treatment of the spine\(^{16}\). However, more than half of the reports cited relief of gynecological and visual complaints, asthma and enuresis. In other words, spinal pain seems to be associated with rather a specific set of visceral disorders.

Summary

In animal experiments, noxious stimulation of spinal tissues results in autonomically-mediated changes in visceral function. In humans, spinal pain may be accompanied by changes in visceral function, however, it is not clear how frequently this occurs. Nonetheless, clinicians should be alert to the possibility of secondary visceral dysfunction when treating patients with spinal disorders and, additionally, should be alert to the possibility that visceral symptoms may signal spinal disease.

References

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