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Review Paper

A critical review of the evidence for a pain-spasm-pain cycle in spinal disorders

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Summary

The existence of a pain-spasm-pain cycle in musculoskeletal disorders has been debated for over 40 years. This paper reviews critically the evidence for such a cycle in patients with back pain.

Clinical studies indicate that a substantial proportion of patients with back pain have muscle spasm. Patients with acute back pain have increased muscular activity on electromyography (EMG). EMG studies of patients with chronic back pain show, on the whole, an increase in activity in static postures, and a reduction of muscle activity during movement.

Experimental evidence shows that pain may cause muscle spasm and that muscular activity can be painful. Further evidence for the existence of a pain-spasm-pain cycle comes from studies which show that analgesics can reduce muscle spasm, and that a variety of muscle relaxant techniques can reduce pain.

There are substantial methodological problems in many of the studies cited, but the evidence reviewed provides general support for the existence of a pain-spasm-pain cycle. However further work is required to determine the nature of spasm and to evaluate methods for its detection, particularly in acute back pain syndromes. The value of therapeutic intervention may then be assessed.

Key words: Literature review, Pain, Spasm, Musculoskeletal disorders, Spine

Introduction

The first suggestion of a pain-spasm-pain cycle is generally credited to Janet Travell[1] who wrote in 1942:

> According to this view, limitation of motion is primarily a reaction to pain rather than the result of a structural lesion. If muscle spasm causes pain, and pain reflexly produces muscle spasm, a self-perpetuating cycle might be established...

There is continued argument about the existence of such a cycle, and some authorities doubt that muscle spasm is an important clinical entity at all. Despite this an array of treatments, e.g. heat, massage, mobilisation, drugs, injections, and biofeedback, have been developed and are used, at least in part, to reduce muscle spasm.

The purpose of this review is to examine the published evidence that patients with back pain have muscle spasm, and that their symptoms may be aggravated by a cycle of pain, spasm and further pain.

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Do back pain patients have muscle spasm?

Clinical studies

One of the notable features of the literature on back pain is the frequency with which authors refer to 'muscle spasm', and the rarity with which they define what they mean. The assumption that "muscle spasm may soon be detected with a little practice"[2], may well be incorrect. Failure to standardize the definition of this physical sign may account for apparent inconsistencies between the work of different authors. Furthermore, there is little information on minute-to minute variation in muscle spasm, or on between-observer variation in its detection. In one study[3], the measurement of spasm was abandoned because of poor repeatability, though a group of American osteopaths appeared to be able to detect spasm with greater reliability[4].

Only three clinical studies indicate the prevalence of muscle spasm in back pain patients. Barton[5] undertook a retrospective analysis of the records of 144 patients presenting to a Virginia family practice with low back pain. Muscle spasm had been recorded in 18% of charts (no definition of muscle spasm).

Magora[6] studied 429 Israelis selected from a popula-

tion survey on the basis that they reported having back pain. Thirteen per cent were judged to have 'tight or contracted' spinal muscles. Sixteen per cent had a lumbar scoliosis, the majority of which were judged to be due to asymmetrical muscle contraction.

Gunn and Milbrandt[2] reported the prevalence of muscle spasm in 60 back pain patients attending a Canadian Rehabilitation Centre. Muscle spasm or tightening was reported in 63% of patients who had tender muscle points, in 20% of patients without such points, and in none of a control group.

Although these three studies suffer severely from lack of detail, they give a general indication that muscle spasm is judged to be present in an appreciable proportion of patients with back pain.

Electromyographic studies

A direct measure of muscle activity would avoid the problem of the definition of spasm evident from clinical reports. Electromyography (EMG) measures that activity, and therefore provides direct evidence of whether or not a muscle is contracting. Initially, these signals could only be analysed qualitatively, but more recently, methods have been devised for quantification and statistical analysis of EMG signals.

The question which investigators have attempted to answer becomes: "Do patients with back pain show an increase in the electrical activity in muscle as demonstrated on EMG?" The results of 23 of these studies on patients with back pain are summarized in Table 1.

There are a number of problems in the interpretation of these studies. Most were carried out on patients with chronic back pain, but clinical details in the papers are often very sparse and it is therefore difficult to extrapolate the results to other groups of patients with back pain, and impossible to establish relationships between muscle spasm and particular back pain syndromes.

There are many technical difficulties involved in making EMG measurements. These are beyond the scope of this review. However, it should be noted that some studies contain very sparse reference to methodology—e.g. the position of the patient or what allowance was made for movement artefact. In some studies[25,26] a proportion of subjects were unable to adopt all the required postures. Exclusion of these subjects could have led to serious bias in the results. A further problem is that most studies used external electrodes placed on the back, and it was difficult to define precisely the muscles in which activity was being measured.

However, the following conclusions can be drawn from the studies summarized in Table 1:

1. The studies by Taverner[13] and Waylonis[18] suggest that patients with acute pain show a greater increase in muscle activity than those with long-standing pain.

2. Patients with palpable abnormalities in muscle showed increased muscle activity in those areas in studies by Denslow and Clough[7], Elliott[9], Arroyo[15], England and Deibert[19], and Fischer and Chang[29]. Increased muscle activity was found if the affected area was palpated in studies by Denslow and Hassett[8], Elliott[9], and Fricton et al.[28] No increase in activity was found by Kraft et al.[17].

3. Chronic back pain patients were found to show increased back muscle activity when doing a test with an unrelated muscle group by Holmes and Wolff[11] and Fowler and Kraft[20]. A psychologically stressful interview appeared to produce an increase in back muscle activity in the studies of Holmes and Wolff[11] and Flor et al.[27].

4. In no study were patients with chronic back pain found to have increased back muscle activity under all conditions. The results of studies in which back muscle EMG activity was related to posture and movement are summarized in Table 2. The apparently conflicting nature of these studies has caused considerable confusion in the literature. The caveats expressed above are particularly relevant to the interpretation of these studies.

5. Studies of EMG of chronic back pain patients in static postures on the whole demonstrate an increase in muscle activity compared to normal subjects. The main exception to this is the study by Collins et al.[25]. Her study is difficult to interpret, partly because two of her eleven patients were taking muscle relaxants, and partly because eight of the eleven patients were unable correctly to assume all the required postures. Other workers[30,31] have suggested that it is asymmetry of spinal muscle activity rather than the absolute level of activity which is important in the genesis of back pain. This is certainly consistent with the clinical observation that postural abnormalities appear associated with asymmetrical muscle spasm.

6. Studies of dynamic movements on the whole show reduced muscular activity in chronic back pain patients. This has variously been interpreted as indicating chronic muscle fatigue, or as apprehension by the patients when performing potentially painful movements.

The clinical evidence presented, though sparse and poorly described, suggests that muscle spasm is a relatively common clinical finding in patients with back pain. The two electromyographic studies of acute back pain suggest that such patients do have increased spinal muscle activity. Electromyographic evidence for spasm in patients with chronic back pain is conflicting. The evidence, on balance, is that chronic back pain patients have increased spinal muscular activity at rest or following exercise, but that they have reduced muscle activity during spinal movement.

Overall, the results indicate that back pain patients have disordered muscle activity, which at times includes muscle spasm. The following section reviews the evidence that this spasm may be directly caused by pain.

Does pain cause muscle spasm?

The evidence that pain may produce muscle spasm rests on observations that experimental production of pain may be associated with increased activity of muscles distant from the site of the painful stimulus.

Table 1. Summary of EMG studies of muscle activity in back pain patients

Reference	*No. of Patients*	*Type of patient*	*Control*	*Major relevant findings on electromyography*
Denslow & Clough (1941)[7]	16	Patients with osteopathic 'lesions'	Adjacent 'normal' areas	Increased activity in abnormally 'firm' muscle
Denslow & Hassett (1942)[8]	17	Patients with osteopathic 'lesions'	Adjacent 'normal' areas	Increased resting activity or increased activity on gentle local palpation of abnormally "firm" muscle
Elliott (1944)[9]	8	Sciatica+local muscle tenderness	Adjacent non-tender areas	Increased activity in tender muscles, especially on deep palpation
Price et al. (1948)[10]	?	'Acute and chronic back pain'	Normal subjects	Abnormal patterns of activity on movement—some muscles show increased activity, some reduced
Holmes & Wolff (1952)[11]	65	'Backache syndrome'	10 normal subjects	Increased activity of back muscles of B.P. patients on: a) voluntary contraction of distant muscles; b) during psychologically stressful interview
Golding (1952)[12]	120	Back pain	–	Failure to relax back muscles on full flexion in 34 patients
Taverner (1954)[13]	?	Various anecdotal reports	–	No increase in muscular activity in muscle pain, except in patients with acute lumbago attempting position change
Floyd & Silver (1955)[14]	105	Back pain	45 normal subjects	Failure to relax back muscles in full flexion in 34 patients
Arroyo (1966)[15]	?	Fibrositic nodules	Adjacent normal muscles	Continuous electrical activity
De Vries (1968)[16]	8	Chronic back pain	4 normal subjects	Three developed back pain on prolonged standing of whom 2 showed increase in spinal muscular activity
Kraft et al. (1968)[17]	16	'Fibrositis'	–	Areas of 'muscle spasm' found to show no muscular activity
Waylonis (1968)[18]	131	Cervical disc disease	–	Increased muscular activity in all 18 patients with 'acute disc', but only minor changes in those (113) with chronic disc disease
England & Deibert (1972)[19]	10	Chronic B.P. with osteopathic 'lesion'	Adjacent 'normal' areas	Increased muscular activity in 9 subjects

Pedersen et al.[32] found that mechanical stimulation of any structure in the spine of the anaesthetized cat was followed by electromyographically confirmed spasm in the dorsal and hamstring muscles.

In human subjects, both Cobb et al.[33] and Mooney and Robertson[34] found that experimental injection of pain producing substances (hyperosmolar sodium chloride) produced spasm of distant muscles. In Mooney and Robertson's experiment there was radiographic confirmation that the injection was precisely placed in one of the spinal facet joints, with reflex spasm being measured in the hamstring muscles of the leg.

These secondary increases in muscle activity are thought to involve spinal cord reflex mechanisms, and the anatomical pathways by which they are likely to occur have been described in some detail[35].

Although it is difficult to extrapolate these findings to the clinical situation, there is clear experimental evidence from these studies that pain may produce reflex spasm of distant muscles.

Does muscle spasm cause pain?

It is a commonplace observation that cramp is painful, and that overuse of a muscle, for example during athletic training, may lead to soreness of that muscle—either immediately or after a delay of a few hours.

The suggestion that muscle spasm is in itself painful dates back over 60 years[36]. The evidence that spasm actually results directly in pain rests on experimental observations in the laboratory that pain occurs predictably following repetitive muscle activity[37,38] and on demonstration of the physiological pathways which mediate this effect.

Table 1. *cont.*

Reference	*No. of Patients*	*Type of patient*	*Control*	*Major relevant findings on electromyography*
Fowler & Kraft (1974)[20]	10	Chronic neck and shoulder pain	35 normal subjects	Patients show increase in muscular activity while filling in a questionnaire
Jayasinghe et al. (1978)[21]	7	'Current' B.P.	4 normal subjects	Prolonged standing associated increase in muscular activity in patients, and decrease in muscular activity in controls
Wolf & Basmajian (1978)[22]	9	Chronic B.P. patients (6 post-surgery)	66 normal subjects	Reduction in spinal muscular activity, particularly when performing dynamic tasks
Kravitz et al. (1981)[23]	22	Back pain >6 months	17 normal subjects	Resting muscular activity same. Increase in spinal muscular activity in B.P. patients when contracting other muscle groups (minimal statistical significance)
Hoyt et al. (1981)[24]	40	Back pain >1 year	40 normal subjects	B.P. patients show increased spinal muscle activity standing, but not when sitting or lying
Collins et al. (1982)[25]	11	Back pain >6 months (2 on muscle relaxants)	11 normal subjects	Various tasks—control group show greater muscular activity
Soderberg & Barr (1983)[26]	25	Back pain >3 months	20 normal subjects	B.P. patients show failure to increase muscular activity for some tasks, and failure to relax back muscles in static positions following tasks
Flor et al. (1985)[27]	17	Back pain >6 months	17 patients with chronic pain (not back pain)+17 normal controls	Increased spinal muscle activity in B.P. patients at rest and during psychologically stressful interview
Fricton et al. (1985)[28]	16	Myofascial pain around shoulder (average duration 4–9 years	Opposite (normal) shoulder muscles	Increased activity in painful muscle following palpation of tender area
Fischer & Chang (1985)[29]	9	Back pain 1–25 years	12 controls Patients with no spasm and normal subjects	Increased activity over muscles in spasm during sleep

There have been a number of investigations of the physiological mechanisms underlying painful muscular contractions since the classical work of Lewis[39] in which he postulated the production of 'substance p' in muscle as a mediator of pain.

The hypothesis that pain producing substances accumulate in muscle during contraction has considerable support. It is known that the blood supply to muscle is decreased during voluntary contraction[40–42], that the pain following muscular exercise is very similar to pain induced by experimental reduction in the blood supply to muscle, and that experimental reduction of blood flow reduces the time that a subject can contract a muscle group before getting pain[37]. If pain producing substances are produced by muscle during exercise, then the relative reduction in blood supply will reduce the efficiency with which they can be cleared from the muscle, and therefore increase the chance that pain receptors will be activated.

There have been many candidates for the hypothetical 'pain producing substance'. One of the more likely candidates is potassium, which is known to be released from muscle cells during contraction[43]. Experimental injection of potassium into muscle causes pain[44] and the concentrations of potassium required to produce pain probably lie within the physiological range[45]. In experimental animals, infusion of potassium in physiological concentrations increases the output of sensory fibres leading from muscle to the spinal cord[46].

In addition to the chemical mediation of pain, it has been suggested that muscle spasm may cause pain by direct mechanical irritation of pain receptors within the walls of blood vessels supplying the muscle in spasm[35].

The mechanism by which muscle spasm causes pain remains to be proven. There do, however, appear to be plausible physiological mechanisms which would account for the phenomenon, and no reason to doubt that muscle spasm may, at least sometimes, be painful.

Table 2. Summary of studies on the effect of position on spinal EMG activity in chronic back pain patients

		Static postures										*Dynamic movements*				
References	No. of patients in this part of investigation	Unspecified resting	Sitting	Standing	Prolonged sitting	Prolonged standing	Prone/semi prone	Supine	Standing following lift	45° forward bend	Full forward flexion	Situps	Dynamic tasks (mixed)	Prone, shoulder raising	Prone, moving head	Lateral trunk flexion
Price et al. (1948)[10]	?	+												−/●	−/+	−/+
Holmes & Wolff (1952)[11]	1				+											
Golding (1952)[12]	120										●/+					
Taverner (1954)[13]	?						●									
Floyd & Silver (1955)[14]	105										●/+					
De Vries (1968)[16]	8					●/+										
Jayasinghe et al. (1978)[21]	11					+										
Wolf & Basmajian (1978)[22]	77		●/−										−			
Kravitz et al. (1981)[23]	22						●									
Hoyt et al. (1981)[24]	80		●	++			●									
Collins et al. (1982)[25]	22		−−					−−		−−						
Soderberg & Barr (1983)[26]	45		●	●					++		++	−−				
Flor et al. (1985)[27]	34		++													
Fischer & Chang (1985)[29]	9	++														

Where more than one symbol is given, these refer to individual subjects, where no grouped analysis is reported.
KEY: *No Statistical test performed:* Increased activity +; Normal activity●; Decreased activity −; *Statistically significant effect:* Increased activity ++; Normal activity●; Decreased activity −−.

Evidence for a pain-spasm-pain cycle

Some evidence has been presented to suggest that patients with spinal disorders have muscle spasm, that spasm may be a direct reflex response to pain, and that spasm may itself cause further pain. Further evidence for a pain-spasm-pain cycle comes from attempts to interrupt the cycle. If the cycle theory is correct, then direct reduction of pain ought to reduce muscle spasm, and direct reduction of muscle spasm ought to reduce pain.

Evidence that analgesics reduce spasm

Part of the evidence that led Travell[1] to propose the pain-spasm-pain cycle was her finding that injection of local anaesthetic agents into tender points in muscle resulted in an immediate improvement in the range of motion of the shoulder joint. Subsequently, it has been shown that the relief of pain associated with injection of local anaesthetic into myofascial trigger points is associated with immediate reduction in electromyographically recorded spasm[9,15].

More convincing evidence that analgesics can interrupt a pain-spasm-pain cycle comes from experiments where local injection of anaesthetic results in reduction of spasm in a distant muscle. For example, Mooney and Robertson[34] demonstrated that injection of local anaesthetic into symptomatic spinal facet joints produced reduction in electromyographically recorded spasm in the hamstring muscles of the leg. Increased stretching of hamstring muscles has also been demonstrated following surgical division of sensory nerves arising from the spinal facet joints[47].

Evidence that relaxation (biofeedback) reduces pain

Many investigators, assuming that back pain is caused by muscle spasm, have attempted to train patients to relax their spinal muscles using biofeedback techniques. It is known that patients are not able accurately to assess their own back muscle tension[20], and the use of visual or auditory feedback of electromyographically recorded muscle tension significantly increases the ability of patients to relax their back muscles[48].

There have been many reports of reduction of pain by EMG biofeedback (reviewed by Turk and Flor[49]). The technique has been subjected to randomised controlled trial in only two studies[50,51]. In the study by Flor et al.[50], the use of EMG biofeedback was associated with a reduction in spinal EMG levels while standing, and with reduction in pain. In the study by Nouwen[51], the technique produced reduction in spinal EMG levels while standing, but the reduction in pain in the biofeedback group was not statistically significant.

It is inappropriate here to describe the many problems in interpreting studies of EMG biofeedback. The technique is certainly widely used in managing patients with back pain. However, this can offer only general support to the pain-spasm-pain theory, as it is possible that the effect of biofeedback is mediated by some

mechanism independent of the pain-spasm-pain cycle—for example, non-specific relaxation and alteration of the subjects' response to painful stimuli.

Evidence that relaxation by physical therapy reduces pain

It is a common observation that physical stretching of a muscle may relieve the pain of a person with cramp. According to the pain-spasm-pain theory, if the spasm in a muscle can be overcome, then pain should be relieved.

This has been studied by Lewit and Simons[52] in an uncontrolled series of 244 patients with musculoskeletal pain apparently associated with muscle spasm. They described a technique used physically to stretch painful tight muscles in order to reduce muscle spasm. They reported immediate pain relief in 94% of patients.

Reduction in electromyographically recorded muscle activity by osteopathic manipulation[19] could also be related to physical stretching of tight muscle. It is equally possible, however, that reflex muscle spasm in this situation is reduced by the removal of incoming painful stimuli by the manipulation. Either explanation is compatible with the theory of a pain-spasm-pain cycle.

Evidence that muscle relaxant drugs reduce pain

Following reports of the successful relief of back pain with muscle relaxant drugs[53] over 20 years ago, a number of clinical trials have been conducted on the effect of muscle relaxant drugs on pain. In two double-blind controlled trials, muscle relaxant drugs were effective in reducing pain[54] or analgesic need[55]. In other controlled studies [56,57] the results have been equivocal. A number of studies of these drugs are inadequately controlled, use insensitive outcome measures, or have too small a sample of patients to be statistically useful.

The literature on the efficacy of muscle relaxant drugs has not been reviewed in detail because, although muscle relaxant drugs are widely used, their efficacy can only be adduced as evidence for a pain-spasm-pain cycle if it is possible to be certain that they have no other effect. This is not always the case: for example, myanesin[53] has a brief analgesic action, and diazepam[56] has a central psychotropic action. Caution therefore needs to be exercised in concluding that evidence of the efficacy of muscle relaxant drugs is evidence for the existence of a pain-spasm-pain cycle.

Discussion

The spine is an immensely complicated structure, whose mechanisms are still poorly understood. In the majority of patients with back pain—both acute and chronic—the physician is unable to make a precise pathological diagnosis. Any unselected group of patients will contain patients with many different diagnoses, some of whom will get better, and some of whom may not. Given such a heterogeneous group of patients, it would be quite remarkable if the presence of a pain-spasm-pain cycle or any other clinical phenomenon could be detected uniformly among the group.

Pain and spasm are common clinical features of patients with back problems, and it is known that they can arise independently under certain circumstances. For instance, there are some patients who have marked muscle spasm, but no pain (e.g. some patients with acute torticollis). Equally there are patients who have severe pain, but no spasm (e.g. 'hypermobility' syndrome). Between these extremes, there are patients who are found clinically, and can be shown in the laboratory, to have both pain and spasm.

None of the evidence cited provides proof of the existence of a pain-spasm-pain cycle. Indeed a number of individual studies are seriously flawed by methodological problems. However, the balance of evidence indicates that pain and spasm do not always arise independently in spinal disorders. There are established physiological mechanisms, albeit incompletely described, which indicate the pathways whereby pain causes muscle spasm, and muscle spasm causes pain. Further evidence for the pain-spasm-pain cycle comes from attempts to block the cycle: reduction in pain can lead to reduction in distant spasm, and reduction of spasm by three unrelated mechanisms can lead to reduction in pain. There is therefore a large body of evidence which is consistent with the existence of a pain-spasm-pain cycle.

It follows that there is a rational basis for treatments which are aimed at reducing muscle spasm, but published work does not indicate which groups of patients are most likely to respond to such measures. Indeed, one of the weaknesses of research done to date is that the great majority has been carried out on patients with long-standing backache, whereas spasm is perhaps a more frequent clinical finding in patients with acute back pain.

There is ample scope for future research in this field. Areas where research is needed include the definition of spasm and measurement of reliability of its detection, more work on the role of spasm in acute back pain, and more carefully designed studies to evaluate muscle relaxant techniques in chronic back pain.

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