NEUROPHYSIOLOGICAL APPROACHES TO CHRONIC PAIN FOLLOWING SPINAL CORD INJURY

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Abstract. Pain occurring in patients with spinal cord injury can be classified on clinical grounds into five types: peripheral, central, visceral, mechanical and psychic. An attempt has been made to correlate each type of pain with present neurophysiological knowledge. Mechanisms as to how unpleasant sensations reach the conscious level can be deduced when clinical and neurophysiological data are pooled. Eight case histories are presented which typify each class. The authors' evaluation and treatment offered is presented for each type.

Key words: Spinal cord injury; Pain; Neurophysiology; Treatment.

Introduction

THE existence of what can be broadly termed 'pain perception' by individuals with spinal cord damage has been described by Pollack (1951), Davis (1967), Sweet (1975) and Guttman (1976) among others. More recently, Burke and Woodward (1976) have proposed a classification of pain and phantom sensations following spinal paralysis which provides a useful clinical scheme for studying pain and altered sensations. Most authors have referred to at least five types of pain, namely: (1) radicular or 'end-zone'; (2) central or diffuse; (3) visceral; (4) phantom; and (5) cauda equina.

Pain, therefore, is a condition occurring in the spinal cord injured patient which has been well recognised and is a phenomenon which we must accept. However, since the perception of pain is a subjective and even an emotional experience which defies precise measurements, the fact remains that we still do not have an adequate way of gathering evidence that proves the patient's description of discomfort is due to factors that exist at or below the level of his lesion. We still lack the optimum tools to satisfactorily measure abnormalities of sensory conduction.

Clinical testing of sensation in spinal cord injured patients usually searches for the preservation of (1) vibration; (2) proprioception; (3) light touch; and (4) the discrimination of sharpness from dullness, and hot from cold. The diagnosis of completeness of the lesion depends upon the absence of all these modalities. It is recognised by experienced clinicians that these five modalities may have altered sensations, such as tingling or burning, superimposed upon a normal or diminished awareness of them during clinical examination. Such patients would still be diagnosed as incomplete. However, patients without these five modalities would all be labelled as having complete lesions. Some clinicians, however, have recognised that certain patients, when stimulated in ways other than by classical examination, are capable of perceiving some sensations from the periphery. For example, when some individuals are stimulated repetitively with a noxious or non-noxious input and/or when stimulated at several locations at the same time,

a kind of temporal and spatial summation seems to occur which results in an awareness on the part of the patient that something is happening. These sensations are not usually reported by the patient as normal, and may be unpleasant or painful. They may be poorly localised, manifest a latent period before the individual is aware of them, and may continue well after the source of stimulation is removed. In the past, such sensations have confused clinicians and have led them to question whether the patient's description of what he felt arose on the basis of suggestion by the physician, or constituted some form of illusion. These sensations, however, do conform to our current understanding of slow and fast systems of transmission within the peripheral and the central nervous system. Qualities of sensation tested in the classic clinical examination mostly fall within the fast or large fibre (A & B) system. The other sensations described above are requiring more intense and prolonged stimulation, and conform to what is known about the characteristics of transmission in the slow or small fibre (A delta, C) system.

Yet neurophysiological techniques remain inadequate to satisfactorily measure this slow fibre system in the clinical setting.

Our understanding, therefore, of what constitutes a complete lesion and what is perceived as pain in the spinal cord injured patient may be hampered by our lack of sophistication of our methods of evaluation and measurement. This paper attempts to bridge some of the current available neurophysiological knowledge with the clinical problems of pain in the spinal cord injured patient.

Materials and Methods

At The Institute of Rehabilitation and Research, within the Department of Neurophysiology and the Programme for Restorative Neurology, in 1980, 105 Spinal Cord Injured (SCI) patients have been referred for clinical and neurophysiological evaluation of abnormal sensation *e.g.*, dysaesthesias and intractable pain, and for abnormal movements, *e.g.*, spasticity, in the past 5 years. During this time, 49 patients have been referred for evaluation of abnormal sensation. We have developed a system of classification consisting of five major categories of abnormal sensation perceived by patients who have suffered a spinal cord injury. Clinical examples of each category follow:

1. Segmental Nerve (including Cauda Equina) Pain

(a) R. R.—This 22-year-old male was involved in a motor-cycle accident on 4 September 1980, causing a fracture dislocation of T12 on L1.

A motor level of T12 bilaterally and a sensory level of L3 on the left and L1 on the right were recorded. Deep tendon reflexes were absent below the level of the lesion. Bulbocavernosus reflex was weaker on the right than the left. He underwent a laminectomy at L1 the same night and a posterior Harrington instrumentation and fusion 6 weeks later. Acceptable but not anatomical reduction was obtained.

The patient complained of pain (which slowly increased in intensity over the next 3 months) in the anterior aspect of the right thigh almost since the time of the injury. It was episodic, lasting 2 to 3 seconds and stabbing in character. It was worse while he was inactive, particularly when he was trying to get to sleep, and was relieved by carbamazepine, amitriptyline or transcutaneous stimulation (TNS). Concomitant with this, he regained motor function on the left to the L3 myotome and on the right to the L1 myotome (with a trace of hip flexion and adduction on the right). He regained sensation on the left to the S2 dermatome for light touch, and to the L5 dermatome for pin prick. Sensation returned to the L1 dermatome on the right for both modalities. He, therefore,

did not perceive either of these sensations in the region where he perceived the pain. However, he did vaguely but consistently perceive deep sustained pressure when applied in this area over the anterior aspect of the thigh.

Electromyography was consistent with denervation in all lumbo-sacral myotomes but the tibial and peroneal nerves were electrically excitable, and voluntarily controlled motor units were found in the muscles seen to move clinically. Somatosensory evoked potentials (SEP) were obtained upon stimulation of both femoral nerves.

The patient underwent paravertebral and peripheral nerve blocks with local anaesthetic which were performed with SEP monitoring. When the evoked response was obliterated, the patient experienced about 50 per cent relief of pain. The same results were achieved when a 5 per cent phenol solution was used. However the beneficial effect lasted only several weeks.

Comment—Evidence of fast fibre conduction (by SEP) as well as slow fibre (by sustained pressure) was noted, despite the absence of all five classical clinical modalities in the anterior thigh area. The awareness of pain was influenced by removing sensory input from the affected area distal to the neural injury (nerve block) and by increasing input (general activity) from the areas remote from the injury.

(b) W. W.—This 31-year-old male was involved in a helicopter crash on 28 July 1978, causing a fracture dislocation of L1 on L2. A motor level at T12 with some trace of movement in the left leg muscles and a sensory level at L2 with bilateral sparing of light touch in all dermatomes below this was recorded. The saddle area was flaccid and pin prick was not appreciated. He underwent an anterolateral decompression of L2 and a posterior fusion with Harrington instrumentation.

He noted rapid improvement of neurological function over the next 6 weeks. Two and a half years after injury, he was first seen by the authors for pain in his left leg which had been present since his injury but was getting worse gradually.

Localised to the left leg below the knee, and involving the anal area, it came in short 3- to 5-second attacks and was usually dull but occasionally sharp and agonising. It was worse at night while he was trying to sleep, and was also aggravated when he perceived his rectum to be full. During defaecation, his pain was increased, but it diminished after his bowel movement was finished. The pain was unrelieved by analgesic or sedative-hypnotic medication. He had regained nearly normal strength and sensation in the right leg, but still had diminished strength and sensation below L3 on the left, where only sustained pressure and repetitive stroking were perceived as unpleasant feelings.

Somatosensory evoked potentials were obtained from both the right peroneal and tibial nerves, but from neither of these nerves on the left. The patient underwent a trial of carbamazepine but obtained no relief. Peripheral nerve blocks were then carried out, which relieved 100 per cent of the pain.

Comment—Only evidence of slow fibre conduction (by repetitive stroking) was noted. Awareness of pain was influenced unfavourably by increasing input below the area of injury (repetitive stroking, sustained pressure, and defaecation). It was favourably influenced by blocking input below the injury (tibial and common peroneal nerve blocks) and increasing input above the injury (general activity).

2. Spinal Cord Pain

(a) D. R.—This 52-year-old male was involved in an automobile accident on 3 May 1980. He struck his head against the windshield and noticed immediate paralysis of arms and legs. Initial examination revealed a sensory and motor level at C4 with sparing of only deep touch below this. Priapism was noted. X-rays showed a slight subluxation at C3/4 but no fractures. Myelograms showed no obstruction to the flow of dye and no cord enlargement. He was treated with skeletal traction for 3 weeks, then a cervical collar.

He noted slow but continuous neurological recovery from the first 48 hours. By 12 months post-injury, he could walk slowly but steadily using one cane, and was independent in all activities of daily living except those requiring finger dexterity. By

then, he still manifested a sensory level at C4, but below this, he could discriminate sharp from dull, recognise light touch, and had accurate position sense for all but the toes and distal finger joints.

About 10 weeks post-injury, the patient became aware of the sensation of tingling and numbness in his feet which gradually spread to his lower legs, hands, and forearms over the next few months. It became relatively constant and was decidedly worse with activity and when light repetitive stroking was applied to the involved areas. It subsided to a minimum when he was lying down, perfectly still. It affected his mood, causing depression and irritability. The pain was unrelieved by carbamazepine, diphenylhydantoin, phenothiazines or transcutaneous stimulation. Amitriptyline afforded partial (10 to 15 per cent) relief and caused some improvement of his insomnia. It was interesting that as the patient continued to regain neurological function, his tingling sensation worsened and he became impotent.

Somatosensory evoked responses were unobtainable upon stimulation of peripheral nerves in the upper and lower extremities. Electrospinogram revealed intact conduction throughout the CNS except through the cervical spine where temporal dispersion was marked. A blockade of afferent input via inflation of a sphygmomanometer held above systolic pressure on the left arm for 10 minutes (ischaemic test) completely abolished his pain in that limb. Sleep laboratory studies revealed an organic basis for his impotence. Epidural stimulation is planned if the abnormal sensations do not abate by 18 months.

Comment—Clinical evidence of fast fibre conduction to conscious awareness (posture-position sense, sharp-dull discrimination) was noted. However, temporal dispersion occurred (on electrospinogram) across the injury site. Pain was increased by peripheral input from everywhere below the lesion (general activity, light stroking—particularly of hands and feet) and decreased by blocking input (ischaemic tests, solitude).

(b) A. R.—This 25-year-old male sustained a fracture-dislocation of C4/5 when performing a somersault whilst practising gymnastics on 17 January 1974. He immediately lost the use of his legs and, after trying twice to sit, lost function in his arms as well. He underwent reduction and laminectomy at C4 and C5 the same day and was allowed to sit after 6 weeks treatment with skull tongs and traction.

Over the years his motor level has remained at C4 on the right and C5 on the left. His sensory level is at C5 bilaterally, but there are areas over both upper and lower extremities where sustained pressure over the skin and deep tissues produces an awareness which is delayed in onset and is slow to vanish; but which can, nonetheless, be localised to the approximate area being stimulated.

Several months after injury, he noted the onset of a tingling, burning pain in his feet, hands, rectum and penis, which gradually spread more proximal. It was constantly present except while sleeping and was augmented during activity. The penile pain was worse during voiding. The pain proved unresponsive to transcutaneous stimulation (in fact, this made it worse—but frequency settings are not known), carbamazepine, amitriptyline and various non-narcotic analgesics.

Somatosensory evoked potentials were unobtainable from stimulation of peripheral nerves in the upper and lower extremities but poly-EMG revealed the existence of long-loop reflexes and the influence of activity above the lesion upon spasticity below the lesion. The tonic vibratory reflexes were also present. Thus, neurophysiological supportive evidence of incompleteness was obtained, reinforcing the clinical impression.

The patient was also troubled by severe phasic and tonic hypertonicity. Because of his incompleteness, a trial of implantation of an epidural stimulator was undertaken. Electrodes were placed one segment apart at the inferior borders of C6 and C7. Stimulation at 33 hertz augmented the distal tingling and burning; however, when the frequency was increased to 195 hertz, this pain almost completely disappeared and remained absent as long as stimulation continued.

Comment—Clinical evidence of only slow fibre conduction to conscious awareness was noted on examination (sustained pressure). Pain was increased by peripheral input (activity) and decreased by diminishing input (sleep). Depolarisation of the dorsal

columns at low frequencies (under 50 hertz) augmented the pain, while depolarisation at higher frequencies (195 hertz) diminished it. Thus, the frequency of the input appears to have some bearing on the conscious perception of pathological pain sensation.

3. Visceral Pain

(a) J. W.—This 25-year old male was involved in an automobile accident on 14th December 1980, and sustained a compression fracture of C5. He underwent anterior fusion and was immobilised in a cervical collar for 8 weeks. He was initially noted to be complete at C4 level for motor and C5 for sensory testing.

Over the ensuing 3 months, the patient reported return of sensation. Sharp-dull discrimination remained at C5 while light touch extended to C8. All sensory modalities were absent below this as far as S1 on the right and L2 on the left, where sensation to deep sustained pressure returned. This return extended into the perianal region.

He underwent a sphincterotomy approximately 3 months post-injury. After this, he began to complain of burning abdominal pain throughout the entire abdomen, but more intense in the suprapubic area. It was unrelated to any specific event *e.g.* food intake, bowel programme, voiding or catheterisation. His appetite and bowel programme remained unaffected. The pain increased in intensity over the next 4 weeks. It was unrelieved by non-narcotic analgesics, tricyclic antidepressants, phenothiazines, or transcutaneous stimulation. Diazepam afforded dramatic relief, *i.e.* 80–90 per cent. The pain appeared to be worsened by his generalised flexor spasms but when baclofen was discontinued, his spasms intensified, but the pain was unchanged.

Extensive gastro-intestinal and genito-urinary evaluation did not reveal any intra-peritoneal or pelvic source for his pain. He did require a bladder neck resection because his hyper-reflexic detrusor converted to a hypo-reflexic one postoperatively (the first such conversion we have witnessed) yet his pain was unaltered by this procedure. SEP testing revealed no response to stimulation of peripheral nerves in the upper or lower extremities. He underwent polyelectromyographic evaluation, where spasms were elicited, and the extent of their spread was recorded. There was no direct correlation of spasm to pain demonstrated as a cause and effect relationship, although severe spasms tended to aggravate his pain. This case proved to be difficult to manage since numerous psychosocial problems were also present. Efforts centre on keeping his diazepam to the least effective dosage in order to maintain his pain at a level which he could accept. Continuous epidural and sympathetic and/or coeliac ganglion blocks are planned if the diazepam ultimately fails.

Comment—Only slow fibre conduction to conscious awareness was noted on clinical examination (sustained pressure). Pain appeared to be worse with increased input (spasms) and diminish with decreased input (diazepam), but no other strong correlations could be found.

(b) R. B.—This 37-year-old male fell from a rope and sustained a fracture of C7, in August of 1965. He underwent a laminectomy of C6/C7 that day. He has remained a C7 quadriplegic with motor sparing in C8 on the right, and sensory sparing to T2 bilaterally. He is clinically complete below T2.

There have been numerous urological complications. After an ileal diversion in 1973, he complained of left flank pain which radiated to the upper chest and back and into the groin. The pain has persisted despite the removal of a left staghorn calculus in 1976. It is constant, though worse when he is active, especially exercising. It has a dull, aching burning quality and is unrelated to eating, but is sometimes increased during his bowel programme. Only pentazocine and diazepam have provided partial relief.

SEP's were unobtainable from the lower extremities. Poly Electromyography revealed no radiation of motor activity below the lesion due to voluntary effort above. Tonic vibratory reflexes were absent. Thus, this patient's lesion was neurophysiologically as well as clinically complete.

Continuous epidural injection of saline caused no relief, but 3/4 per cent bupivacaine

abolished his pain when the level of anaesthesia encroached upon T1. Paravertebral blocks from T9 to T11 produced similar results and this area was then injected with 6 per cent phenol which abolished almost all of his pain.

Comment—Evidence of neither slow nor fast fibre conduction was detected in this patient. Yet, blocking input (epidural anaesthesia) to T1 abolished the pain possibly by blocking the sympathetics, while increasing input (general activity, exercise) made it worse.

4. Muscle Tension or Mechanical Pain

J. I.—This 40-year-old male fell from a platform on 4 June 1979, and sustained a fracture dislocation of C7 on T1. He underwent an anterior cervical fusion of C7 to T1. He was initially noted to have a C7 sensory and motor level with sensory sparing of light touch and deep touch to T1. Over the next 6 months this descended as far as T3, but otherwise, he remained complete below his level of injury.

About the time that he began to sit at a 90° angle, he complained of pain in his back in the T2 to T6 region, beginning in the midline and extending bilaterally into both shoulder blades. It was noticed to commence after sitting for 3 hours, was dull, aching, continuous, and aggravated by activity. It was relieved by reclining, by dextropropoxyphene and by transcutaneous stimulation (above his sensory level) but not by tricyclic antidepressants. Evaluation of his thoracic spine for vertebrogenic abnormalities was unrevealing. No other anti-inflammatory or non-narcotic analgesic medication altered his pain, however, he noted improvement after using transcutaneous stimulation, performing hyperextension exercises, and limiting his sitting to within his 3-hour limit.

Comment—Neither fast nor slow conduction across the lesion was detected on clinical examination. Diminishing input (rest, dextropropoxyphene) decreased the pain while increasing certain input (prolonged sitting, exercise) increased the pain; yet increasing other input (TNS) diminished the pain.

5. So Called Supratentorial or Psychogenic Pain

B. S.—This 30-year-old female noted the onset of pain throughout her body for 2 days followed by anaesthesia and paralysis below T4 in October 1974. Transverse myelitis was diagnosed from the clinical and investigative studies. She had no return of neurological function, and when reviewed 4 years after the onset, she remained a T4 complete paraplegic.

The pain ultimately settled over the entire abdominal area and around her back. This persisted continuously 24 hours a day, and was occasionally made worse by activity. It was unrelieved by numerous narcotic and non-narcotic analgesics, phenothiazines and tricyclic antidepressants and by TNS. Extensive investigations for gastro-intestinal and urological abnormalities were negative.

Testing for somatosensory evoked potentials showed no response upon stimulation of peripheral nerves in either lower extremity. Continuous epidural blocks with 0.75 per cent bupivacaine over a 3-day period failed to relieve the pain despite the fact that a T2 level of block was obtained.

As it was felt that she had significant psychosocial problems in addition to the discomfort she perceived, the patient agreed to participate in a pain programme employing psychotherapeutic counselling and behaviour modification. After 8 weeks, she reported mild decrease of pain intensity but a significantly improved plan of coping with the pain.

Comment—Clinical evidence of neither slow nor fast fibre conduction across the level of the lesion was noted. Pain perception proved to be more or less independent of input. Some increased input (activity) worsened the pain while other input (TNS) had no effect. Decreasing the input (medication, epidural anaesthesia), likewise, neither increased nor decreased the pain. Pain perception seemed to be mostly affected by her general pain rehabilitation programme. Such 'input' is a learning experience which helps her modify her responses to the environment at a conscious level.

Discussion

The preceding classification for pain following spinal cord injury has been modified from Schmidt (1978) who has provided a lucid discussion of pain on a neurophysiological basis. He states that pain can be divided into somatic and visceral types. Somatic pain can be further divided into superficial and deep. The superficial has two components, an initial sharp one and a delayed, dull one (Fig. 1). This delayed superficial pain, as well as the deep and visceral nociception, all arise from activation of free nerve endings and are transmitted via thinly myelinated (group A delta—conduction velocity of 10 metres per second, group C conducted velocity of 1 metre per second) nerve fibres. This compares with a velocity of 50 metres per second or more for the nerve fibres carrying the modalities of vibration or proprioception.

We believe that the segmental nerve pain, the visceral pain, and the muscle tension pain described in the case histories correlate respectively with the delayed superficial pain, the visceral pain and the deep pain described by Schmidt; and awareness of these pains results in residual transmission by small fibres both peripherally and centrally. The central pain, we feel, is caused by a disturbance in transmission within the spinal cord itself at the lesion, disturbing conduction of both large and small fibres; and finally, the 'psychic' pain reflects the patient's preoccupation with any number of factors disagreeable to him within the environment. The clinical characteristics of each type of pain are given in Table I.

1. Segmented nerve pain, whether arising from root or rootlet injury in the neck, thorax or cauda equina, in our experience is present almost from the day of injury unless, in the case of cauda equina injuries, simultaneous incomplete spinal cord insult delays its appearance due to spinal shock. It may worsen over time. Typically, it occurs or is augmented in 'waves' lasting several seconds and is stabbing in character but may have an undertone of burning, aching, or formication present for longer periods. It is most irritating during periods of inactivity, *e.g.* before sleep. It has a roughly segmental distribution and is perceived in areas where sensation is impaired or absent when the patient is tested by classical

FIG. 1

The five clinical types of pain are shown along with their postulated origins. Modified from Schmidt, R. F. *Fundamentals of Sensory Physiology*, Fig. 3-16, p. 112.

TABLE I
Parameters relating to the five types of pain listed on the left

Pain type	Time of onset post-injury	Character	Duration	Aggravating factors	Diminishing factors	Possible causative factors
Segmental nerve Cauda equina	Days to weeks	Burning Stabbing	Seconds	Rest	Activity	Slow fibre conduction from skin
Spinal cord	Weeks to months	Tingling Numbness	Constant	Activity	Rest	All fibre conduction within cord
Visceral	Weeks to months	Burning	Constant	Variable	Variable	Slow fibre conduction from viscera
Mechanical	Weeks to months	Dull Aching	Variable	Activity	Rest	Slow fibre conduction from muscle or ligaments
Psychic	Variable	Variable	Variable	Variable	Variable	Preoccupation with unpleasant environmental stimuli

clinical methods. Sensory testing consisting of repetitive touching, pressing or pricking with increasing intensity in simultaneous locations within the affected dermatomes, will usually produce an awareness that something is happening within that segment or at least that limb. This awareness may persist for several seconds after the stimulus is removed. If proper care is utilised to avoid the suggestion to the patient as to what the examiner is expecting, a patient who would otherwise be labelled as complete or having anaesthesia in the affected dermatome, can usually be found to have evidence of a partially viable peripheral nerve with transmission consistent with slow fibre conduction. This clinical information can help explain why nerve blocks, both proximal and distal to the lesion, may produce pain relief. Presumably, distal blockade is effective because it obliterates conduction in slow fibres activated by free nerve ending in the periphery. Sensory action potential within these fibres now no longer cross the injured area where they previously passed unaltered or after being distorted. Proximal blockade of course obliterates all sensory action potentials crossing the injured area including any which may arise due to direct irritation upon the nerve at the injury site. This latter type of pain impulse generation, Schmidt calls projected pain which compares with the experience in the non-spinal cord injured with pain arising from a herniated nucleus pulposus, or in the carpal tunnel syndrome.

Some patients have responded to carbamazepine and some to nerve blocks. We have found transcutaneous stimulation of limited usefulness on a long-term basis. Epidural stimulation has brought variable results depending upon the frequency selected. Higher frequency (*e.g.* 100 hertz) seems to be more effective.

2. Spinal cord pain, in our experience, is usually seen in patients with clinically identifiable incomplete syndromes, particularly the central cord syndrome. It takes several months to appear, is constantly present and has a tingling or numbing character. It is appreciated mainly in the most distal parts, *e.g.* feet and hands, and is aggravated by activity and is relieved by rest and solitude. We believe that this problem is an example of a disturbance of central processing within the spinal cord occurring as sensory action potentials become distorted as they cross the zone of injury. This may perhaps be similar to the perception of 'static' from a telephone receiver when a wire is partly damaged somewhere between the conversing parties. A carefully performed electrospinogram may demonstrate the distortion as temporal dispersion of the fast conducting fibres as shown in Case 2 (*a*). When all sensory action potentials from a particular peripheral area are blocked before they cross the zone of injury, the pain or tingling, in our experience, has consistently been observed to appear.

Treatment of this condition remains difficult. Pharmacotherapy is often ineffective, although we have seen some improvement of affect with tricyclic antidepressants. Permanent nerve blocks are contra-indicated when the patient's lesion is quite incomplete. Epidural stimulation at high frequencies, for example 100 hertz or more, has proved effective in some of our cases, *e.g.* Case 2 (*b*). This variation of effectiveness due to frequency may in part explain the generally poor results reported by Richardson (1980) where the frequency was not stated.

3. Visceral pain, in our experience, may occur at varying intervals from the date of injury but usually appears within a few weeks or months. It is usually constant, but worse at certain times and burning in character. It has an imprecise location in the abdominal or pelvic area. Fortunately, we have not frequently seen this problem. In our case, and those discussed by Burke and Woodward, there were no identifiable gastro-intestinal, genito-urinary or pelvic abnormalities which could account for the persistence of the symptoms, though both cases 3 (*a*)

FIG. 2

Electrosinogram: A. The left series was recorded from a normal individual while stimulating the left median nerve just above sensory threshold. Records are taken from bipolar electrodes located at: (a) supra-clavicular fossa (bottom or 4th graph); (b) across the C7 segment (3rd graph); (c) across the brain stem (2nd graph); and (d) across the contralateral hemisphere (top or 1st graph). Negative (upward) peaks are shown 10, 15 and 16 milliseconds (ms) on graphs 2, 3 and 4 for which the time base shown applies.

B. The right series was recorded from patient 2(a), while stimulating both median nerves just above sensory threshold. The (a) bottom two graphs (5 & 6) are recorded from the left and right supraclavicular fossae respectively; (b) the 4th graph across the C7 segment; (c) the 3rd across the brain stem; and (d) the top two graphs (1 & 2) across the left and right hemispheres respectively. The negative peak in graphs 5 & 6 is much reduced, and is not clearly discernible in 4. The peak in graph 3 is delayed and dispersed as well as in graphs 1 & 2.

and 3 (b) noted the onset of pain after a surgical procedure. Obviously, a pathological process in these areas must first be ruled out before we attribute the problem to neurogenic causes.

Nociceptors within the viscera are normally activated by distention, excessive contraction, ischaemia and by irritation by certain chemicals and toxins (Lance & McLeod, 1975). Afferent conduction is via the sympathetic nervous system from the thoracic and abdominal cavities or the sacral parasympathetic nerves from the pelvis. Participation of the vagus is doubted (Lance & McLeod, 1975) though it may play some role (Schmidt, 1978). It is not known at this point from what source neurogenic visceral pain arises in the spinal cord injured. Is it due to: (a) a continuous slow fibre discharge due to unrecognised alterations in visceral function; (b) a phenomenon occurring at the sympathetic chain ganglia; or (c) a distortion of the afferent impulses from the viscera crossing the zone of injury in the spinal cord? We hope to resolve this problem through the use of selected blocks *i.e.* epidural, sympathetic and coeliac ganglion. In Case 3 (a), diazepam was the only pharmacological agent which brought relief. Anticholinergic and antacids were ineffective. Whilst his spasms were benefited from the diazepam, they clearly did persist and worsened when his baclofen was removed. We concluded in this case that spasms might augment the pain but did not cause it.

4. Muscle tension or mechanical pain may also vary in its time of onset but usually coincides with the resumption of the activity which aggravates it. Demands upon muscles which remain functional after spinal cord injury increase significantly for all aspects of daily living, including the maintenance of balance. Further, in the spinal cord injured patient, muscles may be recruited to participate in activities for which they are unaccustomed. The trapezius and the latissimus dorsi muscles for example may participate in maintaining truncal balance since they may cross the bony and neurological lesion, nevertheless, they remain fully innervated in a high paraplegic or low tetraplegic, whilst the paraspinal musculature which normally controls this activity manifests diminished or absent strength and endurance below the lesion.

The pain is of a dull, aching character, and is aggravated by activity and relieved by rest. We believe that free nerve endings with slow fibres, known to be present in skeletal muscle, are activated when involved muscles reach a state of fatigue. However, it is not known what alterations take place within the muscle to activate this deep pain, *e.g.* ischaemia, abnormal contractions, or increased concentration of metabolic products.

Certainly, just as in visceral pain, pathological processes such as infection, instability and deformity, must be ruled out. After doing so, we have treated this problem with non-narcotic analgesics and physical measures (ice, heat, ultrasound, TNS, and modification of posture within the wheelchair). In successful cases, this may reduce pain perception by inhibiting slow fibre synapses within the substantia gelatinosa (Melzak). Mobilisation and strengthening exercises are also used to increase endurance and thereby delay the onset of fatigue. We have found carbamazepine and tricyclics to be ineffective, and do not recommend surgical procedures unless other pathological conditions exist.

5. So called supratentorial, or psychic pain, poses a common and difficult problem for all clinicians, no less for those caring for the spinal cord injured. We wish to state quite clearly that by proposing this classification we are not ignoring the fact that overlap does exist, particularly with this last category. Changes in affect in persons with chronic pain are the rule rather than the exception. Nonetheless, despite the fact that manifestations of pain or pain behaviours are a result of both the afferent volleys from the site(s) of tissue pathology reaching consciousness *and* the subject's preoccupation with them, there still remain those occasions when all that can be done to: (a) remove the tissue pathology; (b) modify the resulting afferent input to the CNS; or (c) modify the perception of the input at the cortical level has been done and the patient is still not satisfied. Until such time when we can better recognise and treat the effects of the tissue pathology, we are left with the strategy of assisting the patient to learn how to cope in a more effective way. This, in our experience, involves the utilisation of psychotherapeutic counselling and the structuring of a programme employing the principles of behavioural modification and the changing of elements in the environment in the functional, social, and vocational spheres. Success rates, even after all this, may remain quite modest.

SUMMARY

The authors have attempted to classify pain following spinal cord injury into clinically useful categories with neurophysiological correlates. In all categories, except perhaps the last, modification of sensory input had an effect upon the pain perception. The authors feel that pain in spinal cord injury should be carefully

evaluated clinically (and neurophysiologically when possible) and managed by modification of input along the lines reported above.

RÉSUMÉ

La douleur chez les patients souffrant de blessure à la moelle épinière peut se classer cliniquement selon cinq types différents: périphérique, centrale, viscérale, mécanique et psychique. On a essayé d'établir un lien entre chaque type de douleur et ce qu'on sait aujourd'hui au sujet de la neurophysiologie. C'est en rassemblant les données cliniques et neurophysiologiques qu'on peut déduire quels sont les mécanismes permettant aux sensations douloureuses d'atteindre le niveau conscient. On va présenter huit cas qui caractérisent chaque catégorie. L'évaluation de l'auteur et le traitement suggéré sont présentés pour chaque type.

ZUSAMMENFASSUNG

Schmerzen die in Patienten mit Rückenmarkverletzungen vorkommen, können in fünf Gruppen klassifiziert werden: periphere, zentrale, viscerale, mechanische und psychische Schmerzen. Jede Schmerzengruppe wurde mit dem vorhandenen neurophysiologischen Kenntniss korreliert. Die klinische und neurophysiologische Data können zum Verstehen der Mechanismen der Schmerzperzeption bei verschiedenen Patienten helfen. Acht typische Patienten von jeder Gruppe sind beschrieben mit den Resultaten von neurophysiologischen Testen und vorgeschlagenen Behandlungen.

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