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Diagnostic Spinal Anaesthesia in Chronic Spinal Cord Injury Pain

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Summary

In a double blind study, 21 patients with chronic spinal cord injury (SCI) pain underwent placement of a lumbar subarachnoid catheter and injection of placebo and lidocaine. The effects on pain intensity, distribution, altered sensations and sensory level of anaesthesia were monitored. Four patients responded briefly to placebo, while 13 demonstrated a mean reduction of pain intensity of 37·8 ± 37% for a mean duration of 123·1 ±95·3 minutes in response to lidocaine. The pain response to subarachnoid lidocaine differed significantly ($p < 0·01$) from placebo. Spinal anaesthesia was also associated with changes in pain distribution and altered sensation. A spinal anaesthetic-induced sensory level could not be achieved cephalad to the sensory level of neurological injury in 5 patients who presented with spinal canal obstruction. This study has demonstrated that response to diagnostic spinal anaesthesia in chronic SCI pain is complex, requiring individual interpretation in each patient and consideration of the following factors; symptomatology, etiology, pain perception, spinal canal anatomy, CSF chemistry and local anaesthetic pharmacology.

Key words: *Chronic pain; Intrathecal injections; Spinal anaesthesia; Spinal cord injury; Subarachnoid space.*

Despite improved medical care and better understanding of long-term disability and handicap in spinal cord injury (SCI) patients, effective treatment of chronic SCI pain is difficult. The incidence and symptomatology has been studied in depth (Botterell *et al.*, 1951; Davis 1975; Burke *et al.*, 1976; Waisbrod *et al.*, 1984;

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Donovan *et al.*, 1982; Davidoff *et al.*, 1987) however, such clinical studies lack clear definition of the various pain syndromes. Furthermore, little attention has focused on diagnostic regional anaesthetic interventions in order to obtain information about nociceptive mechanisms and pathways.

This study was designed to evaluate the effects of spinal, subarachnoid anaesthesia on chronic SCI pain. Differential spinal anaesthesia using a graduated technique (Winnie *et al.*, 1968) has been applied successfully to distinguish somatic and sympathetic mediation of nociception from placebo effect, the latter providing additional insight into psychogenic factors. Pollock (1951) reported that spinal anaesthesia effectively relieved SCI pain when administered above the spinal cord lesion. In the present study, an intermittent drug administration technique via an indwelling lumbar subarachnoid catheter was used to compare the effects of spinal anaesthesia and placebo.

Materials and methods

Twenty one patients (M = 17; F = 4, age: 18–58, mean = 42) with chronic pain (> 6 months duration) secondary to traumatic, non-progressive SCI (> 18 months post-injury, cervical = 5, thoracic = 14, lumbar = 2) were referred for diagnostic spinal anaesthesia (Table I). Pain was distributed over the thorax, lower abdomen, lumbar region, buttocks, lower extremities, rectum or coccyx and characterised as burning, aching, throbbing, sharp, pressure or dull. Notably, pain was regarded as severe, 'function-limiting' (Davidoff *et al.*, 1987) and refractory to traditional rehabilitative therapies including physical and occupational therapy, transcutaneous electrical stimulation and non-steroidal anti-inflammatory agents. Most patients described pain as constant with some minor fluctuation following activity or bedrest, although 4 patients (no. 5, 8, 15, 16) also presented with spontaneous intermittent paroxysms of sharp pain every 15 to 30 minutes. All analgesic medications were discontinued prior to the study.

Spinal anaesthesia was performed in the post-anaesthesia care unit where an intravenous line of 5% dextrose in lactated Ringer's solution was started. Patients were placed in the right lateral position and the area over the lumbar vertebral column cleaned with betadine for 15 minutes. Using strict asepsis, a 23 g polyethylene catheter was guided into the subarachnoid space via a 20 g Tuohy needle placed in the L3–4 or L4-5 vertebral interspace and taped in position. The patient was turned supine and catheter position confirmed by aspiration of clear cerebrospinal fluid (CSF) from the catheter hub.

A baseline assessment of pain was performed consisting of the patient's recording the intensity of pain on a 10 cm visual analog scale (VAS), evaluation of diagrammatic representation of pain distribution, quality of pain, presence of altered sensations and sensory level measurement (pin-prick or touch). Two separate injections of preservative-free saline (2 ml) (placebo) and 5% lidocaine in dextrose were administered via the subarachnoid catheter in each patient. In patients with thoracic and lumbar SCI, lidocaine was titrated every 5 minutes in 25 mg aliquots to a maximum dosage of 100 mg (2 ml) in an attempt to produce a sensory level of anaesthesia cephalad to the patient's SCI level, while in quadriplegic patients spinal anaesthesia was performed so as to produce a sensory level of T4 in 3 incomplete patients and an absence of reflex motor responses (lower

extremities) to noxious stimuli in 2 complete quadriplegics. The sequence of placebo and lidocaine injection was randomised and double-blinded and all pain assessments were performed by one individual. Following injection of local anaesthetic or placebo, pain assessments (VAS, pain distribution, frequency, altered sensations and sensory level) were repeated every 15 to 30 minutes to monitor the duration of any recorded changes. A decrease in pain was assessed as a positive response while a negative response was determined by the absence of any

Table I Description of patients

Pt	Age	Sex	Level of injury	Pain symptomatology
1	48	M	T8	Pressure pain—right thigh and hip joint (constant)
2	41	F	C7	Throbbing pain—buttocks and lower extremities (bilateral, diffuse, constant)
3	55	M	T8	Burning pain—buttocks, feet (bilateral), posterior aspect of left lower extremity from knee to ankle (constant). Constant dull rectal ache
4	48	M	L4	Burning pain—lower extremities (bilateral, diffuse, constant)
5	32	M	T12	Dull ache—thighs (bilateral, constant, L>R). Burning pain—feet (bilateral, constant). Intermittent sharp pain—left thigh region every 15–30 minutes
6	38	F	T12	Burning pain—thighs, feet (R>L, constant). Burning—midline abdominal region with cutaneous peri-umbilical hypersensitivity (constant)
7	51	F	T12	Burning pain—penis, hips, buttocks and lower extremities (bilateral, diffuse, constant)
8	44	M	T12	Burning pain—sacrum, coccyx and thighs (bilateral, constant). Intermittent sharp pain—right inguinal region every 15 minutes
9	41	M	T10	Burning pain—lumbar region (midline), buttocks, lower extremities (bilateral, R>L, constant)
10	48	M	T12	Stabbing pain—abdomen (left lower quadrant, fluctuating)
11	35	M	T10	Burning pain—sacrum, coccyx, thighs (bilateral, constant)
12	52	M	L2	Burning pain—lower extremities (bilateral, diffuse) and rectum (constant)
13	18	M	C6	Burning pain commencing at the level of SCI—upper extremities (bilateral, medial aspect from axilla to wrist, finger tips), upper thoracic region, lumbar region, buttocks and lower extremities (bilateral, diffuse, constant). Constant dull rectal ache
14	32	M	T10	Burning pain—lower abdomen (midline peri-umbilical, and lower quadrants), lower extremities (from knees to ankles and forefeet), (bilateral, constant)
15	43	M	T12	Burning pain—buttocks, lower extremities (from mid-thighs to toes), (bilateral, constant). Intermittent sharp pain—right inguinal region every 30 minutes
16	33	M	T8	Burning pain—abdomen (midline, peri-umbilical), lower extremities (bilateral, diffuse) and rectum. Intermittent sharp pain radiating down both lower extremities (posterior aspect) to feet every 15 minutes
17	53	F	T4	Ache—lumbar region (midline) radiating around flank to abdomen (bilateral, constant)
18	58	F	T6	Burning pain—buttocks, lower extremities (posterior aspect and heels), (bilateral, constant). Chest tightness in transitional zone (bilateral, fluctuating)
19	34	M	C6	Burning pain commencing at the level of injury—thorax, lumbar region, buttocks, lower extremities (bilateral, diffuse, constant)
20	36	M	C5	Burning pain—right hemi-abdomen (constant). Intermittent sharp pain in right upper quadrant every 30 minutes
21	39	F	C4	Burning pain—right buttock, right lower extremity, forefeet (bilateral, left upper extremity (from elbow to fingers) (constant)

change in pain status. For negative responses, 60 minutes elapsed before administering the next agent, while for positive responses, injection of the next agent was performed after pain symptomatology had returned to baseline (pre-injection) status for 60 minutes. The catheter was removed following injection of both agents. The Student's t-test and one-way analysis of variance were used for statistical analysis and comparison of the percentage change in VAS and duration for each agent.

Results

All 21 patients tolerated the injection of local anaesthetics and placebo without major side-effects. Mild hypotension associated with minor changes in heart rate were treated with intravenous crystalloid. In 2 patients (no. 5, 12), pain intensified briefly during recovery from lidocaine (rebound). Another patient (no. 7) developed fever after removal of the catheter. However, subsequent investigation revealed a urinary tract infection which was treated with antibiotics. None of the patients exhibited post-dural puncture headache after removal of the catheter or any infective complications such as meningitis.

The individual patient responses are shown in Tables II, and III. In 9 patients, injection of placebo preceded the administration of lidocaine, while in the

Table II Pain response to subarachnoid placebo and lidocaine (Decrease—percentage decrease in VAS; Duration—minutes; NC—No change)

	Placebo		Lidocaine	
Pt	Decrease	Duration	Decrease	Duration
1	NC		NC	
2	50	30	75	270
3	NC		NC	
4	NC		70	360
5	NC		80	45
6	50	30	25	120
7	NC		60	120
8	NC		NC	
9	NC		75	90
10	NC		100	180
11	100	60	60	60
12	NC		80	90
13	NC		NC	
14	NC		45	90
15	NC		NC	
16	NC		NC	
17	NC		20	25
18	NC		NC	
19	NC		15	90
20	25	30	NC	
21	NC		90	60
Mean	10·7	37·5	37·8	123·1
S.D.	25·7	15	37	95·3
S.E.M.	5·6	7·5	8·1	26·4
t	1·9	1·5	4·7	4·6
p	NS	$< 0·01$	$< 0·01$	$< 0·01$

remaining 11 patients, lidocaine was administered first. In response to placebo, 4 patients (no. 2, 6, 11 and 20) exhibited short-lived decreases in pain (10·7 ± 25·7%, p = NS) lasting less than 60 minutes (37·5 ± 15, $p < 0·01$). Seventeen patients demonstrated a negative placebo response. Following lidocaine, 13 patients demonstrated a mean reduction in pain intensity of 37·8 ± 37%, $p < 0·01$) for mean duration of 123·1 ± 95·3 minutes ($p < 0·01$), while 8 patients showed no change. The decrease in pain intensity following lidocaine differed significantly from placebo ($f = 7·6$, $p < 0·01$).

Following spinal anaesthesia pain was often described as being partially reduced and associated with changes in distribution (Tables II, III). For example, one patient (no. 14) reported that pain in the thighs and knees decreased considerably although burning pain in the left foot was unchanged, while another (no. 12) reported that rectal pain was unaffected despite reduction of pain in both lower extremities. In 1 patient (no. 10), disappearance of pain in the left lower quadrant was associated with temporary development of pain in an adjacent area on the left

Table III Changes in pain distribution and altered sensations following spinal anaesthesia

Patient no.	Description of response
1	Numbness and cold sensation in buttocks and lower extremities
2	Throbbing pain changed to burning pain in lower extremities. Tingling sensation in lower extremities during initial 30 minutes of spinal anaesthesia
3	Tingling sensation in left foot
4	Numbness in both lower extremities. Burning pain changed to stinging sensation in both lower extremities
5	No change in foot pain, sensation of warmth in thighs and feet. Cessation of intermittent thigh pain for 140 minutes
6	Numbness and vibration in lower extremities, pain in right thigh region unchanged. Peri-umbilical hypersensitivity and burning unchanged
7	Burning pain in calves and heels 'patchy'. Sensation of cold in both lower extremities
8	No change in altered sensations or intermittent pain
9	Decrease in burning pain in buttocks, and thighs. Right thigh region decreased more than left, no change in foot and calf pain
10	Numbness in thighs and calves. Paraesthesias in feet during initial 20 minutes of spinal anaesthesia. Developed dull ache in adjacent left thigh region for 60 minutes as pain in left lower quadrant decreased
11	Numbness in midline lumbar region and lower extremities, pain in sacral area unchanged. Decreased pain in sacral region and lower extremities following placebo
12	Warm sensation and vibration in buttocks and lower extremities. Burning pain in lower extremities decreased, rectal pain unchanged
13	No change in altered sensation
14	Numbness and warmth in abdomen and lower extremities. Pain in abdomen 'patchy.' Foot pain unchanged
15	Numbness and warm sensation in buttocks. Burning sensation in buttocks unchanged but decreased slightly in feet. Intermittent pain unchanged
16	No change in altered sensation
17	Aching pain in midline lumbar region slightly decreased. Pain in left lumbar paraspinal region and left lower quadrant unchanged
18	Numbness in the lower thoracic region posterolaterally. Transitional zone pain unchanged
19	Numbness and tingling in buttocks and lower extremities. Sensation of tightness in midline lumbar region
20	Slight decrease in abdominal pain following placebo
21	Burning pain in right buttock and lower extremity decreased. Forefoot pain and left upper extremity pain unchanged. Sensation of tightness in lower extremities

thigh. Several patients also described changes in altered sensations (temperature, tingling sensations, vibration, paraesthesias, numbness, sensitivity of skin to touch) in the lower extremities or buttocks following spinal anaesthesia (Table III). For example , 1 patient (no. 4) reported that burning lower extremity pain changed to a stinging sensation, while another patient (no. 2) reported that throbbing pain changed to burning pain following lidocaine administration instead of any subjective decrease in pain intensity.

Three patients (no. 6, 17, 20) experienced difficulty with perception and cognition of pain symptomatology following spinal anaesthesia. In particular, this included patients who demonstrated modest decreases in pain intensity of 15 to 30% following lidocaine (no. 6, 17) or placebo (no. 20). Two patients (no. 17, 20) expressed uncertainty as to whether any subjective change in pain status had occurred, even though evidence of pain reduction was demonstrated by the VAS, while 1 patient (no. 6) stated that a higher level of activity was needed (i.e. wheelchair mobility) in order to evaluate the effects of spinal anaesthesia on pain symptomatology.

The dosage of lidocaine (mean = 80 ± 20·8 mg) administered and sensory levels obtained are demonstrated in Table IV. A spinal anaesthetic induced sensory level cephalad to the patient's fixed SCI sensory level could not be achieved in 5 patients (no. 1, 8, 15, 17 and 18) with thoracic SCI (N = 14) despite maximal dosages of lidocaine. Indium cisternography, a procedure in

Table IV Lidocaine dosage (mg) and rostral sensory level of spinal anaesthesia (SA). (SCI—fixed sensory level due to neurologic injury)

Pt	SCI	SA	Dosage
1	T10	*	100
2	C7	T4	100
3	T8	T6	50
4	L4	T8	50
5	T12	T6	75
6	T12	T8	75
7	T12	T7	75
8	T12	*	100
9	T10	T8	75
10	T12	T10	50
11	T10	T6	75
12	L2	T6	50
13	C6	T4	100
14	T10	T6	75
15	T12	*	100
16	T8	T6	50
17	T4	*	100
18	T6	*	100
19	C6	T4	100
20	C5	^	100
21	C4	^	100
Mean			80
SD			20·8
t			17·2
p			< 0·01

*—Spinal canal obstruction.
^—Complete cervical SCI.

which 0·5 ml indium DPTI is injected intrathecally via the L4–5 interspace and is tracked as it spreads rostrally over the next 48 hours, was performed in 3 patients (no. 15, 17, 18), demonstrating complete spinal canal obstruction at the level of injury in all 3 (Fig. 1). In the remaining 2 patients (no. 1, 8), indirect evidence of

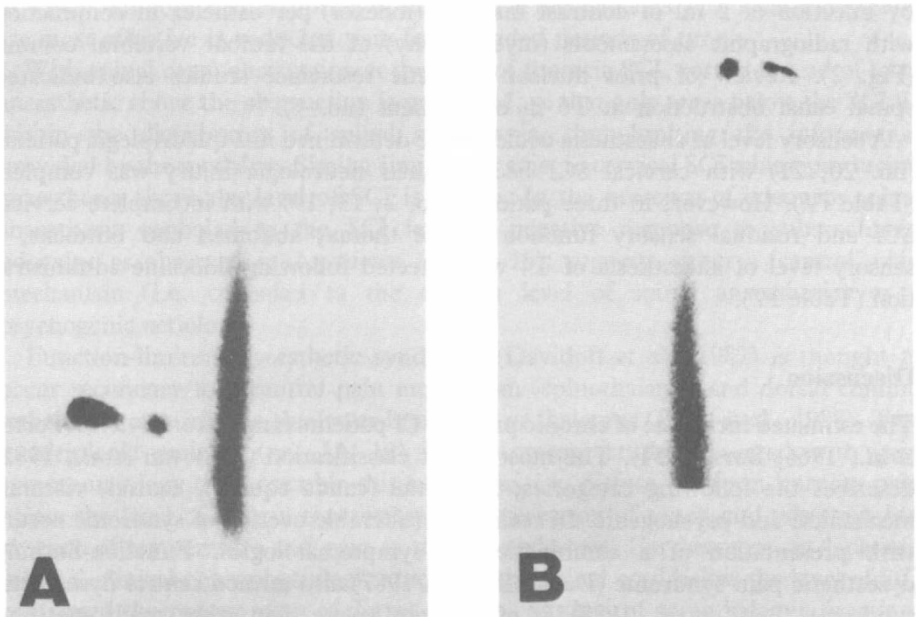

Figure 1 Indium cisternography (patient no. 17). **A**—2 hours after subarachnoid injection of indium, showing injection site and initial spread within CSF. **B**—24 hours later, spinal canal obstruction prevents rostral spread of indium toward base of skull.

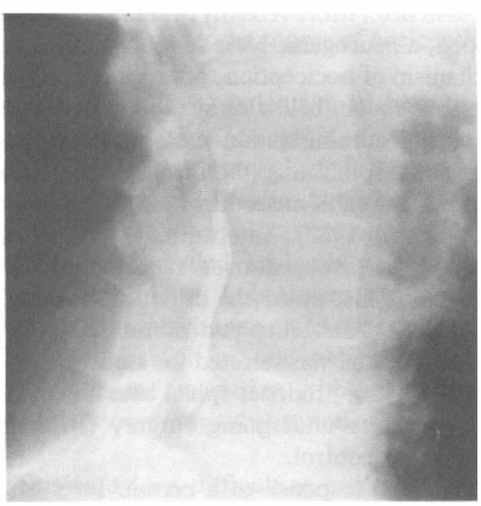

Figure 2 Myelography (patient no. 1). Injection of iohexol per catheter verifies correct placement within the lumbar subarachnoid space (despite inability to produce a sensory level cephalad to the neurologic SCI level).

spinal canal obstruction was present; xanthochromic CSF was obtained during performance of spinal anaesthesia and subsequent chemical analysis revealed markedly elevated protein levels viz. 431 mg% (no. 1) and 1427 mg% (no. 8). In addition, on completion of the spinal anaesthesia assessment, catheter position within the lumbar subarachnoid space was verified in both patients (no. 1, 8) by injection of 2 ml of contrast material (iohexol) per catheter in conjunction with radiographic assessments (myelography) of the lumbar vertebral column (Fig. 2). Review of prior nuclear magnetic resonance studies also indicated spinal canal obstruction at T6 in one patient (no. 1).

A sensory level of anaesthesia could not be determined in 2 quadriplegic patients (no. 20, 21) with cervical SCI because their neurologic injury was complete (Table IV). However, in three patients (no. 2, 13, 19) with incomplete cervical SCI and residual sensory function in the thorax, abdomen and buttocks, a sensory level of anaesthesia of T4 was detected following lidocaine administration (Table IV).

Discussion

The estimated incidence of chronic pain in SCI patients ranges from 3–94% (Porter *et al.*, 1966; Bors, 1951). The most recent classification (Donovan *et al.*, 1982) describes the following categories; segmental (cauda equina), central, visceral, mechanical and psychogenic. In reality, considerable overlap of syndrome occurs with presentation of a combination of symptomatologies. Function-limiting dysesthetic pain syndrome (Davidoff *et al.*, 1987) also termed central dysesthetic syndrome (Beric *et al.*, 1988), is often more severe than other syndromes and associated with incomplete SCI (Davidoff *et al.*, 1987), while the exact incidence and severity of psychogenic pain is unknown. In the past, chronic SCI pain was viewed as a non-organic psychodynamic manifestation of central nervous system injury hence the term 'phantom' defined as 'something apparent to the sense but with no substantial existence.' More recently, as radiographic and neurophysiologic technologies developed, a neurogenic basis for chronic SCI pain became accepted. The origin and mechanism of nociception, often called the 'neural pain generator' is thought to exist either above, below or at the actual site of SCI.

The technique of drug administration used in this study is an extension of Winnie's (1968) differential spinal anaesthesia, although utilisation of subarachnoid catheters for the administration of anaesthesia has not gained popularity (Peterson *et al.*, 1983; Denny *et al.*, 1987). Intermittent administration of agent via a subarachnoid catheter was favoured over repeated percutaneous injection to decrease the risk of meningitis, avoid the difficulties associated with positioning patients with major physical disability and minimise interference with patients' perceptual processes. Lidocaine was selected for study since a concentration of 5% and maximal dosage of 100 mg produces spinal anaesthesia to a segmental level of approximately T4 in patients undergoing surgery (Bridenbaugh *et al.*, 1988). Placebo served as the study control.

In chronic SCI pain, the response to a certain level of spinal anaesthesia is influenced by the relationship of the 'neural pain generator' to the actual SCI. The level of spinal anaesthesia may be manipulated to involve areas that are producing pain either below, at or above the SCI in turn elucidating the exact site of pain

production. If the source of nociception exists within the vertebral column, spinal anaesthesia with lidocaine should produce analgesia for approximately 60 minutes (Bridenbaugh *et al.*, 1988). However, in the present study, analgesia produced by lidocaine (123 minutes) exceeded the expected duration, which may have represented a 'carry-over' effect as observed following stellate ganglion blockade, in which temporary interruption of nociceptive mechanisms with local anaesthetic are most effective in reducing pain for extended periods of time.

With spinal canal obstruction at the level of thoracic SCI, rostral spread of local anaesthetic above the obstruction is prevented, so that only areas below the SCI lie within the distribution of spinal anaesthesia, thus limiting the information provided by the modality. Similar limitations exist in cervical SCI where producing anaesthesia above the level of SCI is unsafe. In the presence of adequate spinal anaesthesia cephalad to the SCI level, a negative response to subarachnoid lidocaine as observed in 2 patients (no. 3, 16), suggests either a 'central' pain mechanism (i.e. cephalad to the sensory level of spinal anaesthesia) or a psychogenic aetiology.

Function-limiting dysesthetic syndrome (Davidoff *et al.*, 1987) is thought to occur secondary to a central pain mechanism (spinothalamic and dorsal column imbalance) occurring in the lower brainstem or thalamus (Beric *et al.*, 1988). Two quadriplegic patients (no. 13, 19) in the present study presented with pain symptomatology that resemble this syndrome, i.e. diffuse, uniform burning pain below the level of injury, together with preservation of touch and vibration but absence of temperature and pain in the affected areas. Furthermore, in 1 patient (no. 13) clinical neurophysiologic studies of the spinal cord below the level of SCI confirmed the preservation of dorsal column, but loss of spinothalamic function. Following spinal anaesthesia to a sensory level of T4, one patient (no. 13) reported no change in pain, while the other (no. 19) demonstrated a 15% reduction of pain. These findings support the hypothesis that function-limiting dysesthetic pain syndrome has a predominant 'central' aetiology. For the remaining 3 quadriplegic patients in the present study, a positive response to spinal anaesthesia was observed in 2 patients (no. 2, 21) suggesting that the source of nociception was caudad to the SCI.

The observed placebo effect in 4 patients may have represented a normal response, secondary to anticipation of analgesia. However, 17 patients did not exhibit placebo effects indicating that psychologic factors may not be as significant as previously thought. Sequence of placebo administration is also important if patients experience clear effects following local anaesthetic. In 1 patient (no. 5), the response to spinal lidocaine included analgesia and a new sensory level of anaesthesia cephalad to the SCI level. Subsequent administration of placebo was not associated with these effects which then clued the patient that placebo had been administered. As described earlier in one patient (no. 9), development of discomfort in the left thigh in association with relief of pain in an adjacent abdominal area, termed 'symptom substitution' suggests strong psychologic overlay.

Certain results in this study are difficult to explain using a traditional spinal anaesthesia model, particularly the observations of partial analgesia seen in most patients. In chronic SCI pain, several different aetiologies may each contribute to the pain syndrome. Spinal anaesthesia may 'unmask' pain symptomatology by

selectively interrupting some nociceptive mechanisms but not others, in turn producing partial analgesia or changes in pain distribution. The observations of persistent rectal and foot pain in 2 patients in the present study (no. 12, 14), despite significant reductions of pain in other affected areas supports this hypothesis, suggesting that pain included a neurogenic component from the vertebral column (positive response) and a 'central' component (negative response) rostral to the spinal anaesthetic. Other mechanisms that may also be involved include opioid and noradrenergic receptors or agonists, since epidural administration of clonidine, morphine and buprenorphine have been demonstrated to be effective in reducing deafferentation pain secondary to SCI (Glynn *et al.*, 1986).

SCI patients may experience difficulty or uncertainty in reporting subjective changes to their pain and altered sensations as observed in 3 patients (no. 6, 17, 20) in the present study. Environmental noise and mental tasks may serve as distractions while emotional affect, position in bed, levels of activity, current non-analgesic medications and diurnal factors also influence pain symptomatology. Therefore, a 15 to 30% reduction in pain intensity following spinal anaesthesia is less useful in providing information about the source of nociception, since it may represent placebo effect or problems related to patient's perception of pain (false-positive response). In contrast, a 75 to 100% decrease in pain following spinal anaesthesia elucidates the origin of nociception and locus of the neural pain generator, while excluding psychogenic and central pain aetiologies.

The exact site of action of spinal local anaesthetics is controversial. It is currently held that local anaesthetic accumulates along the posterior and lateral aspects of the spinal cord and in the spinal nerve roots, areas which are heavily myelinated (Greene, 1981). Injury to the spinal cord is associated with marked chronic pathologic change, including collagenous scarring in the damaged area of the spinal cord and fibrous gliosis above and below the margins of injury (Hughes, 1978). Anatomic planes become indistinct as meninges fuse with the spinal cord and canal in turn producing spinal canal obstruction, as observed in 5 patients in the present study. A traumatic syrinx may also develop with upward or downward cavitation. In cauda equina injuries, a 'scarred-stump syndrome' has been described consisting of microstructural abnormalities, chemical changes, synaptic sprouting and arachnoiditis (Burke, *et al.*, 1981). These anatomic changes may considerably affect CSF and spinal cord blood flow limiting spread of local anaesthetic to the 'neural pain generator' sites and retarding subsequent disposition. Disposition of drug may also be affected by the action of local anaesthetic on spinal cord blood flow. Subarachnoid bupivicaine, an amide local anaesthetic related to lidocaine decreases canine spinal cord blood flow 29% (Kozody, *et al.*, 1985). The effects of neuronal demyelination and remyelination after compressive SCI (Helgason *et al.*, 1987) on local anaesthetic action are unknown, although nonmyelinated C-fibers are resistant to local anaesthetic blockade (Scurlock *et al.*, 1975). It is possible that structural changes to the substance of the spinal cord affect or even obliterate the spaces of Virchow-Robin, which penetrate the spinal cord and provide direct access for local anaesthetics to deeper areas (Greene, 1981).

The effects of increased CSF protein on local anaesthetic pharmacology are unknown although lidocaine is 64·3% protein bound in plasma (Tucker *et al.*, 1988). An increase in the concentration of CSF protein secondary to spinal canal obstruction may be associated with greater binding of local anaesthetics and

decrease the unbound diffusable fraction. This may account for the negative response to subarachnoid lidocaine in 2 patients (no. 1, 8) with elevated CSF protein. Unfortunately, CSF protein was measured in only 2 patients in the present study.

Clinical neurophysiologic monitoring of duration of reflex inhibition following spinal anaesthesia may provide additional information about local anaesthetic action and disposition. This may be particularly useful in the cervical SCI patient with minimal sensation below the level of injury. Here, in the absence of sensory cues, monitoring of spinal reflexes can provide a measure of the extent of spinal anaesthesia below the SCI.

In conclusion, this study has demonstrated that in chronic SCI pain, response to spinal anaesthesia is complex requiring individual interpretation in each patient. Several unique factors may influence this response including the character of pain symptomatology, the aetiology, the locus of the pain generator, perception of pain, the spinal canal anatomy, the CSF chemistry and local anaesthetic pharmacology. In certain instances, these factors may confound assessment of clinical responses limiting the usefulness of the modality or even leading to erroneous assumptions about pain mechanisms. In addition, the findings of this study advocate the measurement of CSF protein and assessment of spinal canal anatomy prior to performing diagnostic spinal anaesthesia. Extension of the present study to a large cohort of SCI patients is indicated to address refinement of the methodology of diagnostic spinal anaesthesia, assess the effects of confounding variables and study the role of other possible mediators of nociception including opioid, GABAergic and noradrenergic mechanisms. Long term follow-up is also needed to evaluate whether the information derived from diagnostic spinal anaesthesia impacts subsequent medical or surgical pain management.

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