Early and Subtle Signs in Low-Back Sprain

C. CHAN GUNN, MA, MB, B CHIR
and WILLIAM E. MILBRANDT, MD

The authors have previously reported myalgic hyperalgesia as a useful localizing sign in "low-back sprain" patients with no physical findings. This paper describes some other subtle signs related to the phenomenon of denervation supersensitivity which is well known to physiologists and clinicians involved in peripheral nerve disease, yet its related signs have not been applied to low-back pain. Following denervation of some neurons, muscle and peripheral receptors become supersensitive to transmitter substances and to different forms of stimuli. Since the peripheral nerve is a mixed nerve, findings are multiphasic and include autonomic dysfunction, trophic changes, cutaneous and myalgic hyperalgesia, and increased muscle tone. One or more of these signs occurred in 30 patients with secondary low-back pain but less often in 30 patients with primary or mechanical low-back pain; their presence, though slight, in asymptomatic controls may identify those individuals with a vulnerable back. [Key words: Low-back pain, denervation supersensitivity, autonomic dysfunction, trophic changes, hyperalgesia, increased muscle tone]

Is THE PRESENT state of the art of low-back examination adequate? As often as not, following a careful examination, no localizing physical findings are found and therefore no conclusion as to diagnosis or prognosis is possible. Treatment in these "low-back sprain" patients then is at best empirical, pending either resolution of symptoms or further deterioration, until the classic signs (such as limitation of straight leg-raising, areflexia, or muscle atrophy) develop, when neuropathy may be so advanced that its sequelae are frequently irreversible and total functional recovery prejudiced.

In a recent paper,[11] the authors introduced a method of examination for myalgic hyperalgesia or muscle tenderness at motor points as an early manifestation of radiculopathy. This paper presents other subtle signs of peripheral neuropathy based on the application of recent physiologic concepts. A clinical study evaluating the usefulness of these physical signs in low-back sprain patients is included.

NEUROPHYSIOLOGIC CONSIDERATIONS

It is generally agreed that spondylotic symptoms may occur following the structural disintegration and morphologic alterations in the intervertebral disc which lead to pathoanatomic changes in the surrounding structures, especially the bones and meninges; but spondylosis, per se, as part of the aging process produces no symptoms as it is with us all and, by the eighth decade, almost universal.[3] Symptoms only occur when and if the degenerative changes in the intervertebral discs or secondary changes in the adjacent vertebrae impinge on pain-sensitive structures and nerve root(s). Whatever the actual pathogenesis may be, (and many have been proposed: compression by degenerative intervertebral disc material, interference with the blood supply, hypertrophy of the ligamentum flavum, excessive movement between adjacent vertebrae, stress on the posterior joints or constitutional stenosis of the spinal canal) and

From the Clinical Research Unit at the Rehabilitation Clinic, Workers' Compensation Board of British Columbia, Vancouver, British Columbia, Canada.
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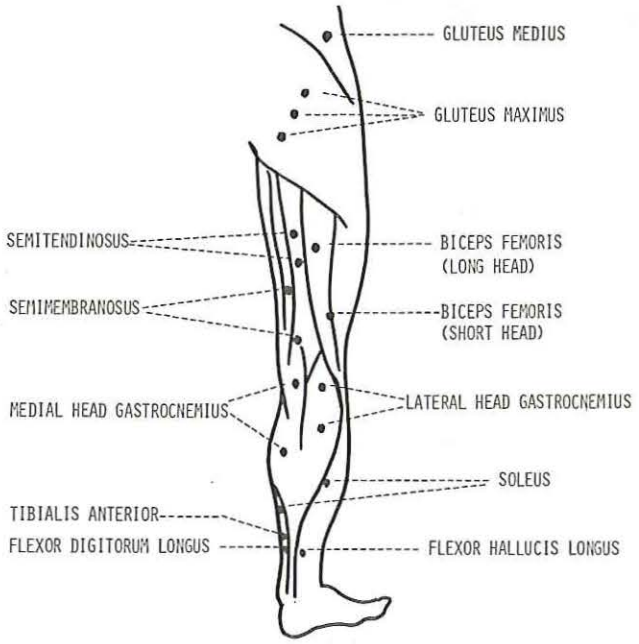

Fig 1. Some tender motor points of the lower extremity (posterior aspect).

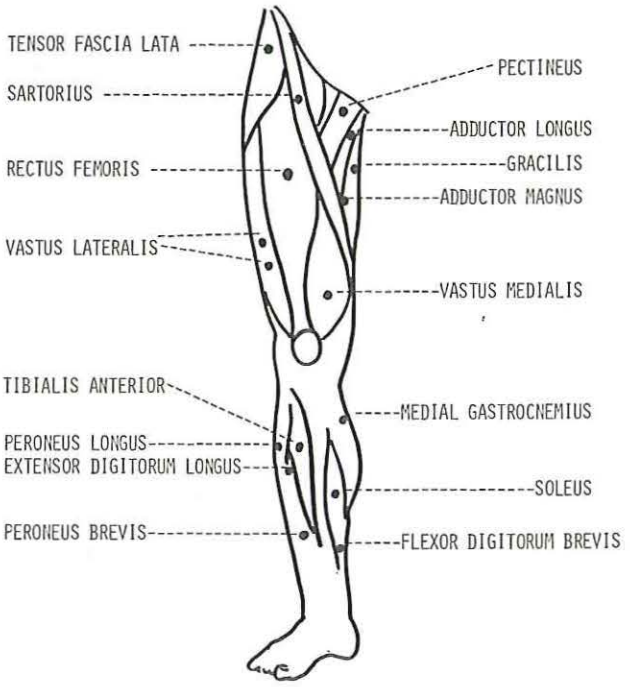

Fig 2. Some tender motor points of the lower extremity (anterior aspect).

whether precipitated by one traumatic episode or by minor accumulative incidents, the end result is pain which may be felt locally and/or transmitted intrasegmentally by the spinal nerve.

Local pain or primary backache[22] occurs at pain-sensitive tissue sites and structures as a result of irritation, injury, or inflammation. The nerve root in its course through the intervertebral foramen may be locally painful due to a) sensitive nerve fibers of the nerve

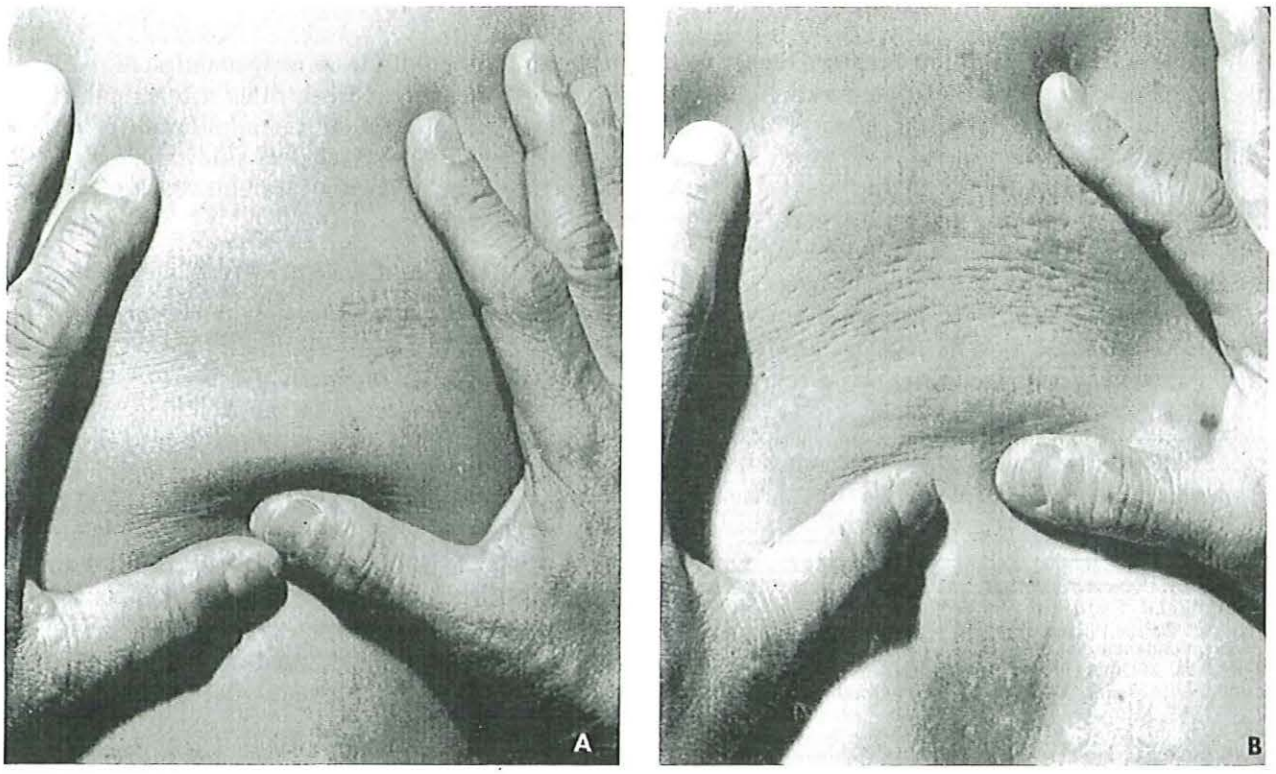

Fig 3A. Wrinkling of normal skin when gently squeezed together. **B.** Trophedematous skin when gently squeezed together—the "peau d'orange" effect.

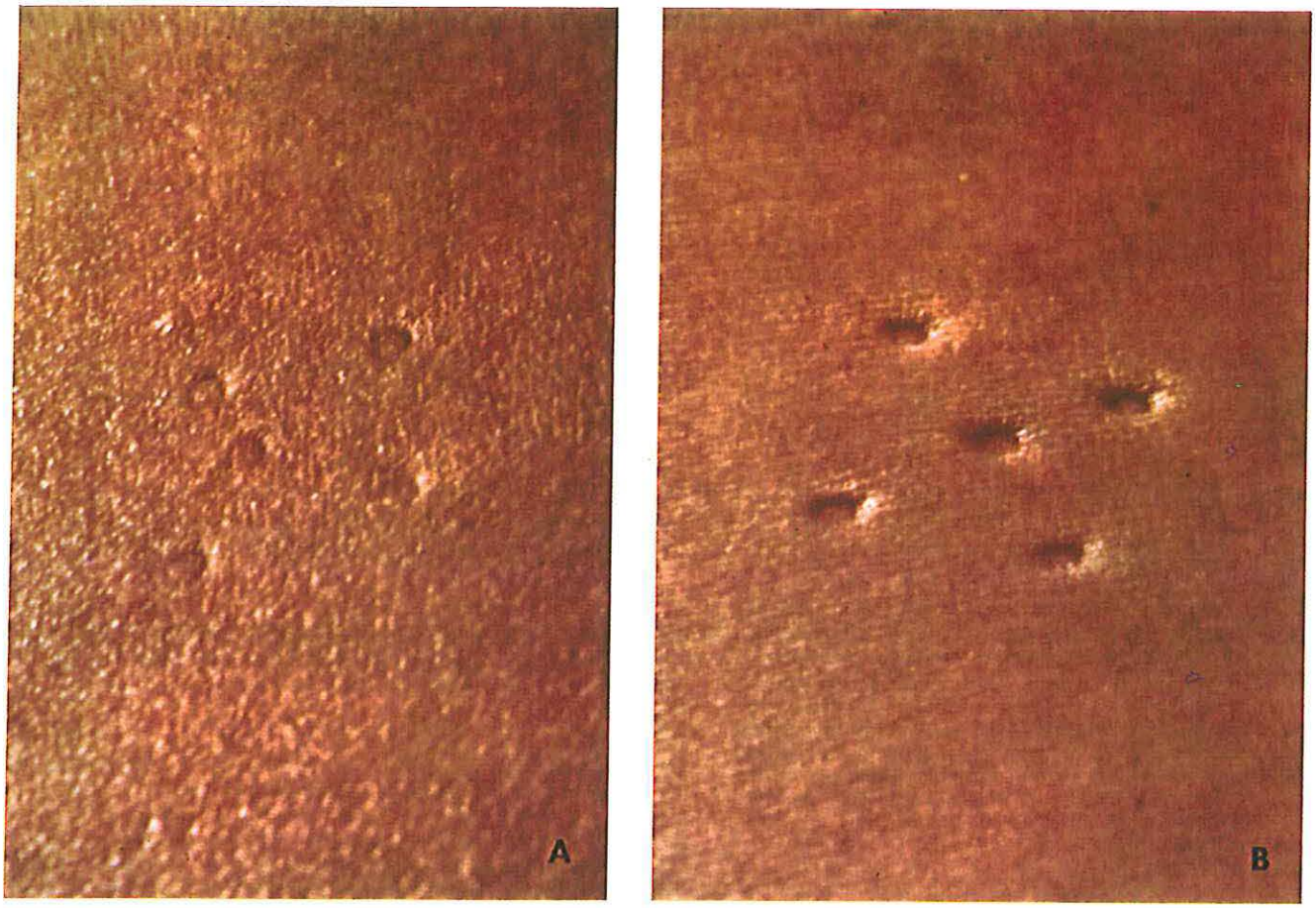

Fig 4A. Results of the "matchstick test" on normal skin—negative. **B.** Positive results of the matchstick test on trophedematous skin.

root dural sheath, b) involvement of the dorsal sensory root and/or c) the sensory fibers of the motor root.[3] Local pain and pathology may commonly be limited only to the back without intrasegmental referral and without neurologic deficit or referred pain, but it is less usual for referred symptoms to occur without some concomitant local back pain.

In transmitted pain or secondary backache,[22] when the peripheral nerve or the dorsal root ganglion[14] is involved, two main types of nerve fiber degeneration are recognized—axonal degeneration and segmental demyelination. In axonal degeneration, axon death leads to secondary breakdown of the myelin sheath. (Where the cause is focal trauma, the term "wallerian degeneration" is used, and where axonal degeneration occurs due to diseases other than trauma, the term "axonal neuropathy" is usually applied.[2]) In segmental demyelination, the Schwann cell and myelin sheath are damaged, but the axon basically remains intact, although conduction of the nerve action potential may be blocked. In most injuries both axonal degeneration and segmental demyelination occur together to a varying degree. Variable degrees of damage with variable degrees of reversibility may occur, ranging from transient neurapraxia to axonal stenosis and axonotmesis. Proximal to the injury, a number of associated changes also occur, including retrograde degeneration and central chromatolysis in the cyton (cell body). Sunderland defined five degrees of injury.[18] In the first degree there is loss of conductivity of axis cylinders at the site of injury without any grossly apparent break in continuity of the structures comprising the nerve trunk, and regeneration is relatively effective. Even when injury reaches Grade IV, when there is damage to the axon, connective tissue, and perineurium, the nerve remains macroscopically intact but regeneration is poorly orientated and less effective. Early recognition of a nerve subjected to spatial compromise and its differentiation from a simple mechanical backache is therefore important to prevent irreversible damage and to initiate meaningful treatment.

In neuropathy and following denervation there occur a number of characteristic changes in the functional properties of muscle and peripheral receptors. These become supersensitive to transmitter substances and to different forms of stimuli.[8,21] In normal muscle, the muscle fiber membrane zone that contains receptor sites activated by acetycholine is confined to that area immediately adjacent to the end-plates,[6] but after denervation the area sensitive to acetycholine spreads along the surface membrane until the entire fiber responds to the

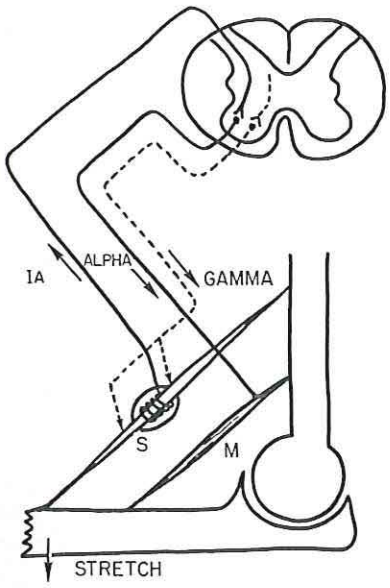

Fig 5. Illustration of the gamma-loop. A gamma motoneuron is shown innervating the intrafusal muscle fibers (m) of a muscle spindle (s). Excitation of gamma motoneurons by pathways, such as segmental reflex paths of tracts descending from the brain (broken line approaching the gamma motoneuron cell body) will cause the contraction of the polar regions of muscle spindles. This results in stretch of the equatorial regions of the spindles, with distortion of the afferent terminals belonging to group Ia fibers. The group Ia fibers that discharge will excite alpha motoneurons to the same and to synergistic muscles through the monosynaptic reflex pathway (and inhibit alpha motoneurons to the antagonist muscles).

agent.[1] This rise in sensitivity develops approximately synchronously along the fiber and is not accompanied by a significant reduction in the end-plate sensitivity. A similar increase in the number of receptors also occurs at autonomic synapses.[21] The early manifestations of radiculopathy described in this paper relate to this denervation supersensitivity and are distinct from those following total denervation.

The segmental nerve is a mixed nerve carrying afferent and efferent fibers, and injury may cause sensory, motor, autonomic, or a combination of these dysfunctions in the corresponding myotome, dermatome, and sclerotome. These areas of reference do not coincide spatially and the resulting multiphasic picture often causes confusion. Referred pain into the dermatome is felt as paresthesia ("numbness," "deadness," or "tingling"), in the myotome as muscle pain and tenderness, and in the sclerotome as a dull, aching or boring, deep pain which is characteristically difficult to localize as it has a tendency to radiate either proximally or distally. To confuse the picture, atypical distributions of sensory disorders conforming to vasal topography may occur in autonomic dysfunction. Skin, connective tissue, musculature, and viscera lying within a vascular nerve region may be involved in the "autonomic irritation syndrome."[9] Every blood vessel is enmeshed by the autonomic nervous system with a double innervation: through the posterior roots (the peripheral nerves), and also through perivasal routes (the rami spinales longi and the sympathetic trunk). The nerves at the surface of the media are predominantly sympathetic, and those in the adventitia are predominantly afferent. Both may undergo degeneration in a nerve root injury. This perivascular nociceptor receptor system is carried into the cancellous interior of the vertebrae, sacrum, and ilium. A parallel system is also present in the epidural and paravertebral veins.[22]

The clinical presentations of a peripheral nerve injury are thus determined by the type of nerve fibers involved (motor, sensory, autonomic, or mixed), their size (large, small, mixed), distribution (proximal, distal, diffuse, neurologic, or vasal[9]), pathology (axonal degeneration, segmental demyelination, mixed) and degree of injury. In the examination of the patient, therefore, signs are sought in both rami (since by definition, a lesion at root

Table 1. Segmental Innervation of the Muscles of the Lower Limb Tested for Tender Motor Points*

Predominant cord segment	*Muscle (segmental innervation)*	*Peripheral nerve*
L2	Sartorius (L2, L3)	Femoral
	Pectineus (L2, L3)	Obturator
	Adductor longus (L2, L3)	Obturator
L3	Quadriceps femoris (L2-L4)	Femoral
L4	Quadriceps femoris (L2-L4)	Femoral
	Tensor fascia latae (L4, L5)	Superior gluteal
	Tibialis anterior (L4, L5)	Peroneal
L5	Gluteus medius (L4-S1)	Superior gluteal
	Semimembranosus (L4-S1)	Sciatic
	Semitendinosus (L5-S1)	Sciatic
	Extensor hallucis longus (L4-S1)	Deep peroneal
S1	Gluteus maximus (L4-S2)	Inferior gluteal
	Biceps femoris, short head (L5-S2)	Sciatic
	Semitendinosus (L4-S1)	Sciatic
	Medial gastrocnemius (S1, S2)	Tibial
	Soleus (S1, S2)	Tibial
S2	Biceps femoris, long head (S1, S2)	Sciatic
	Lateral gastrocnemius (S1, S2)	Tibial
	Soleus (S1, S2)	Tibial

* Most muscles receive their innervation from more than one segment of the spinal cord, as indicated in this table in parentheses. The segments listed on the left are those generally accepted as the predominant source of innervation of the muscles in question, all of which are innervated by the anterior rami whose fibers pass along the nerves indicated on the right. The posterior rami from these same cord segments are distributed to the corresponding levels of the erector spinae muscles, but there is extensive overlapping of the posterior rami.

Table 2A. Study Group A—30 Patients With Low-Back Pain and Tender Motor Points Under Age 30

				First exam (in pain)								Second exam (pain-free)						
				Increased muscle tone		Tender motor points*						Increased muscle tone	Tender motor points					
Patient number	Age/sex	Diagnosis	Roentgenogram report and other reports	Erector spinae	Legs	Backs	Legs	Trophedema	Matchstick test	Saddle sign	Horseshoe sign		Backs	Legs	Trophedema	Matchstick test	Saddle sign	Horseshoe sign
1	30/M	Low-back strain	Degenerative L4-5-S1	++	+	+	+	+	−	+	+	−	−	−	+	−	+	−
2	29/M	Low-back strain	Normal	+	+	+	+	+	−	+	−	−	−	−	−	−	+	−
3	25/M	Low-back strain	Normal	+	+	+	+	+	−	+	−	−	−	+	−	−	+	−
4	29/M	Low-back strain	Normal	−	−	+	+	+	−	−	−	−	−	−	−	−	−	−
5	17/M	Low-back strain	None	+	+	+	+	+	−	+	−	−	−	−	−	+	+	−
6	28/M	Low-back strain and right leg pain	Normal	+	+	+	+	+	+	+	+	−	−	−	+	−	+	−
7	26/F	Low-back strain and bilateral sciatic pain	Normal	++	++	+	+	+	−	−	−	−	−	−	−	−	−	−
8	30/M	Low-back strain and bilateral sciatic pain	Normal	−	−	+	+	−	−	+	−	−	−	−	−	−	+	−
9	28/M	Low-back sprain (with resolved right sciatica)	Normal	−	−	+	−	+	−	+	+	−	−	−	−	−	+	−
10	30/F	Left sciatica	Slight degenerative changes L/S	+	+	−	+	−	−	+	−	+	−	−	−	−	+	+

* Myalgic hyperalgesia maximal at tender motor points.

level must involve both anterior and posterior primary rami), local and referred, in dermatomes, myotomes, and sclerotomes, and related to motor, sensory, or autonomic dysfunction. Late and blatant signs are eschewed in this presentation in order to stress the more subtle irritative responses indicative of early and reversible neuropathy rather than late and severe denervation. The key to the identification and anatomic localization of the level of injury lies in the detection of abnormalities in different structures belonging to the same segment but receiving their ultimate segmental innervation through different peripheral nerves.

SIGNS ASSOCIATED WITH DENERVATION SUPERSENSITIVITY

Autonomic Dysfunction

These signs are the first to be discussed as they are often the ones first seen by the examiner.

Pilomotor Reflex: The autonomic system is a division of the peripheral nervous system and is by definition entirely motor (except for vascular and visceral afferents). Although "automatic" in the sense that most of its functions are carried below the conscious level, it is highly integrated in structure and function with the rest of the nervous system. Autonomic efferent fibers supply the piloerector muscles, smooth muscles of the blood vessels, and sweat glands. When autonomic ganglia and effector muscles are affected, denervation supersensitivity develops. For example, immediately as the patient undresses and as cool air plays on exposed skin, there may be seen a pilomotor effect or cutis anserina ("goose flesh") in the dermatomes of affected segmental levels. We have previously reported on this phenomenon in relation to cervical spondylosis.[10,12] It is essential therefore to watch the patient undress, examining the skin carefully, as the reflex may be present for only a brief moment; but this pilomotor reflex may be reenforced or induced by firm digital pressure over any tender motor point within the affected segment. (The North American practice of providing a female patient with a gown for modesty conceals this sign.)

Table 2B. Study Group A—Patients With Low-Back Pain and Tender Motor Points Ages 31-45

				First exam (in pain)								Second exam (pain-free)						
				Increased muscle tone		Tender motor points*						Increased muscle tone	Tender motor points					
Patient number	Age/ sex	Diagnosis	Roentgenogram report and other reports	Erector spinae	Legs	Backs	Legs	Trophedema	Matchstick test	Saddle sign	Horseshoe sign		Backs	Legs	Trophedema	Matchstick test	Saddle sign	Horseshoe sign
11	39/M	Low-back strain	Normal	−	−	+	+	+	+	−	−	−	−	−	+	−	−	−
12	36/M	Low-back strain superimposed on previous degenerative disease	Minor degree changes L5-S1	+	+	+	+	+	−	+	−	−	−	+	−	−	−	−
13	41/M	Low-back strain (discectomy 7 years ago)	Narrowed disc space L/S	+	+	+	+	+	−	−	−	−	−	−	+	−	+	−
14	42/M	Low-back strain	Degenerative lipping at L3-4, spondylolisthesis L5-S1	+	+	+	+	+	−	+	−	−	−	−	−	−	+	−
15	37/M	Low-back strain	Normal	−	−	+	+	+	−	+	−	−	−	−	−	−	+	−
16	39/M	Low-back strain	Slight degenerative changes L4-S1	−	−	+	+	+	+	−	−	−	−	+	+	−	−	−
17	44/M	Low-back strain	Moderate degenerative changes L/S	+	+	−	+	+	−	+	−	−	−	−	−	−	−	−
18	37 M	Low-back strain	Normal	+	+	+	+	−	−	−	−	−	−	−	+	−	−	−
19	49/M	Low-back pain and "gluteal bursitis"	Degenerative changes L3-4	−	−	+	+	+	−	+	+	−	−	−	+	−	+	−
20	42/M	Bilateral sciatic pain	Degenerative changes L6-S1	+	++	+	+	+	−	−	−	+	−	+	+	−	−	+

* Myalgic hyperalgesia maximal at tender motor points.

Vasomotor Disturbances. In complete division of a peripheral nerve, the denervated region of the skin has a pink or rosy appearance because of vasodilatation brought about by interruption of sympathetic fibers to that part, but in partial nerve injuries vasoconstrictor disturbance due to denervation supersensitivity consists of mottling of the skin, ie, combined pallor and cyanosis.[13] The skin under such conditions has a lower temperature which only becomes discernible after sufficient exposure to cool air (from 10 to 15 minutes). In rare instances, a reticular pigmentation may occur, resembling erythema ab igne even when the patient has not applied local heat.

Sudomotor reflex. In complete denervation, resulting anhidrosis or absence of sweating is the basis for the sweat tests; however, in partial nerve palsies, there is an increased tendency to sweat.[13] This sudorific reflex may occur either spontaneously or under the duress of emotion and during examination when painful movements are reproduced. The sweating usually extends beyond the anatomic confines of the nerve concerned, covering an extensive area which includes the axillae, palms of hands, and soles of feet. (Autonomic postganglionic outnumber preganglionic neurons by a ratio of at least 32:1.[4]) Sweating may be so profuse that droplets of sweat run down from the axillae. A quick inspection of the examination couch sheet soon confirms the distribution of hyperhidrosis.

Table 2C. Study Group A—30 Patients With Low-Back Pain and Tender Motor Points Over Age 46

				First exam (in pain)								Second exam (pain-free)						
				Increased muscle tone		Tender motor points*						Increased muscle tone	Tender motor points					
Patient number	Age/sex	Diagnosis	Roentgenogram report and other reports	Erector spinae	Legs	Backs	Legs	Trophedema	Matchstick test	Saddle sign	Horseshoe sign		Backs	Legs	Trophedema	Matchstick test	Saddle sign	Horseshoe sign
21	51/M	Low-back strain	Degenerative L/S	+	+	+	+	+	+	+	+	−	−	−	+	−	+	−
22	55/M	Low-back strain, left sciatica	Slight degeneration	+	+	+	+	−	−	+	−	−	−	−	−	−	+	−
23	57/M	Low-back strain, left sciatica	Degeneration	++	+	+	+	+	−	+	−	−	−	−	+	−	+	−
24	56/F	Low-back strain, left sciatica	Narrowing L/S	+	+	+	+	−	−	+	+	−	−	+	+	−	+	+
25	46/F	Low-back strain, spinal fusion L/S, L3-4 pain	Narrowing L/S and degenerative L3-4	−	−	+	+	+	+	+	−	−	−	−	+	−	+	−
26	48/M	Low-back strain	Normal	−	−	+	+	+	−	−	−	−	−	−	−	−	−	−
27	55/M	Right low-back strain	Degenerative changes	+	+	+	+	+	−	+	+	−	−	−	−	−	+	−
28	47/M	Left sciatica	Narrowing L/S	−	−	+	+	−	−	+	−	−	−	−	−	−	+	−
29	53/M	Low-back strain (with resolved left sciatica)	Degenerative changes	−	−	+	+	−	−	−	−	−	−	−	−	−	−	−
30	56/M	Left sciatica	Osteoarthritis L4-5	+	+	+	+	+	+	+	−	−	−	−	−	−	+	−

* Myalgic hyperalgesia maximal at tender motor points.

Myalgic and Cutaneous Hyperesthesia. Each skeletal muscle receives one or more nerve supplies. In the limbs, the supply is usually single, but where a nerve more obviously retains its segmental arrangement, eg, the muscles of the abdominal wall, its nerve supply is multiple. After dividing into several small branches, the nerve together with the principal blood vessels of the muscle enter the deep surface of the muscle nearer to its origin at a small, elongated, oval area—the neurovascular hilus. Its terminals are not distributed to the entire muscle but are confined to a fairly narrow transverse motor band near the center of the muscle.[5] The skin region overlying this motor band is known as the "motor point" and is the site where an innervated muscle is most accessible to percutaneous electrical excitation at the lowest intensity. Myalgic hyperalgesia or muscle pain is mediated by bare nerve endings in fascia and pressure-pain nociceptors around blood vessels. These may become supersensitive secondary to altered autonomic activity[9,19] or to loss of selective inhibitory input from nonnociceptive mechanoreceptors. (The mechanism is probably similar to that of cutaneous hyperesthesia in painful syndromes[15] and postherpetic neuralgia.) Nociceptor afferents are abundant around blood vessels[21] and tenderness is therefore greatest at the neurovascular hilus deep to the motor point. Motor points are fixed anatomic sites and easily found. (Charts showing their distribution are generally available[4]: The earliest chart was prepared by the neurologist Wilhelm Erb in 1882.) We have previously reported[11] that motor points are not tender in the normal individual, but mild and transient points may occasionally be found in asymptomatic subjects after unusual activities such as jogging. Moderately tender points are usually present in individuals who give a history of a vulnerable back or who have had lesser degrees of trauma. Acutely tender motor points are almost constantly found in patients having radiculopathy with the degree of tenderness and the number of points paralleling the patient's condition. To be aware of tenderness at motor points may help

Table 3A. Study Group B—30 Patients With Low-Back Pain Without Tender Motor Points Under Age 30

				First exam (in pain)								Second exam (pain-free)						
				Increased muscle tone		Tender motor points						Increased muscle tone	Tender motor points					
Patient number	Age/ sex	Diagnosis	Roentgenogram report and other reports	Erector spinae	Legs	Backs	Legs	Trophedema	Matchstick test	Saddle sign	Horseshoe sign		Backs	Legs	Trophedema	Matchstick test	Saddle sign	Horseshoe sign
1	26/M	Low-back strain	Normal	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
2	22/M	Low-back strain	Normal	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
3	25/M	Low-back strain	Normal	–	–	–	–	+	–	+	–	–	–	–	slight	–	+	–
4	20/M	Low-back strain	Normal	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
5	23/M	Right L2-4 muscular strain	None	+	–	+	–	+*	–	–	–	–	–	–	–	–	–	–
6	19/M	Rotational strain	Normal	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
7	22/M	Low-back strain	Normal	–	–	+	–	+*	–	–	–	–	–	–	slight	–	–	–
8	22/M	Low-back strain	Normal	–	–	–	–	–	–	+	–	–	–	–	–	–	+	–
9	18/M	Low-back strain	Normal	–	–	+	–	+†	–	–	–	–	–	–	slight	–	–	–
10	24/M	Contusion	Normal	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–

* Trophedema found unilaterally on side of pain.

† Trophedema found only in the back at level of pain.

with the diagnosis of pain and tenderness around the hip. A gluteus medius motor point over the upper lateral quadrant of the buttock is frequently attributed to "gluteal bursitis," a tensor fascia motor point to trochanteric bursitis and tenderness at the gluteus maximus motor points (focal) has been mistaken for a tender sciatic nerve (linear) (Figures 1 and 2).

Trophic Disturbances. Nutritional changes form an important part of any neurologic disorder and may occur in the skin, nails, subcutaneous tissues, muscles, bones, and joints. In addition to a neurologic basis, factors such as activity, blood supply, and lymph drainage are involved in the causation of trophic changes. When a peripheral nerve is completely interrupted, the skin loses its delicate indentations, becomes inelastic, smooth, and shiny; but when interruption is partial, trophedema occurs.[13] There is gradual fibrosis of the subcutaneous tissue and overlying skin tends to be fissured and prone to heavy folds.[13] This alteration in the quality of skin produces a "peau d'orange" effect similar to that described for malignant lumps of the female breast, and likewise is accentuated when the skin is gently squeezed together or when the back is fully extended (Figure 3). Trophedematous subcutaneous tissue has a boggy, inelastic texture when rolled between thumb and finger, distinguishable from subcutaneous fat. When a patch of skin and subcutaneous tissue of a few centimeters in diameter is gently squeezed together, instead of immediately forming a fold of flesh, trophedematous tissue does not budge, or it finally yields altogether with a sudden expanding movement similar to that of inflating a rubber dinghy or air mattress. Trophedema is nonpitting to digital pressure, but when a blunt instrument, eg, the end of a matchstick is used, the indentation produced is clear-cut and persists for several minutes (Figure 4), distinctly longer than that over normal skin. This "matchstick test" may be positive, yielding deep indentations over an extensive area (commonly over the low back and hamstrings), or in mild cases may only yield slight indentations of skin overlying a tender motor point or the neurovascular hilus.

Signs Associated with Dysfunction of the Muscle Nerve

The musculoskeletal system is the most massive system of the body and therefore receives the largest efferent outflow from the central nervous system. Likewise, the predominant input to the central nervous system is also the sensory input from the musculoskeletal system. Sensory fibers arise from nerve endings in a variety of somatic structures in myofascial and articular tissues, entering the cord via the dorsal root to be routed to various centers of the nervous system including the cerebral cortex and the autonomic nervous system. This sensory input from the musculoskeletal system is so extensive, intensive, and unceasing as to be the dominant influence on the central nervous system and therefore the person as a whole.

The motor fibers of the muscle nerve comprise the large myelinated alpha-efferents of the anterior horn

Table 3B. Study Group B—30 Patients With Low-Back Pain But Without Tender Motor Points Ages 31-45

				First exam (in pain)								Second exam (pain-free)						
				Increased muscle tone		Tender motor points						Increased muscle tone	Tender motor points					
Patient number	Age/ sex	Diagnosis	Roentgenogram report and other reports	Erector spinae	Legs	Backs	Legs	Trophedema	Matchstick test	Saddle sign	Horseshoe sign		Backs	Legs	Trophedema	Matchstick test	Saddle sign	Horseshoe sign
11	32/M	Low-back strain	None	–	–	–	–	–	–	+	–	–	–	–	–	–	+	–
12	44/M	Low-back strain	None	+	–	–	–	+	–	+	–	–	–	–	slight	–	+	–
13	40/F	Low-back strain	Normal	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
14	39/M	Contusion	Early degenerative changes L5-S1	+	–	+	–	+*	–	–	–	–	–	–	–	–	–	–
15	42/M	Muscular strain	None	–	–	–	–	–	–	+	–	–	–	–	–	–	+	–
16	45/M	Low-back strain	Normal	–	–	–	–	+	–	–	–	–	–	–	slight	–	–	–
17	40/F	Low-back strain	Normal	–	–	–	–	+	–	+	–	–	–	–	–	–	+	–
18	36/M	Acute lumbar strain	Normal	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
19	33/M	Right side lumbar strain	Normal	+	–	+	–	+†	–	–	–	–	–	–	–	–	–	–
20	35/M	Low-back strain	Normal	–	–	–	–	+	–	+	–	–	–	–	–	–	+	–

* Trophedema found only in the back at level of pain.
† Trophedema found unilaterally on side of pain.

cells which supply extrafusal muscle fibers and the smaller myelinated gamma-efferents which supply the muscle spindles. The motor unit is defined as the anterior horn cell, its branched axon, and the muscle fibers which it innervates. In disorders of the alpha motoneuron there is atrophy of some muscle fibers and reinnervation of others by surviving motoneurons. Supersensitivity of single denervated fibers to circulating acetycholine may be seen in electromyography as fibrillation potentials, but clinical assessment of early weakness in muscle is unreliable and not usually detectable unless atrophy has involved a great number of muscle fibers. (An analysis of the density of the electromyographic interference pattern during maximum effort is a valuable procedure but not accurate quantitatively. There are now electrophysiologic methods for motor unit estimations.[16])

The muscle spindle, about 3 millimeters in length, is enclosed in connective tissue. Its intrafusal muscle fibers are innervated by the gamma motoneurons, and its afferent neurons are from the primary endings or annulospiral endings wound around the intrafusal fibers and the secondary flower-spray endings on both sides of the annulospiral endings. These endings are sensitive to stretch of the central portion of the spindle and form part of the essential feedback mechanism by which skeletal muscle and resting muscle tonus are controlled. The afferent discharge of the spindle via the dorsal root on the motor neurons of the same muscle is excitatory (Figure 5). Thus, when a muscle is stretched, eg, the hamstrings as in the straight leg-raising test, the spindles reflexedly stimulate it to contract and resist stretching. Conversely, shortening of the muscle favors relaxation. Through interneurons and collaterals, spindle activity also determines the activity of antagonists and synergists. The spindle is, in effect, the sensory component of the muscle-stretch or myotatic reflex, commonly misnamed the "tendon reflex" test. Denervation supersensitivity of this "gamma-alpha loop" may lead to hyperexcitation of muscle and contribute toward the spasm seen in neuropathy. Palpation for muscle spasm or "tightening" therefore is an early irritative sign indicative of neuropathy which occurs before depression or interruption of the deep myotatic reflex from denervation. Normal resting muscle is soft, yet has a certain resilience, but its shape and boundaries are not easily discernible to palpation; however, the slight shortening and contraction of early muscle spasm may be soon detected with a little practice, though this early spasm is easily overlooked unless a deliberate scan of all major muscles is undertaken (Table I).

MATERIALS AND METHODS

In this prospective study, 60 patients and 60 control subjects were examined. Since lumbar spondylosis is a progressive aging process, they were divided into three

Table 3C. Study Group B—30 Patients With Low-Back Pain But Without Tender Motor Points Over Age 46

				First exam (in pain)								Second exam (pain-free)						
				Increased muscle tone		Tender motor points						Increased muscle tone	Tender motor points					
Patient number	Age/sex	Diagnosis	Roentgenogram report and other reports	Erector spinae	Legs	Backs	Legs	Trophedema	Matchstick test	Saddle sign	Horseshoe sign		Backs	Legs	Trophedema	Matchstick test	Saddle sign	Horseshoe sign
21	64/M	Contusion	Degenerative L1-3	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
22	56/M	Contusion	Compression fracture L1 (healed)	–	–	–	–	+	–	+	–	–	–	–	slight	–	+	–
23	53/M	Low-back strain	Normal	+	–	–	–	+*	–	–	–	–	–	–	–	–	–	–
24	56/M	Low-back strain	None	–	–	–	–	–	–	+	–	–	–	–	–	–	+	–
25	55/M	Low-back strain	Normal	+	–	–	–	+	–	+	–	–	–	–	–	–	+	–
26	65/M	Low-back strain, contusion	None	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
27	55/M	Low-back strain	Spondylolisthesis L5-S1	–	–	–	–	+	–	–	–	–	–	–	slight	–	–	–
28	54/M	Low-back strain	Osteoarthritis of L4-S1	–	–	–	–	–	–	+	–	–	–	–	–	–	+	–
29	47/F	Low-back strain	Early degenerative lipping L5-S1	–	–	–	–	+	–	+	–	–	–	–	+	–	+	–
30	59/M	Low-back strain	Gross osteoarthritic changes in L/S junction	–	–	–	–	+	–	–	–	–	–	–	–	–	–	–

* Trophedema found unilaterally on side of pain.

age groups: 30 years and under, 31–45 years, and 46 years and over. The 60 patients of Groups A and B (Tables 2 and 3) were randomly selected from those admitted to the Outpatient Rehabilitation Clinic of the Workers' Compensation Board of British Columbia between November 1, 1976 and March 30, 1977. The 30 patients with secondary backache in Group A were admitted as having low-back sprain; that is, they had low-back pain, with or without radicular symptoms but without any localizing signs except for myalgic hyperalgesia or tender motor points (an early sign for radiculopathy). The 30 patients with primary backache in Group B also had low-back sprain but did not have any tender motor points. Both groups were examined during the period when they were in pain and again when they were pain free. Apart from their back pain, all were in good health. Patients were given a physical examination and roentogenograms of the lumbosacral spine including their oblique views were obtained in 53 patients. The examination of the low back emphasized the subtle signs and incorporated our tender motor points procedure.[11] This procedure examines for muscle tenderness maximum at motor points. In accordance with established electromyographic principles, representative muscles of the second through the fifth lumbar and the first two sacral myotomes are examined, selecting muscles with accessible motor points (Table I). Trauma to a nerve root causes irritation or degeneration of nerve fibers, or both. These lesions may be detected during electromyography as increased insertional activity, polyphasic action potentials, fasciculation potentials, fibrillations, and positive sharp waves, or in the procedure described here, as tender motor points in muscles of the affected myotome, even though their segmental in-

Table 4. Control Group C (30 Males)—No Low Back Pain

Age Group	Patient number	Increased muscle tone	Tender motor points: Backs	Tender motor points: Legs	Trophedema	Matchstick test	Saddle sign	Horseshoe sign
Under age 30	1	−	−	−	−	−	+	−
	2	−	−	−	+	−	+	−
	3	−	−	−	−	−	−	−
	4	−	−	−	−	−	−	−
	5	−	−	−	+	−	+	−
	6	−	−	−	+	−	+	−
	7	−	−	−	−	−	−	−
	8	−	−	−	−	−	−	−
	9	−	−	−	−	−	−	−
	10	−	−	−	+	−	+	−
Ages 31–45	11	−	−	−	−	−	−	−
	12	−	−	−	−	−	+	−
	13	−	−	−	+	−	+	−
	14	−	−	−	−	−	+	−
	15	−	−	−	−	−	−	−
	16	−	−	−	−	−	−	−
	17	−	−	−	+	−	−	−
	18	−	−	−	−	−	−	−
	19	−	−	−	−	−	−	−
	20	−	−	−	−	−	−	−
Over age 46	21	−	−	−	+	−	+	−
	22	−	−	+	+	−	+	−
	23	−	−	−	−	−	−	−
	24	−	−	−	−	−	−	−
	25	−	−	−	+	−	+	−
	26	−	−	−	+	−	+	−
	27	−	−	−	−	−	−	−
	28	−	−	−	−	−	+	−
	29	−	−	−	−	−	−	−
	30	−	−	−	−	−	−	−

nervation comes through different peripheral nerves. Thus, the fourth lumbar myotome includes the following muscles: the anterior tibial, innervated by the fourth and fifth lumbar roots coming through the deep peroneal nerve; the tensor fasciae latae, innervated by the fourth and fifth lumbar roots coming through the superior gluteal nerve; and the quadriceps femoris, innervated by the second, third, and fourth lumbar roots coming through the femoral nerve. Abnormal findings in several muscles of one myotome differentiate the lesion from a peripheral neuropathy involving one nerve. Examination of the paraspinal muscles which are innervated by the posterior primary rami is also necessary to confirm that the pathologic process involves the segmental nerve at the root level, in which case muscles innervated by both anterior and posterior rami are involved.

Control Groups C and D, also divided into three age groups, consisted of 30 men and 30 women (Tables 4 and 5), who were healthy and pain-free subjects drawn from the lay staff of the Workers' Compensation Board of British Columbia. They were examined on the one occasion only. No roentgenograms were obtained in these control subjects as radiologic findings in pain-free subjects are generally of no relevance.

For the purpose of this study, "trophedema" is defined as any alteration from the normal of skin elasticity

Table 5. Control Group D (30 Females)—No Low Back Pain

Age Group	Patient number	Increased muscle tone	Tender motor points: Backs	Tender motor points: Legs	Trophedema	Matchstick test	Saddle sign	Horseshoe sign
Under age 30	1	−	−	−	−	−	+	−
	2	−	−	−	−	−	+	−
	3	−	−	−	−	−	−	−
	4	−	−	−	−	−	+	−
	5	−	−	−	−	−	−	−
	6	−	−	−	+	−	−	−
	7	−	−	−	−	−	+	−
	8	−	−	−	−	−	−	−
	9	−	−	−	−	−	+	−
	10	−	−	−	−	−	+	−
Ages 31–45	11	−	−	−	−	−	−	−
	12	−	−	−	+	−	+	−
	13	−	−	−	−	−	−	−
	14	−	−	−	−	−	+	−
	15	−	−	−	−	−	−	−
	16	−	−	−	−	−	+	−
	17	−	−	−	+	−	−	−
	18	−	−	−	−	−	−	−
	19	−	−	−	+	−	+	−
	20	−	−	−	−	−	−	−
Over age 46	21	−	−	−	−	−	−	−
	22	−	−	−	−	−	−	−
	23	−	−	−	−	−	+	−
	24	−	−	−	−	−	+	−
	25	−	−	−	+	−	+	−
	26	−	−	−	−	−	−	−
	27	−	−	−	−	−	+	−
	28	−	−	−	+	−	+	−
	29	−	−	−	−	−	−	−
	30	−	−	−	+	−	−	−

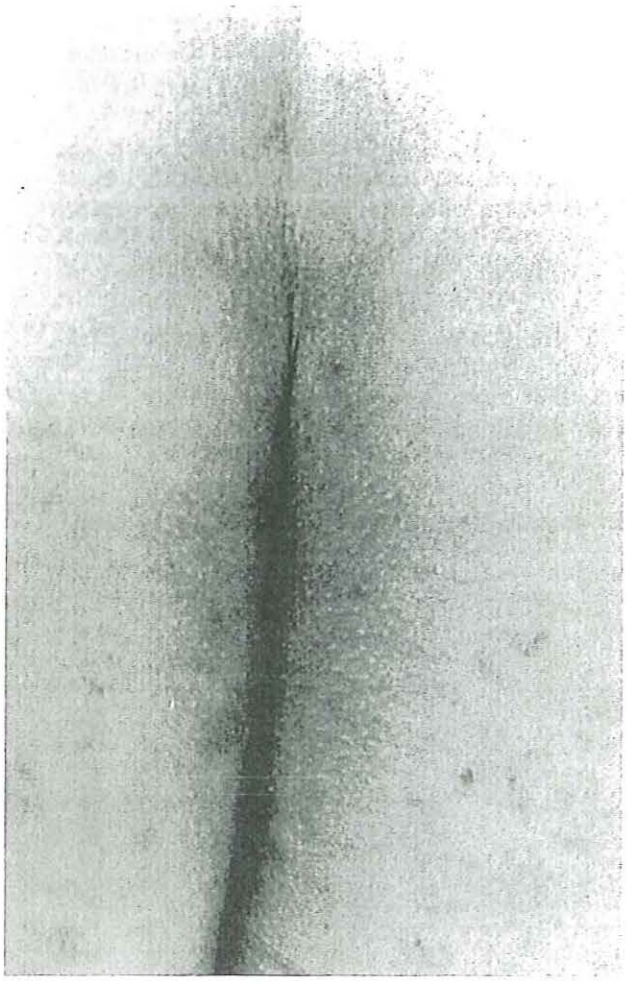

Fig. 6. The "saddle sign" pilomotor effect at the S4-5 dermatomes.

and subcutaneous tissue consistency. A patch of skin and underlying subcutaneous tissue of about 5 cm in diameter is gently squeezed and rolled between thumb and index finger. Elasticity and texture of the skin and consistency of the subcutaneous tissue over the painful site is compared with that over pain-free areas, noting any loss of the normal delicate indentations of the skin, peau d'orange effect, and resistance to formation of free skin folds. Lesser degrees are distinguished from severe trophedema by the matchstick test. The end of a matchstick or similar blunt instrument is pushed firmly into the skin, spaced out at regular intervals approximately 2 cm apart and the indentations noted. In severe trophedema the indentations are deep, well-defined, and persist for several minutes (Figure 4B), distinctly longer than that over normal areas (Figure 4A).

The pilomotor effect is best observed under an oblique light. Prior to this study we have noted that a pilomotor reflex is not uncommonly seen in the saddle distribution of S4–5 dermatomes in both normal individuals as well as patients with low-back pain. Suspecting that the reflex in this area may be of no clinical significance because of its common occurrence, we have in this study described it as the "saddle sign" (Figure 6) to distinguish it from the less commonly seen pilomotor effect at higher segmental dermatomes (especially those of S1–3) (Figure 7). This latter was labeled as the "horseshoe sign" because of its topographic distribution (see Figure 8). The sudomotor effect is not included here as it may be due to emotional causes, although in practice we do take note of excessive sweating following painful movements. Minor variations in skin temperature are likewise not recorded as they are difficult to detect by touch. A slight increase of muscle tone or muscle tightening is recorded as positive (+) when the muscle is palpably firmer and its shape and size easily discernible; a severe increase of muscle tone or obvious spasm is recorded as (++). We have found that the gluteus medius, hamstrings, and calf muscles are accessible to palpation, comparing muscle tone on the painful side to the asymptomatic.

RESULTS

Group A

Group A consisted of 30 patients with low-back sprain and tender motor points in both rami (Table 2) and secondary low-back pain. These 30 patients (27 men and 3 women) did not have the classic signs or other localizing signs but all had myalgic hyperalgesia and showed tender motor points in the back and lower limbs indicative of some degree of radiculopathy. Other indicators of neuropathy are presented in Table 6.

Group B

Group B consisted of 30 patients with low-back sprain but without tender motor points in both rami (Table 3) and primary-low back pain. These 30 patients also did not have the classic signs or other localizing signs, nor did they have any tender motor points in the legs. Findings are shown in Table 7.

Groups C and D

These were controls, consisting of 30 men in Group C and 30 women in Group D. Only one of these control subjects had mildly tender motor points in the lower limbs. None showed increased muscle tone. Seventeen (28.3%) showed a mild degree of trophedema but the matchstick test for severe trophedema was negative. Twenty-eight subjects (46.6%) showed a pilomotor reflex in the saddle distribution of S4–5 dermatomes but none had this effect at higher dermatomes.

DISCUSSION

It is generally agreed that virtually everyone eventually has some degenerative joint disease in the low back, but that as a rule, problems only arise when the degeneration has reached a certain degree and some incident, which may be minor, precipitates symptoms.

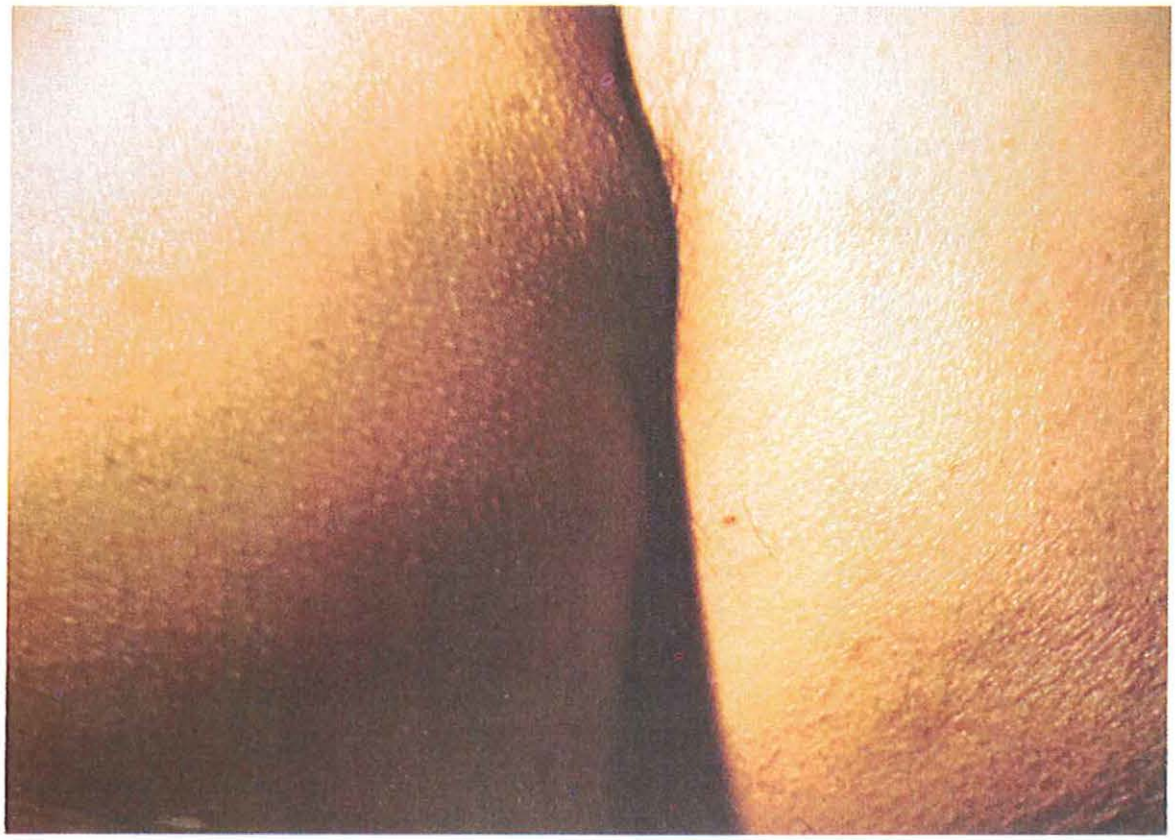

Fig 7. The "horshoe sign" pilomotor effect extending beyond S4-5 dermatomes, here seen unilaterally on left side only.

Symptoms may be localized to the low back affecting local pain-sensitive structures, or if the nerve root is involved, the pain is then transmitted intrasegmentally to distal locations including the dermatome, myotome, and sclerotome.

Conventional localizing signs for nerve root involvement are late to appear and often absent in patients with low-back pain; in these patients a working diagnosis of low-back sprain is inevitably used. Although the phenomenon of denervation supersensitivity is well known to physiologists and clinicians involved in the care of patients with peripheral nerve diseases, its related physical signs have not been applied to the clinical investigation of low-back pain.

In this study it was found that one or more of these early and subtle signs of nerve irritability occurred in all 30 patients with secondary low-back pain (Group A). These signs were present when the patients were in pain and largely resolved when they became pain free. Increased muscle tone was seen in muscles of both rami, indicating the lesion to be at root level, as paravertebral muscle spasm and tightening of gluteal, hamstrings, or calf muscles in 19 (63.3%) patients. Slight trophedema was present in 23 (76.6%), and a positive matchstick test to indicate moderate to severe trophedema occurred in 6 (20.0%) patients. The saddle sign was present in 21 (70.0%) patients but the horseshoe sign in only 7 (23.3%).

In Group B (30 patients with primary low-back pain but without significant tender motor points), at least one of the early signs was seen in 21 (70.0%) patients. Increased muscle tone was seen in 6 (20.0%) patients (as compared to 19 [63.3%] of Group A and none in the control groups) but it was found only in the erector spinae muscles suggesting that the irritation was primarily a local phenomenon or only affected the posterior primary ramus. There was slight trophedema in 16 patients (53.3%) but severe trophedema as shown by a positive matchstick test was not found. In 4 patients, the mild trophedema was unilateral on the painful side; in 2 other patients, this was confined to the levels at which the injury occurred. The saddle sign was present in 12 (40.0%) patients but the horseshoe sign was not seen.

In Control Groups C and D mild trophedema was found in 17 (28.3%) subjects, and the pilomotor reflex in the saddle distribution occurred in 28 (46.6%), which confirmed our suspicion that this is not an uncommon finding in asymptomatic individuals, but the pilomotor effect at higher segmental levels was not seen.

We conclude that these subtle signs were almost con-

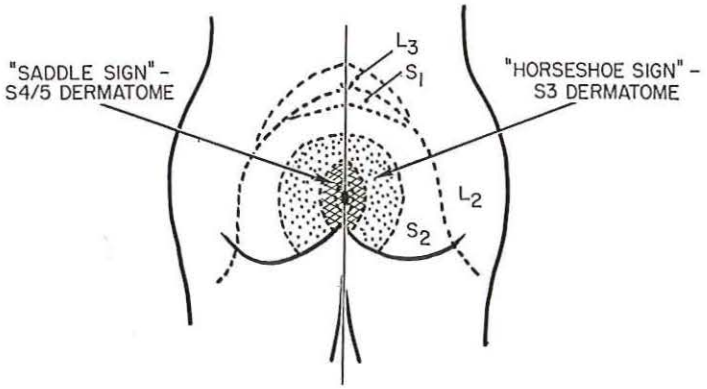

Fig 8. Dermatomes of the posterior sacral rami (note that L4 and L5 dermatomes are not represented in the low back). Areas of the "saddle sign" and the "horseshoe sign" are graphically shown.

stant companions of patients with secondary low-back pain and were less often seen in patients with primary or mechanical low-back pain, in which case the signs were also less severe. Mild trophedema may be of clinical value when it occurs localized to the level of injury in the low back or unilaterally on the symptomatic side. The horseshoe sign may be of significance as it occurred only in Group A patients but not in Group B or control subjects. Since the saddle sign and lesser degrees of generalized trophedema also frequently occurred in the control groups, these signs may be too sensitive to be of any clinical value, though their presence may identify that group of individuals possessing a vulnerable back and few symptoms or signs[17] and may suggest the possible presence of early and insidious neuropathy, even in the youngest age group. It has recently been shown that pain in peripheral neuropathy is primarily associated with the rate and kind of nerve fiber degeneration, though not all patients with peripheral neuropathy necessarily have spontaneous pain.[7] Patients with acute breakdown of myelinated fibers (either wallerian or axonal degeneration) tend to have pain more often and to a greater degree than do subjects with more chronic forms of nerve fiber degeneration. Pain is probably not caused simply by the different proportions of large to small fibers remaining after nerve degeneration as anticipated by the gate theory of pain. Low-back pain is not the inevitable result of the gradual attritions of age, wear, and tear which are constant with us all, but symptoms will tend to manifest themselves when there is an acute breakdown superimposed upon a chronic or abnormal breakdown of myelinated fibers, whatever its primary cause. Most acute compression peripheral neuropathies involving a healthy nerve are not painful, or are only briefly so (peroneal palsy and radial nerve "Saturday night" palsy). It has also been shown in experiments that acute mechanical injury to a dorsal root does not produce a sustained discharge[20] unless there has been preexisting minor chronic injury to the nerve or unless the dorsal root ganglion is involved.[14] It is common knowledge that in asymptomatic subjects, the mere appearance of degenerative changes in roentgenograms is not of much clinical significance, but in these people, disability after back injury will tend to be prolonged and signs of radiculopathy more commonly found.[11] Our findings of early signs in about half our

Table 6. Indicators of Neuropathy in 30 Patients in Group A

	First examination (patients in pain)		*Second examination (patients pain-free)*	
Indicator	*No. patients*	*%*	*No. patients*	*%*
Increased muscle tone or spasm	19	63.3	2	6.6
Mild trophedema (matchstick test negative)	23	76.6	12	40.0
Severe trophedema (matchstick test positive)	6	20.0	1	3.3
Pilomotor reflex in S4-5 dermatomes (saddle sign)	21	70.0	20	66.6
Pilomotor reflex in other dermatomes (horseshoe sign)	7	23.3	3	10.0

Table 7. Examination Findings for 30 Patients in Group B

	No. patients	*%*
Increased muscle tone or spasm	6	20.0
Mild trophedema (matchstick test negative)	16	53.3
Severe trophedema (matchstick test positive)	0	0.0
Pilomotor reflex in S4-5 dermatomes (saddle sign)	12	40.0
Pilomotor reflex in other dermatomes (horseshoe sign)	0	0.0

control groups, including the young, may be ominous as they imply that they are a large and vulnerable group.

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Address reprint requests to

Dr. C. Chan Gunn
Clinical Research Unit
Rehabilitation Clinic
Workers' Compensation Board of B.C.
6951 Westminster Highway
Richmond, B.C. V76 1C6
CANADA

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