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Sensorimotor control of the spine

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Abstract

The spinal viscoelastic structures including disc, capsule and ligaments were reviewed with special focus on their sensory motor functions. Afferents capable of monitoring proprioceptive and kinesthetic information are abundant in these structures. Electrical stimulation of the lumbar afferents in the discs, capsules and ligaments seems to elicit reflex contraction of the multifidus and also longissimus muscles. The muscular excitation is pronounced on the level of excitation and with weaker radiation 1 to 2 levels above and below. Similarly, mechanical stimulation of the spinal viscoelastic tissues excites the muscles with higher excitation intensity when more than one tissue (ligaments and discs for example) is stimulated. Overall, it seems that spinal structures are well suited to monitor sensory information as well as to control spinal muscles and probably also provide kinesthetic perception to the sensory cortex. © 2002 Published by Elsevier Science Ltd.

Keywords: Spine; Reflex; Muscle; Receptors; Disc; Ligament; Capsule

1. Introduction

Low back pain is one of the most common medical problems of the middle-aged population, and from society's point of view, it is the most costly musculoskeletal disease in industrialized countries today [2,45,56]. In the majority of cases, the origin of the pain remains obscure. Much of low back pain is thought to arise from damage to the intervertebral disc or the zygapophysial joints, either directly through traumatic injuries or disc prolapse, or indirectly via degenerative processes that transmit unfavourable loading patterns to other spinal structures, e.g. ligaments, tendons and supporting musculature as well as to the sacroiliac joint [26,27,37,50,57,58,60,63]. The mechanisms behind spinal disorders can either act as single variables or in combination. Derangement in the lumbar intervertebral disc and zygapophysial joints can contribute at the same segmental level or at different levels and be independently painful causing direct and referred pain. A similar situation can arise when the sacroiliac joint system itself is disturbed or affected indirectly via derangement in the lumbar spine or its supporting structures [5,15,16,58,41,44]. The relationship between pain and structural derangement is still not fully understood.

2. Reflexes from the different lumbosacral structures

The background hypothesis for the work presented here is that lesions in the avascular supporting structures, depending on location, size, and degree of inflammation, can cause perturbation in the proprioceptive function of the different receptors and result in increased and prolonged muscle activation that may cause pain. Irritation of low threshold nerve endings in the sacroiliac joint, intervertebral disc or the zygapophysial joint tissue may trigger a reflex activation of the gluteal and paraspinal muscles that may become painful over time. To come closer to a solution to many low back problems, a better understanding of muscle function and their interactions with the passive structures is needed. In the following studies, the electromyographic response of the multifidus musculature to nerve stimulation in the peripheral part of the annulus fibrosus lumbar intervertebral discs, the capsule of the zygapophysial joint and the sacroiliac joint was measured.

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2.1. The lumbar intervertebral disc

The intervertebral disc itself may be an intrinsic source of pain that originates from mechanical or chemical disturbances. It has well been established that the intervertebral disc is innervated and that there are pain potentials in the outer part of the annulus fibrosus [18,22,24,29,42,57]. In the superficial layers of the disc, nerves form simple free endings in the fetal stage, which increase in number as the fetus matures. During the postnatal period, various types of receptors develop, and in adult material, five types of nerve terminations can be found. The distribution of the receptors on the surface of the annulus changes with age [42,64]. The receptors within a given disc are not uniformly distributed [46]. After postnatal development, there is a relative decrease in the number of receptors in the anterior region, so that in adults the greatest number of endings occurs in the lateral region of the disc; a smaller number occurs in the posterior region, and the least number occurs in the anterior region [42]. The source of the nerve endings in the lumbar discs is the lumbar sinuvertebral nerves and branches of the lumbar ventral rami and the grey rami communicantes [6,11,57]. Each lumbar sinuvertebral nerve supplies the disc at its level of entry into the vertebral canal and the disc above. The posterolateral corner of each lumbar disc receives branches from the lumbar ventral rami that originate just outside the intervertebral foramina. This region of the disc receives a branch from the grey ramus communicantes before its connection with the ventral ramus. Branches of the grey rami communicantes innervate discs at various levels. This gives the possibility that discogenic pain from a single level may involve more than one recurrent branch of the spinal nerves. Although the lumbar intervertebral discs are innervated by branches of the sympathetic nervous system, it does not necessarily mean that afferent fibers from these structures return to the nervous system via the sympathetic trunk [10,11,35]. It has been suggested that somatic afferent fibers from the discs simply use the course of the rami communicantes to return to the ventral rami. The presence of nerve endings in the lumbar intervertebral disc raises the question as to their function [36,42]. Any free nerve ending associated with blood vessels in the disc may be considered as having a motor or vasosensory function, but because the nucleus fibrosus contains so few blood vessels, this is likely to be the function for the majority of the nerve fibers in the disc [8]. Although there is no absolute explicit evidence that disc pain can be ascribed to a particular type of nerve ending in the disc, there is evidence suggesting that the disc can be associated with pain [6,26,28,29,60,63].

2.2. The zygapophysial joints

Together with the disc, the lumbar zygapophysial joints are responsible for the mechanical guidance of the motion segment [38,39]. The lumbar zygapophysial joints are formed by the inferior articular processes of one lumbar vertebra with the superior articular processes of the lower adjacent vertebra. The joints exhibit features typical of synovial joints, the articulating surfaces are covered by articular cartilage. Sensory innervation of the joint is derived from the posterior ramus of the spinal nerves [7,10,24,29]. This branch supplies filaments to the capsule surrounding the zygapophysial joint, which is attached to the articular processes just beyond the margin of the articular cartilage. Anteriorly, the fibrous capsule of the joint is replaced entirely by the ligamentum flavum, which attaches close to the articular margin. The enclosing joint capsule is thick dorsally and is reinforced by some of the deep fibers of the multifidus muscle [10,40].

2.3. The sacroiliac joint

The mechanism behind sacroiliac joint pain, instability and/or subluxation has been suggested, and despite any proven clinical findings or clearly defined function of the joint, sacroiliac dysfunction has been established as a clinical entity [12,15,16,17,44]. The sacroiliac joint appears to be richly innervated, although there seems to be some uncertainty as to the exact innervation pattern [21,51]. The predominant innervation is via the L4-S1 nerve roots, with some contribution from the superior gluteal nerve. It has also been shown that the joint receives fine nerve branches from the dorsal rami of the S1–S4 spinal nerves [21]. The upper ventral portion of the sacroiliac joint is believed to be supplied by the ventral ramus of the L5 nerve, whereas the lower ventral portion is mainly supplied by the ventral ramus of the S2 nerve. Various nerve fibers as well as a broad selection of sensory receptors and mechanoreceptors have also been found in the joint region [21,25].

2.4. The supporting musculature

The lumbar musculature exerts various forces on the spinal motion segments. Each muscle not only acts as a moment-producer, but also generates compressive and shear forces. The functions of these muscles are to stabilize the spine while providing mobility [8,9,32]. The recruitment patterns for these muscles is not well established. The multifidus muscles are the longest and most medial of the lumbar back muscles. They consist of repeating series of fascicles that originate from the laminae and the spinous processes of the lumbar vertebrae and display consistent patterns of attachments caudally [11]. The key feature of the morphology of the lumbar multifidus is that its fascicles are arranged polysegmentally. Each lumbar vertebra is supplied with a group of fascicles that radiate from its spinous process, anchoring it below to mamillary processes. The fibers of the multifidus are designed to act together on a single spinous process of two to four levels. All the fascicles originating from the spinous processes of a given vertebra are innervated by the medial branch of the dorsal ramus that originates from below that vertebra. The muscles that act directly on a particular vertebral segment are innervated by the nerve of that segment [7,10,40]. Although the paraspinal musculature has been studied numerously, its role in the formation of low back pain is far from clear [13]. Electromyographic (EMG) evaluation of various back lesions have contributed to the current understanding of low back pain [19,23,30,31,59]. The clinical picture often seen is one of tense and painful paraspinal muscles and reduced flexibility in the lumbar spine. This is thought to be caused by reflex stabilization by the paraspinal muscles.

3. Neuromuscular interaction between the spinal structures

3.1. Methodological considerations

Eighty adolescent domestic pigs, initially weighing 45–50 kg, were used in the studies. Each animal was sedated by an intramuscular injection of Ketamine (30 mg/kg body wt; Parke–Davis, UK). A venous catheter was installed, and the animal was tracheotomized and put on a respirator. The animal was anaesthetized with intravenously administered-chloralose (100 mg/kg; Sigma, USA). Additional maintenance doses were given every l5 min. Ringer's solution was continuously infused through the catheter and the pulse rate was monitored throughout the experiments.

3.1.1. Stimulation of the intervertebral disc and zygapophysial joints

With the animals in a prone position on the operating table, a midline longitudinal incision was made over the entire lumbar spine to expose the multifidus musculature. Custom-made bipolar stimulating electrodes were implanted unilaterally into the lateral periphery of the annulus fibrosus of the disc and the zygapophysial joint capsule of the L1–L2 motion segment (Figs. 1 and 2). Exact placement of the electrodes was confirmed when the lumbar spine was removed and inspected after completion of the experiment. The initial experimental protocol consisted of unilaterally and separately stimulating the disc annulus and the zygapophysial joint capsule. Additionally, the effect of introducing local anaesthesia (Lidocaine) into the stimulated zygapophysial joint as well as the effect of subperiosteal detachment of the paraspinal muscles were investigated [26]. Another protocol was performed in order to investigate the effects of mechanical capsular stretch, via fluid injection into the joint, on the reflexive muscle response [27]. This experimental protocol consisted of first stimulating the annulus fibrosus with a set of six stimulation and simultaneously recording the Motor Unit Action Potentials (MUAP) from the EMG electrodes. This established the control values for each of the electrode sites. A syringe (0.8 mm needle diameter) containing 1 ml of saline was then introduced into the zygapophysial joint, and an additional set of stimulations was made. These measurements were used to establish whether or not introduction of the syringe needle into the zygapophysial joint could alter the EMG response. The saline was then injected into the zygapophysial joint and the needle was left in place so as to prevent leakage.

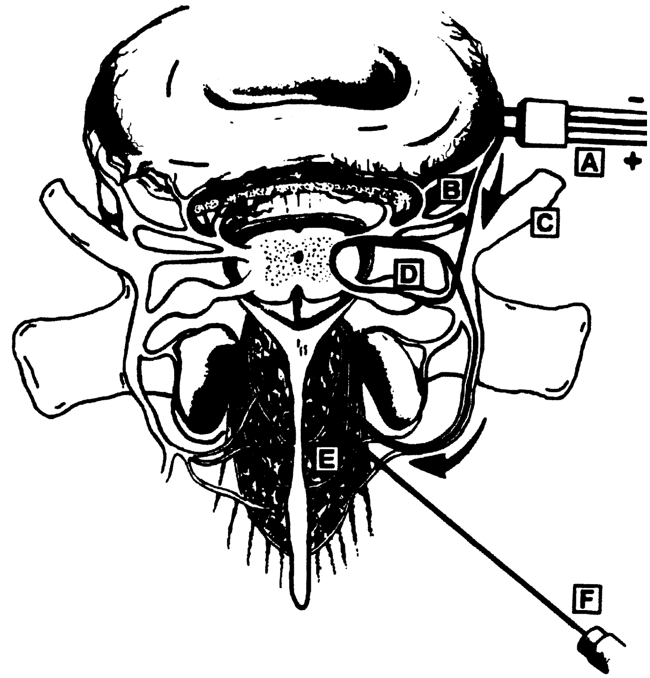

Fig. 1. Schematic representation of the possible innervation between the intervertebral disc and the multifidus muscle: (A) stimulating electrode; (B) nerves in the annulus fibrosus; (C) nerve root (anterior primary rami); (D) dorsal root ganglion; (E) multifidus muscle; (F) electromygraphic needle electrode.

3.1.2. Stimulation of the sacroiliac joint

Via a lateral retroperitoneal approach, hypodermic needles were used for inserting bipolar stimulating wire electrodes into the ventral side of the sacroiliac joint, and also directly under the surface of the joint capsular membrane. This procedure was performed bilaterally, thus establishing two stimulating sites in the sacroiliac joints. The experimental protocol consisted of stimulating separately at each of the four stimulating sites with a set of six stimulations and simultaneously recording the muscular response from six EMG electrodes, which were introduced in the multifidus, gluteus maximus and the quadratus lumborum musculature [28]. For each of the animals, both the left and the right lateral muscle responses were independently investigated.

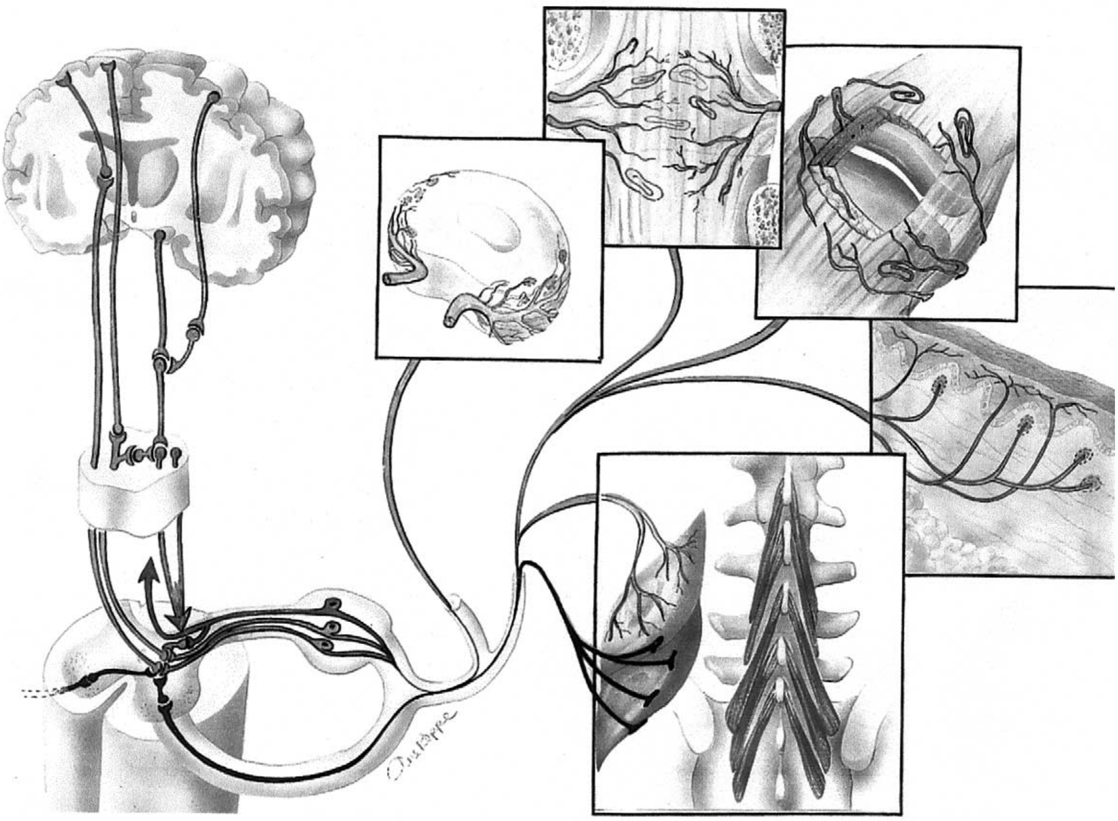

Fig. 2. Schematic hypothesis for a reflex system for motion segment stabilization: (A) proprioceptive and pain signals; (B) intervertebral disc containing low threshold nerve endings; (C) zygapophysial joint capsules containing mechanoreceptors; saline injection into the synovial cavity; (D) network of the paraspinal musculature.

3.1.3. Electromyographic measurements

In order to measure the EMG responses, needle electrodes were inserted into the deepest and most medial multifidus fibers, bilateral to the L2, L3 and L4 spinous processes. Another set of EMG electrodes was inserted into the central region of the longissimus musculature, bilateral to the L4 spinous process. The needle electrodes measured single motor unit action potentials (MUAPs), and were bipolar and concentric with a platinum wire core recording surface [27]. A ground surface electrode was placed centrally on the lower left buttock region. A electrical stimulator (Grass S9 Stimulator, Quincy, MA, USA) was used to deliver a single square pulse to the annulus fibrosus via the stimulating wire electrodes. The conditioned EMG response for all electrodes was recorded using a computerized data acquisition system, with a sampling frequency of 5 kHz for a duration of 25.6 ms, beginning 2 ms before the stimulation. The MUAP was considered a positive response if an identical response occurred at least 5 out of the 6 stimulations within a set and were detected approximately 3–5 ms after the stimulation. For analyzing the data, the peak-to-peak amplitude was measured for each of the positive MUAPs. From the EMG electrodes, the MUAP which measured the largest peak-to-peak amplitude under the control case was used for normalizing the peak-to-peak amplitudes from all six electrodes.

3.2. Neuromuscular responses to the stimulation

Stimulation of the disc annulus fibrosus induced responses in the multifidus on multiple levels and on the contralateral side (Figs. 3 and 4), whereas stimulation of the zygapophysial joint capsule induced reactions pre-

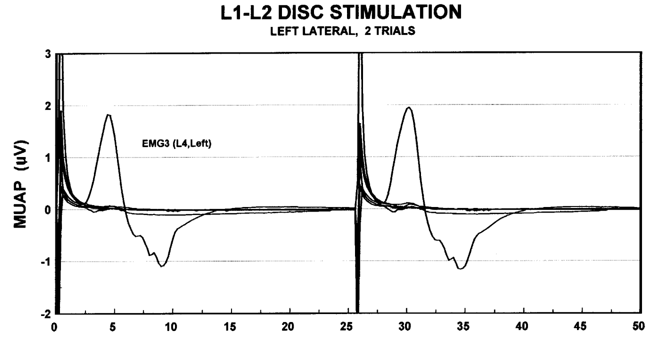

Fig. 3. Examples of the electromyographic responses for all six needle electrodes when stimulating the lateral periphery of the L1–L2 disc.

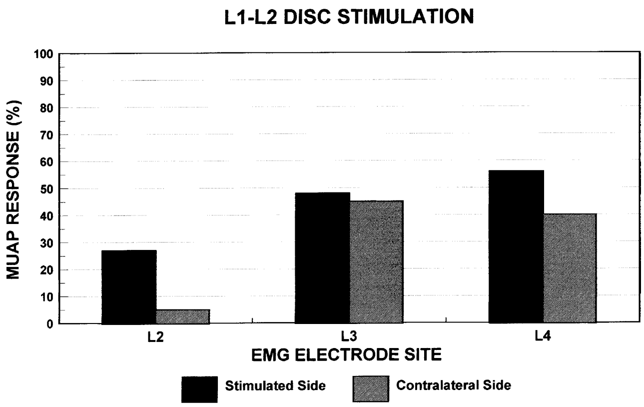

Fig. 4. Overall MUAP response at each of the three lumbar levels for the stimulated and contralateral sides when stimulating the at the disc periphery.

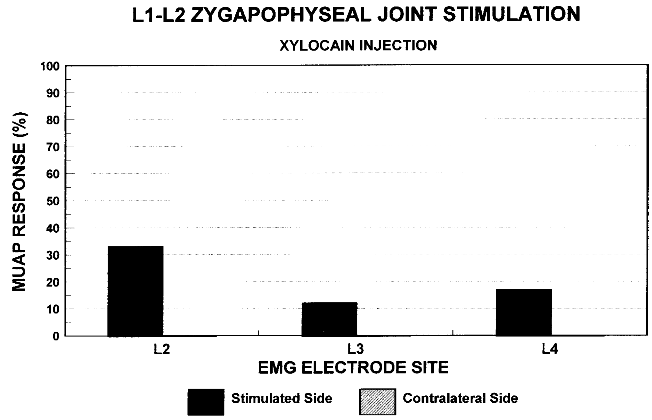

Fig. 6. Overall motor unit action potential response at each of the three lumbar levels for the stimulated and contralateral sides when stimulating the at the zygapophysial joint capsule, after injection of 0.6 ml of lidocaine into the same joint.

dominantly on the same side and segmental level as the stimulation (Fig. 5). Introduction of lidocaine into the zygapophysial joint resulted in a significantly reduced electromyographic response to either stimulation, with the most drastic reduction seen when stimulating the zygapophysial joint capsule (Fig. 6). Subperiosteal detachment of the paraspinal muscles prevented any muscular response. The clinical implications are that there may be interactive responses between injured or diseased structures, i.e. disc or zygapophysial joints and the paraspinal musculature. Reflex activation of the multifidus musculature may have a stabilizing effect for constraining the motion of the lumbar spine.

The mechanically-induced stretch reflex of the zygapophysial joint capsule was verified via introduction of physiological saline into the joint, which resulted in a reduction in the motor unit action potential amplitude. This reduction was manifested as an immediate and constant reduction, a graded reduction, or a delayed reaction, during which the reduction occurred an average of 5 min after the physiological saline injection (Figs. 7, 8 and 9).

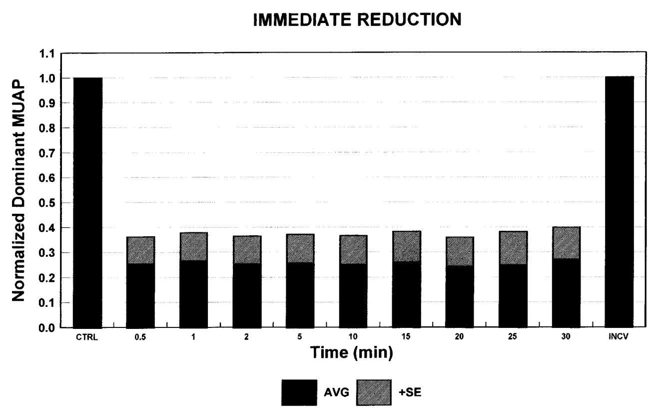

Fig. 7. Normalized dominant motor unit action potentials, experimental group (n=6), which showed a constant reduction within 30 s after saline injection into the zygaphophyseal joint (CTRL=initial response before injection; INCV=response obtained after 30 min, when stimulation voltage was increased 20% from CTRL stimulation voltage).

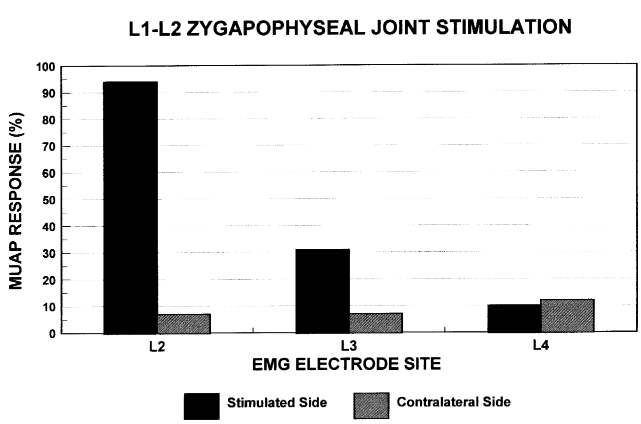

Fig. 5. Overall MUAP response at each of the three lumbar levels for the stimulated and contralateral sides when stimulating the at the zygapophysial joint capsule.

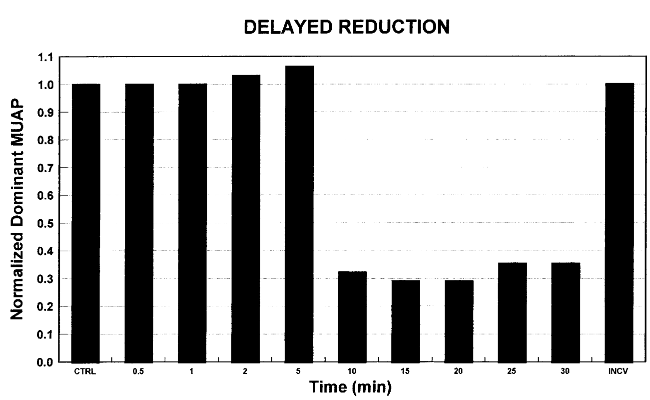

Fig. 8. Normalized dominant motor unit action potentials, experimental group (n=6), which displayed a delayed reduction 5 min after saline injection into the zygaphophyseal joint (CTRL=initial response before injection; INCV=response obtained after 30 min, when stimulation voltage was increased 60% from CTRL stimulation voltage).

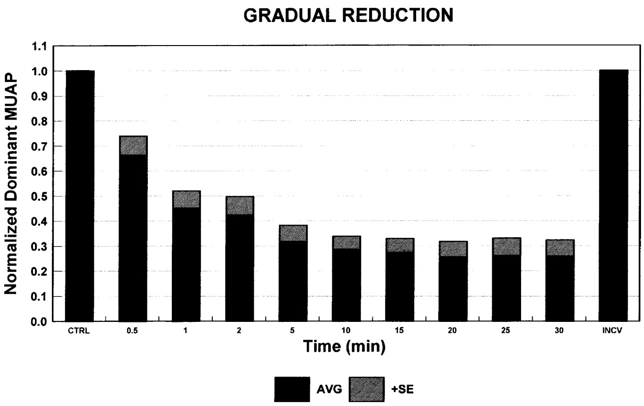

Fig. 9. Normalized dominant motor unit action potentials, experimental group (n=6), which showed a gradual reduction over 30 min after saline injection into the zygaphophyseal joint (CTRL=initial response before injection; INCV=response obtained after 30 min, when stimulation voltage was increased 20% from CTRL stimulation voltage).

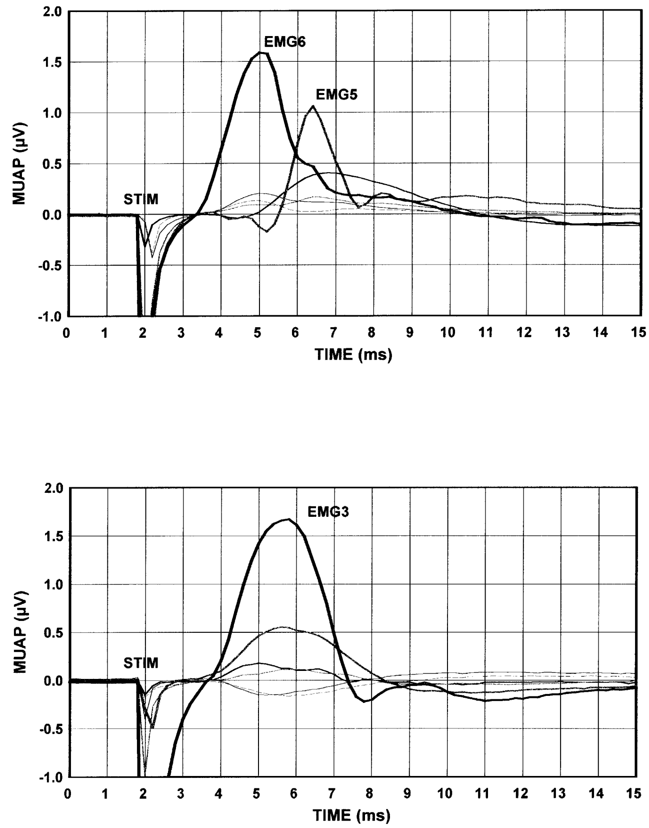

Fig. 10. Examples of motor unit action potentials (MUAPs) measured from all six needle electrodes when stimulating (top) within the sacroiliac joint, and (bottom) on the sacroiliac joint capsule. Muscle sites: EMG3=multifidus (L5 level), EMG5=gluteus maximus, EMG6=quadratus lumborum; STIM=stimulation at 2 ms.

These results indicate that the zygapophysial joint has a regulatory function, controlling the intricate neuromuscular balance in the lumbar motion segment.

On stimulation within the ventral area of the sacroiliac joint, predominant responses occurred in both the gluteus maximus and quadratus lumborum muscles (Fig. 10). When stimulating the capsule however, the greatest muscular responses were detected in the multifidus musculature (Fig. 11). These results indicate a regulatory function for the sacroiliac joint, namely its involvement in activation of the spinal and gluteal muscles, which help control locomotion and body posture, as well as provide stability on the segmental level in the lumbar spine. It is recognized that normal locomotion and posture require multiple levels of neural control. Descending signals from the brainstem activate complex reflex systems in the spinal cord, where the myotactic units with their receptors and polysynaptic circuits are the building blocks. Afferent information is essential in the modification of muscle activation to make it well coordinated and functional. Mechanoreceptors responses to normal loading and movements probably have a primary effect on modulation and modification of descending signals. Injury, certain mechanical loading patterns, degenerative processes and/or inflammation may cause perturbations in the proprioceptive function of different receptors and result in increased or prolonged muscle activation by triggering reflex activation of the involved muscle groups, which over time can cause pain.

4. Reflexes from spinal ligaments

Reflexes from ligaments in many of the joints of the extremities were established in the last 20 years [33,47,52,62]. In the spine, several ligaments are associated with each motion segment eluding to a complex proprioceptive measurement system especially when combined with the sensory inputs from nearby discs and capsules. The existence of sensory receptors in the various spinal ligaments was established [10,24,29,48,53,54,61] . In order to determine if a ligamento-muscular reflex exists from the spinal ligaments to related muscles, the following protocol was used. Six adult cats were anesthetized with a single intraperitoneal injection of Chloralose (60 mg/kg). The skin overlaying the spine was dissected along the midline from the T12 level to the pelvic level. The skin was reflected laterally, and any connective tissue cleaned to expose fully the intact thoracolumbar fascia. Six bipolar of intramuscular fine wire electrodes were inserted in the multifidus muscles, at the levels of L3, L4 and L5, bilaterally. The points of insertion were 1 cm laterally from the spinous process of each level. Each pair of wires was spaced 3–4 mm apart and formed the input to a differential electromyographic amplifier. A ground electrode was placed elsewhere in the preparation. The EMG response from each channel was monitored on the oscilloscope and also recorded and stored on a computer at a sampling rate of 5000 Hz.

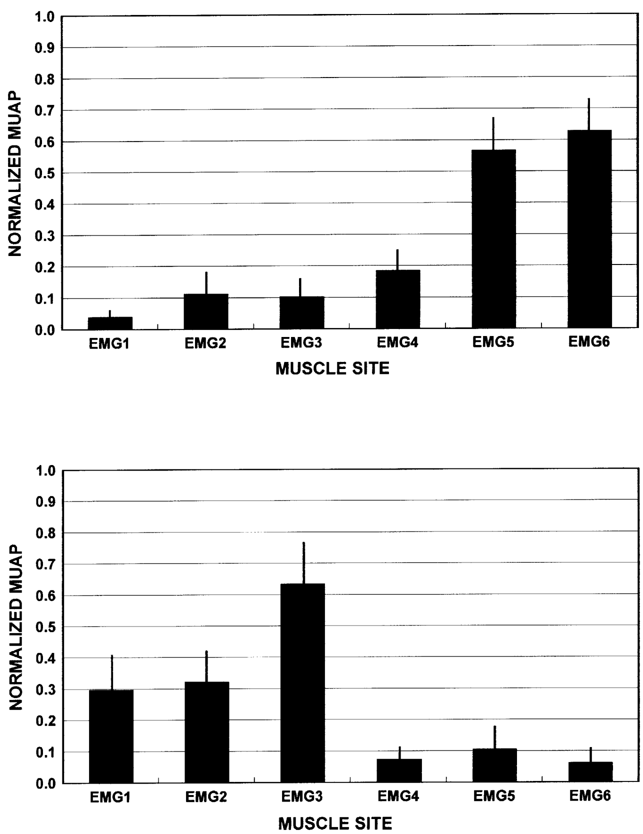

Fig. 11. The mean and standard error of the normalized muscle responses at each of the muscle sites found when stimulating (top) within the sacroiliac joint, and (bottom) on the sacroiliac joint capsule. Muscle sites: EMG1=multifidus (L3 level), EMG2=multifidus (L4 level), EMG3=multifidus (L5 level), EMG4=gluteus medius, EMG5=-gluteus maximus, EMG6=quadratus lumborum; STIM=stimulation at 2 ms.

A bipolar stimulation electrode probe was used to stimulate the supraspinous ligament. The electrode was made of two 1.0 mm diameter solid stainless steel wires, spaced 2–3 mm apart. The stimulating probe was applied to the supraspinous ligament at various locations along the axis from one spinous process to the next, as well as to areas lateral to the process-to-process axis. The stimulus consisted of a rectangular pulse of 100 s duration at a rate of 10 pulses per second. Two seconds periods of stimulation were applied in each trial. The supraspinous ligament was probed with the stimulating electrode from the L1 to the L7 level. A stimulus intensity range of 0–12 mA was available. Stimulus intensity was calibrated so that, when placed over the ligament, a full EMG wave resulted with no further increase in amplitude or wave shape upon further increase of the stimulus intensity. Two sets of 2s EMG recordings were made while stimulating at each level. The time delay between the stimulus pulse application and the resulting EMG was calculated.

Exploration of excitatory locations along the supraspinous ligament between consecutive vertebrae resulted in narrow active segments, 3–4 mm long and 1 mm wide, between each of any two spinous processes tested. The EMG response was present only when applying the stimulus in that narrow segment and directly over the supraspinous ligament. Applications of the probe closer to the spinal processes or slightly laterally from the midline did not result in any visible contraction of the muscles or in any EMG discharge. Fig. 12 shows the exact active zones along the supraspinous ligament. Stimulus intensities necessary to elicit full response ranged between 3 and 7 mA.

A typical EMG discharge in each of the six channels as a result of stimulation of the supraspinal ligament at the L3 level is shown in Fig. 13. Fig. 14 (left) showed the mean (±SD) of the compounded peak-to-peak amplitude of the EMG recorded from L3, L4 and L5 as a response to stimulation of the active zones of the ligament in the L1 to L5 levels of the six specimens tested in this study. The largest EMG (peak-to-peak) were recorded bilaterally from one level caudal to the level of the stimulus application, whereas lower amplitude EMG were recorded from as far as two vertebrae above and below the level of stimulation, bilaterally. This response pattern was consistent and was similar in all preparations. Stimulation of the supraspinous ligament in the L6 segment resulted in a somewhat lower response in the muscles, whereas stimulation of the L7 segment did not result in any EMG response or visible twitch in the muscles.

Fig. 15 shows a typical single EMG response recorded upon stimulation of the supraspinal ligament at the L3 level. In this figure the stimulus pulse is superimposed

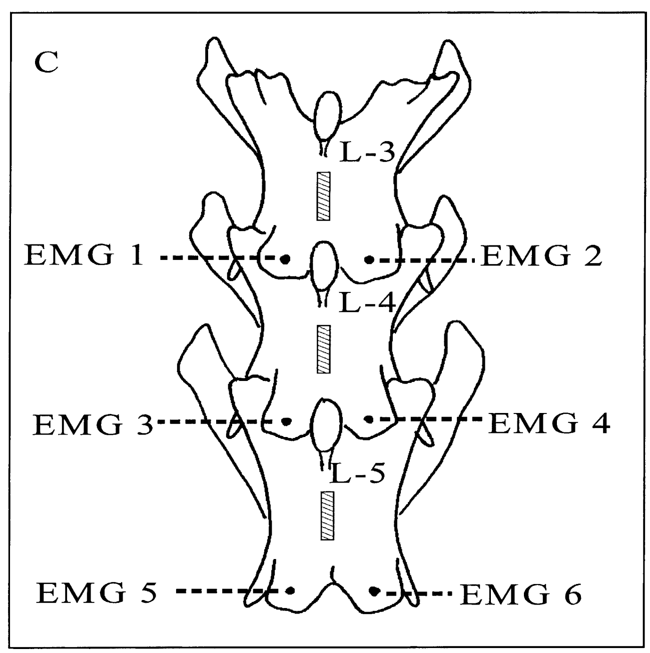

Fig. 12. The active zone of the supraspinous ligament which is sensitive to bipolar electrical stimulation.

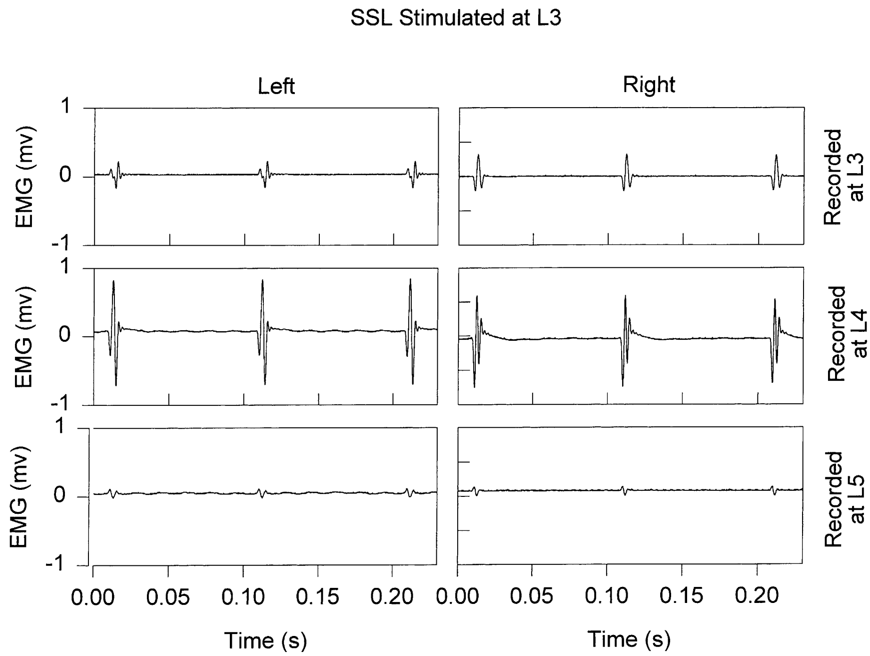

Fig. 13. Typical EMG recordings in three lumbar levels as response to electrical stimulation of L-3 supraspinous ligament.

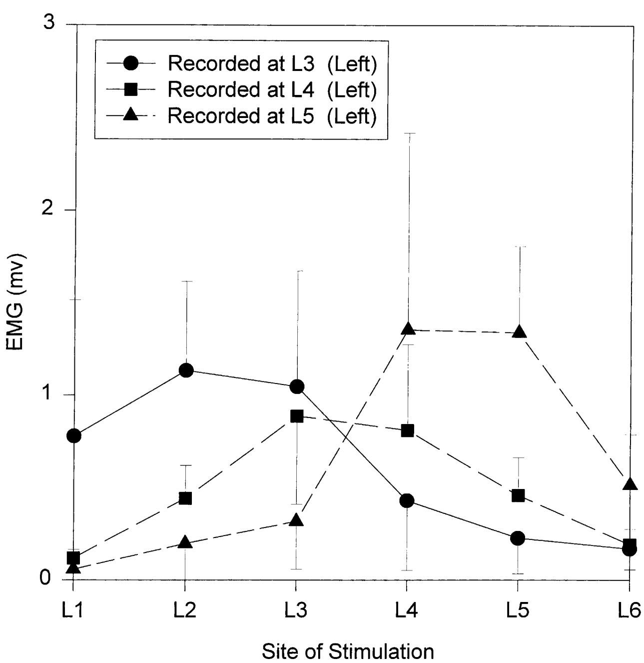

Fig. 14. The mean (±SD) of the peak-to-peak EMG compounded from all the preparation as a function of the level of stimulation of the supraspinous ligament.

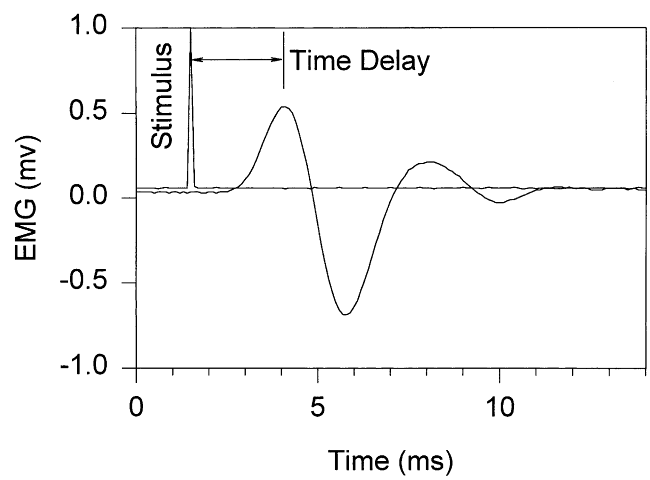

Fig. 15. The latency of the EMG response as measured from the peak of the stimulus artifact to the peak of the evoked action potential.

on the EMG response so that the time delay from the instant of stimulus application and the first peak of the EMG response could be calculated. The range of the mean time delay in any of the EMG waves from any muscle at any stimulation level was from 2.52 to 2.77 ms, and there was no observable pattern of increasing or deceasing delay in recordings from muscles two levels away from the stimulation site.

4.1. Responses to mechanical stimulation

In order to assess the responses of the supraspinous ligament to mechanical deformation, mostly elongation, the following protocol described for the feline preparation in the previous section was modified as follows. The preparation then was placed in a rigid stainless steel frame that allowed the immobilization of various vertebrae as desired. A monofilament suture was inserted around the middle of the supraspinous ligament of each lumbar motion segment and connected (one at a time) to a Bionix 858 Material Testing System (MTS, St. Paul, MN) instrumented with a computer-controlled loading system. The load transducer output was sampled into the computer together with the EMG signals.

The preparation first was placed prone in the frame, and the suture-load transducer were pulled up by the Bionix 858 System in a linearly increasing vertical displacement for 6 seconds, followed by a steady state for 2 s. Three-minute rest periods were implemented between trials to avoid the effect of fatigue, muscle potentiation, reflex habituation, and permanent ligament deformation. Although single recordings were made from loading each ligament, occasionally a second or third recording was made. The second phase of the experiment consisted of external fixation of two adjacent spinal processes with stainless steel rods firmly attached to the experimental frame, such that no movement of the vertebrae was possible. The same mode of ligament deformation used in the first phase then was repeated to assess the response to isolated deformation of the supraspinous ligament. In this condition, no movement or loading of other spinal ligaments, facet joints, discs, or muscles was possible, limiting motion to the deformation of the supraspinous ligament. The maximal load applied to each ligament gradually was increased from trial to trial by 5–10 N, until the ligament ruptured.

Fig. 16 (left) shows a typical EMG response from each of the six multifidus muscles in the L1–L2 to L6–L7 motion segments to linearly increasing force applied to the supraspinous ligament at L4–L5, with the vertebrae not immobilized. Conversely, Fig. 16 (right) shows the EMG response from the different levels of the same preparation with the L4 and L5 vertebrae immobilized. Several important observations could be made from the two figures. In the case where the vertebrae were not immobilized, application of force to the ligament in the range of 0 to 25 N resulted in strong EMG activity at L4–L5 with simultaneous activity in the three levels above (L1–L2, L2–L3, and L3–L4) and one level below (L5–L6). The EMG activity first was initiated in the muscles adjacent to the L4–L5 motion segment at a load of 10 N, and, as the force further increased, the activity in the three superior levels was initiated. The inferior level produced EMG activity at the same load threshold that caused EMG activity at L4–L5.

The EMG response in the immobilized case was evident only when the applied deformation force to the ligament exceeded 23 N, more than twice as much as in the free-vertebrae condition. The EMG activity was completely absent in the L1–L2 and L2–L3 segments and present in the L3–L4 and L5–L6 motion segments, one level superior and inferior to the level of ligament deformation. The EMG amplitudes in the level of load application and one level above and below were also significantly lower than the amplitude seen in the free state.

Fig. 17 (left) shows the EMG response of the lumbar levels that had undergone the same procedure shown in Fig. 16, but after the maximal load to the L4–L5 ligament was increased from 35 to 40 N, while the L4–L5 segment was still immobilized. Electromyographic activity is seen in the same three levels of Fig. 16, i.e. L3–L4, L4–L5, and L5–L6, with the largest EMG discharge still present at the level of ligament deformation (L4–L5). The major difference in the response of this trial compared with that of Fig. 16 is that the EMG response is much more intense in amplitude and frequency content, reflecting the higher stress in the ligament. The load threshold at which the EMG increase first was seen in the multifidus muscle at L4–L5 was 18 N, 5 N lower than in the previous trial, indicating that the rate of loading also may be a factor in the reflex activation of the multifidus muscles. The EMG discharge one level above (L3–L4) that of the deformed ligament still shows minor discharge, whereas the EMG discharge at the level below (L5–L6) shows increased intensity and frequency content of the discharge.

Fig. 17 (right) shows the EMG response of the multifidus muscles that had undergone the same procedure with an immobilized spine, while the maximal load applied to the ligament at L4–L5 was increased from 40 to 50 N. As is clearly visible from the load trace, the ligament ruptured as 45 N load was reached; in this trial, however, the EMG discharge of the level superior to the

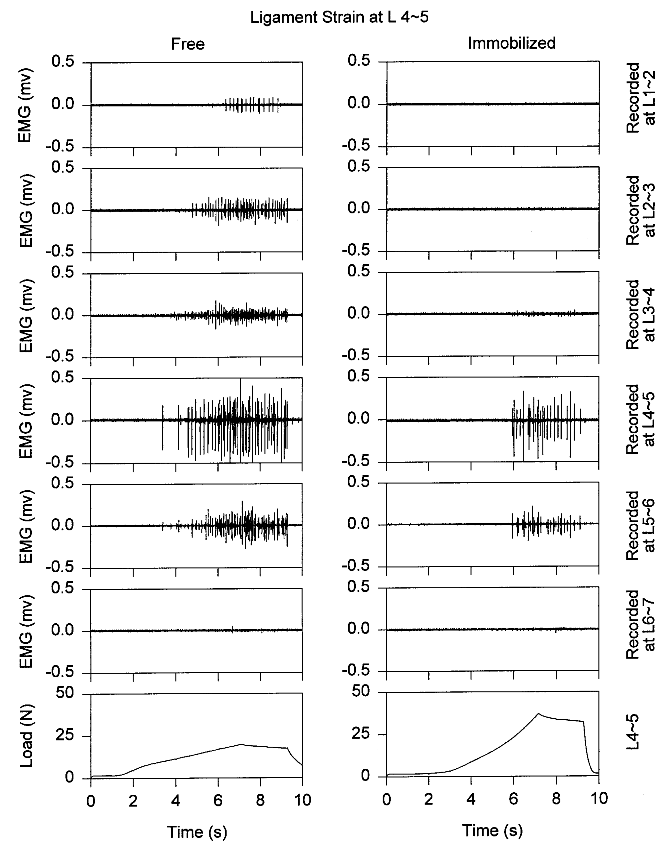

Fig. 16. Typical unilateral multifidus muscles EMG in response to linearly increasing force in the supraspinal ligament of L-4/5, with the lumbar spine free to flex in the left column, and with the L-4 and L-5 vertebrae immobilized in the right column. With immobilized vertebrae the contribution to the reflex from discs and other ligaments is eliminated.

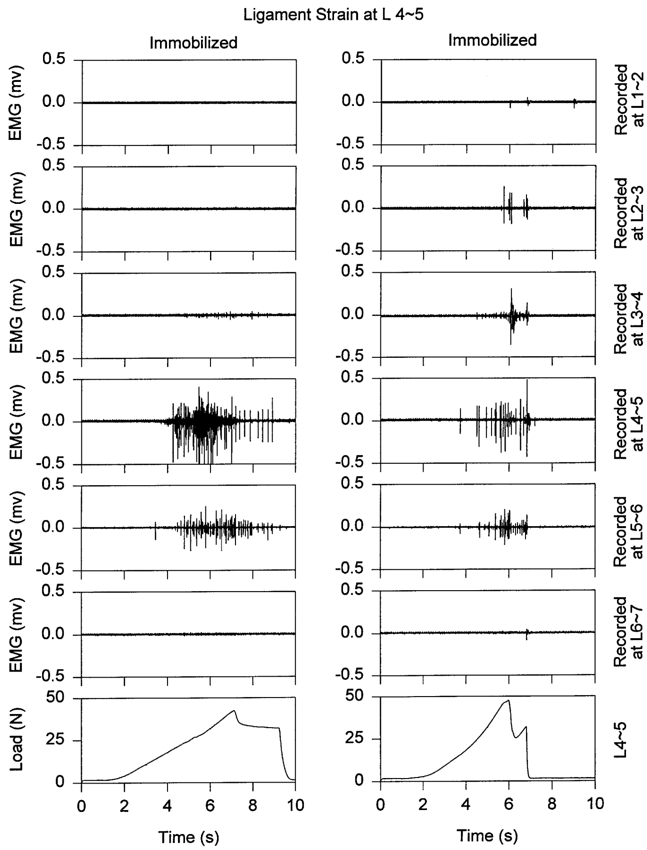

Fig. 17. The effect of increasing the peak force applied to the supraspinous ligament of L-4/5 with the adjacent vertebrae immobilized is shown as appearance of EMG response in levels which were previously silent.

ligament deformation was significantly more pronounced than that in the two previous trials. Furthermore, EMG activity also was registered at L2–L3, whereas there had been no such activity at that level in the two previous trials.

Several important features are disclosed by the compounded data presented in Fig. 18. An EMG discharge generally appeared first in the multifidus muscles at the level at which the ligament was loaded and was triggered at the lowest load threshold, which increased for rostral and caudal levels. Exceptions to this trend exist when loading the ligaments at L3–L4. Although the load threshold at which EMG activity first was seen was lowest in the L3–L4 ligament, EMG activity in this level was absent in two of the 18 trials available from this level. Simultaneously, EMG activity was present at L1–L2 and L2–L3 in 18 of the 18 trials in which the L3–L4 ligament was loaded. Similarly, when the L4–L5 ligament was loaded, EMG activity was present in the multifidus muscle at L3–L4 every time, whereas it was absent in two of the 18 trials available from the L4–L5 multifidus muscle. The mean load threshold at which EMG activity first appeared in the level at which a given ligament was loaded ranged from 4.98 to 9.87 N. The average weight of the feline specimen used in this study was 4.3 kg, or 43 N. The load required to elicit muscular response, therefore, ranged from 11.58% of body weight to 22.9%. The largest load required to elicit EMG activity was recorded at L4–L5 (22.9% body wt) and was lower for rostral and caudal levels. Passively induced anterior flexion of the spine by lifting a padded bar placed under the abdomen of the preparation at L3–L4 did not result in any EMG activity in any of the six channels throughout the L1–L2 to L6–L7 levels, indicating that stretch receptors in the muscles did not participate in this reflex.

5. Clinical implications of reflexes from the passive structures in the spine

The obvious function of spinal ligaments is stabilization and support (Fig. 19). The increased focus on the innervation of different spinal structures has led to a new understanding and awareness that they may play an important role in a complex regulating system [11,14,21,34,63]. Compared to other joints, the spinal motion segment is more complex, consisting of an intervertebral disc and two zygapophysial joints. This gives the segment more stability and firmness needed for protection of the spinal cord, spinal nerves and nerve roots [1]. The complex network of paraspinal muscles suggests an intricate control of loading and motion on the different segments (Fig. 20) [9].

Normal locomotion requires multiple levels of neural control. To support the body against gravity, maintain posture and to propel it forward, the nervous system must coordinate muscle contractions at many joints (Fig. 21). At the same time, the nervous system must exert active control to maintain balance of the moving body, and it must adapt the locomotion pattern to the environment and to the overall behavioral goals. The spinal circuits activated by descending signals from higher centers accomplish this. Neural circuits in the spinal cord play an essential role in motor coordination. Spinal reflexes, where the 'myotactic units' are the building blocks, provide the nervous system with a set of elementary patterns of coordination that can be activated, either by sensory stimuli or by descending signals from the brain stem and cerebral cortex.

In muscle and tendon, the motor and sensory functions of the neural structures for controlling posture and movements are well established [20]. The load-sensitive nerve endings, or mechanoreceptors, found in muscle (muscle spindles) and tendon (Golgi tendon organs), provide proprioceptive information regarding tension levels, essential for controlling muscle tone, and thereby joint stability (Fig. 22). The neurological feedback from passive viscoelastic structures provides sensory information needed to regulate muscle tension, and hence, the stability in the lumbar spine.

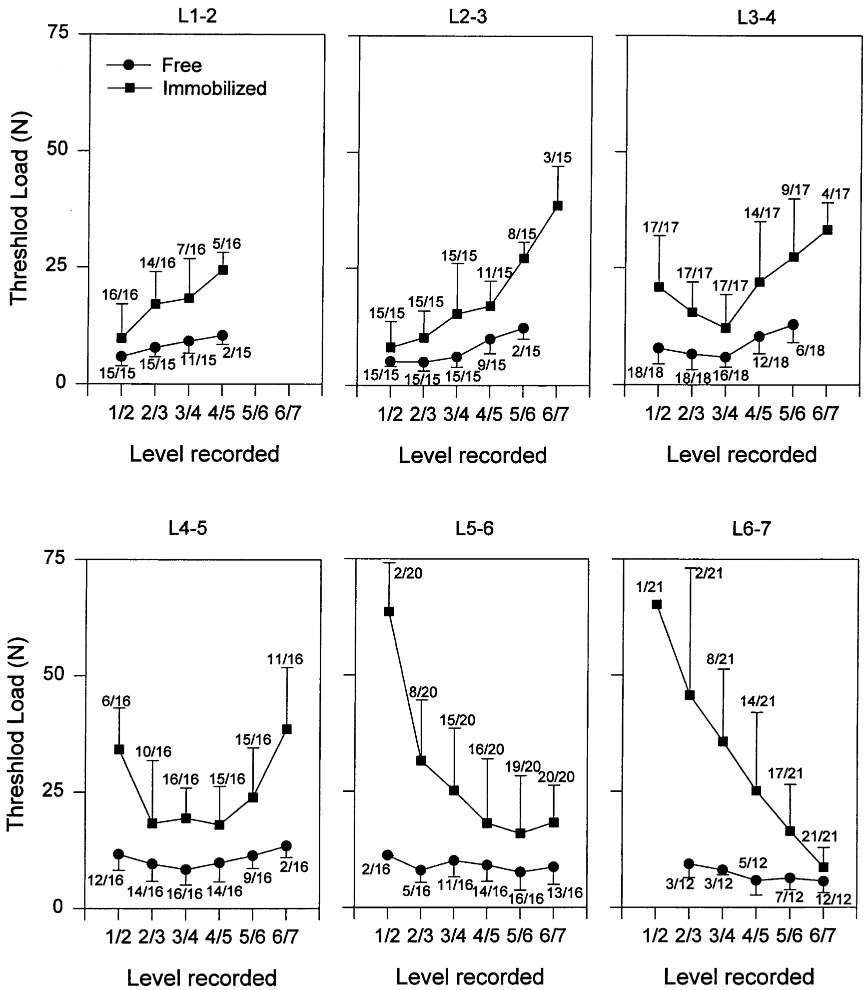

Fig. 18. Graphical representation of data in Tables 1 and 2 showing the radiation of EMG responses up and down the lumbar spine due to mechanical loading at a given lumbar level.

The functioning of the motor system is intimately related to that of the sensory system. The proper moment-to-moment functioning of the motor system depends on a continuous inflow of sensory information (Fig. 23). Sensory information influences motor output in many ways and at all levels of the motor system. Sensory input to the spinal cord directly triggers reflex responses. It is also essential for determining the parameters of programmed voluntary responses. Finally, sensory input, especially proprioceptive information, is integral to both feedback and feed-forward mechanisms, which provide flexibility in the control of motor output. For instant, both feed-forward control and rapid feedback compensatory corrections maintain postural stability during standing and walking. In lifting or pulling, postural muscles contract to maintain equilibrium before destabilizing movements are executed.

The nerve endings in the outer annulus fibrosus of the disc, in the capsule of the zygapophysial joints, and in the ligaments are most likely part of a proprioceptive system responsible for optimal recruitment of the paraspinal muscles [42,49] . Mechanoreceptors are thought to play an important role in the function of monitoring position and movements of joints by regulating and modifying muscle tension. These different nerve endings can record the loading on the different structures of the spine. The descending signals that initiate muscle action are modified by the sensory input from the proprioceptive nerve endings. Recruitment of the paraspinal muscles may thus be coordinated in such a manner that the forces applied to the various structures are properly distributed and in such a pattern that the loading on the motion segment is optimal regardless of position. In such a system, the action of these muscles can provide the different structures with the support that is needed in order to counteract detrimental forces and prevent injury. Overload on specific parts can, by high threshold nerve endings, be detected and in due process, inhibit muscle actions responsible for the increased loading, and thereby prevent injury. This may be the reason why heavy physical loading does not seem to have the impact on degeneration of the spine as earlier assumed [4,45].

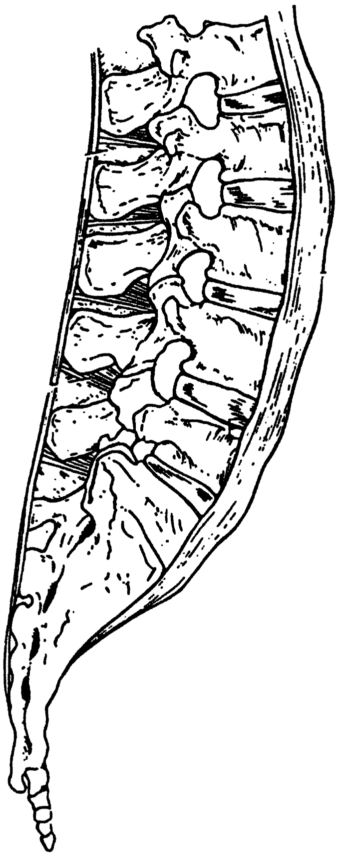

Fig. 19. Schematic drawing of the spine showing the five lumbar motion segments.

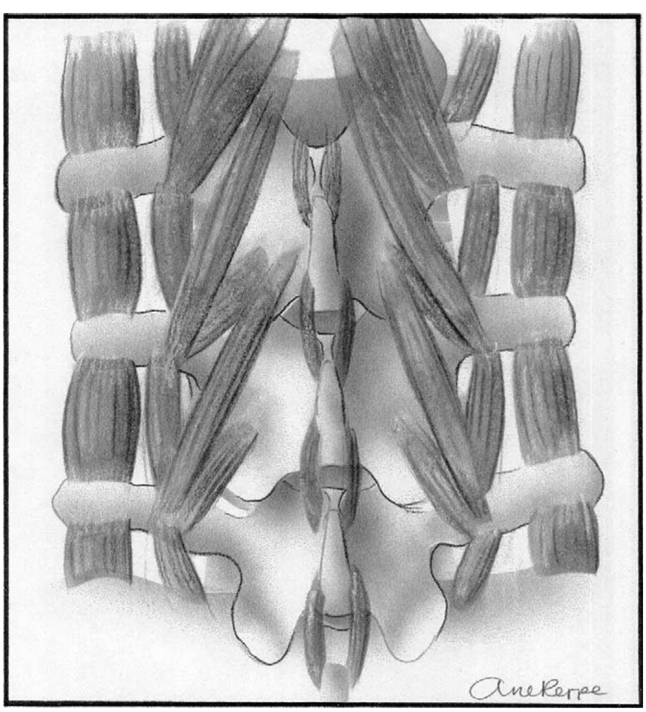

Fig. 20. Arrangement of the interspinalis, intertransverarii mediales and laterales and parts of the multifidus muscles

Possible muscle activation as a result of damage to passive viscoelastic spinal structures is difficult to detect. Painful stimuli seem to have an inhibitory effect on muscle activation. But damage done to ligaments and perhaps other passive structures does not necessary have to result in a lot of pain. Depending on the size of the lesion, the density of the neural structures and damage done to them, and the degree of irritation on the surrounding nerve endings, the firing pattern from these nerve endings may be altered in such a manner so as to cause increased activation of the paraspinal muscles. Studies have shown that experimental pain in muscles does not increase the firing of α-motor units, but it does increase the stretch reflex [43]. Increase in such reflexes may result in inappropriate muscle activation.

The sacroiliac joint is a true synovial joint with an articular shape and a very limited amount of motion (Fig. 24). The joint is relatively small considering the forces transmitted across it. The sacroiliac joint does, however, have an extensive network of strong ligaments that maintain stability and are constructed in such a way that they are self-tightening with increasing load. Roentgen stereophotogrammetric analysis has shown the amount of joint motion to range from 0.5–1.6 mm for translation and up to 4 degrees for rotation [55].

Stimulation of the outer annulus of the disc or zygapophysial joint, both of which have been shown to contain nerve endings, causes activation of paraspinal musculature, not only on the same segmental level, but also on different levels, indicating a complex interaction [27]. Such an interaction is necessary in order to stabilize different segments, not only in relation to each other, but also in the process of maintaining posture. On the other hand, a lesion in one location may cause alterations in muscle activation in other than the actual segment and also on the contralateral side. Avramov et al. [3] have shown that loading excites three patterns of nerve discharges from the zygapophysial joints; short duration bursts during change in loading, prolonged discharges at low levels and prolonged discharges at high load levels. These results indicate that different units in the joint capsule have different levels of stress threshold. The range of motion and innervation of the sacroiliac joint seems well suited for detecting various loading patterns during locomotion. In man, the slanted position of the L5–S1 motion segment and the relative position of the sacroiliac joint appear to have physiological importance for load detection. The afferent input from sacroiliac joint receptors, as well as mechanoreceptors in the intervertebral disc and zygapophysial joints, will contribute to different degrees of muscle activation and may constitute an integral regulatory system [28]. Changes in loading on the sacroiliac joints may result in altered activation of the stabilizing muscles, and thus play an important regulatory function in stabilization and movement of the upper body during postural changes.

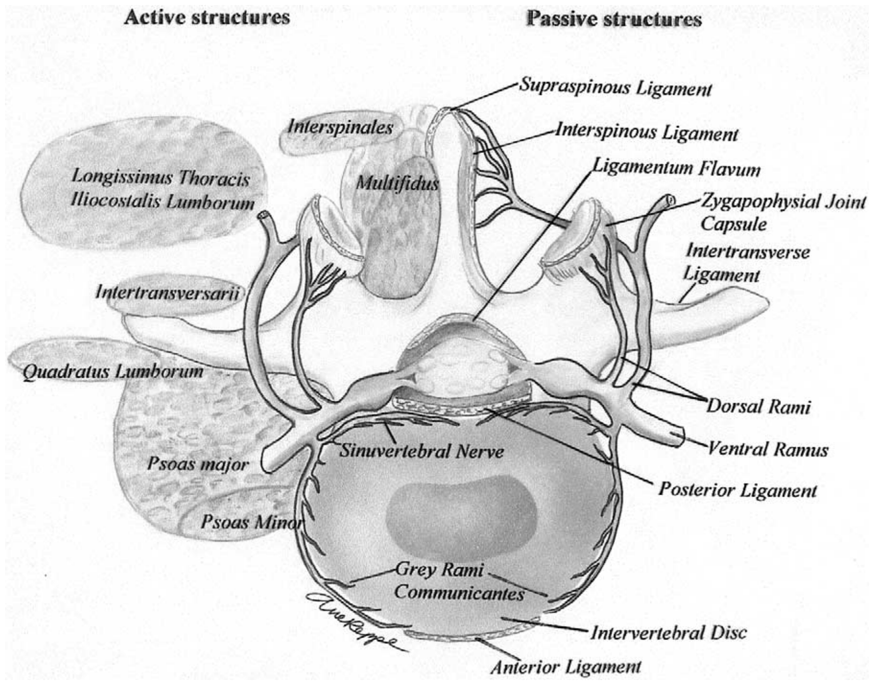

Fig. 21. Schematic drawing of the bilateral active and passive structural arrangement and sensory innervation on the L3–L4 level.

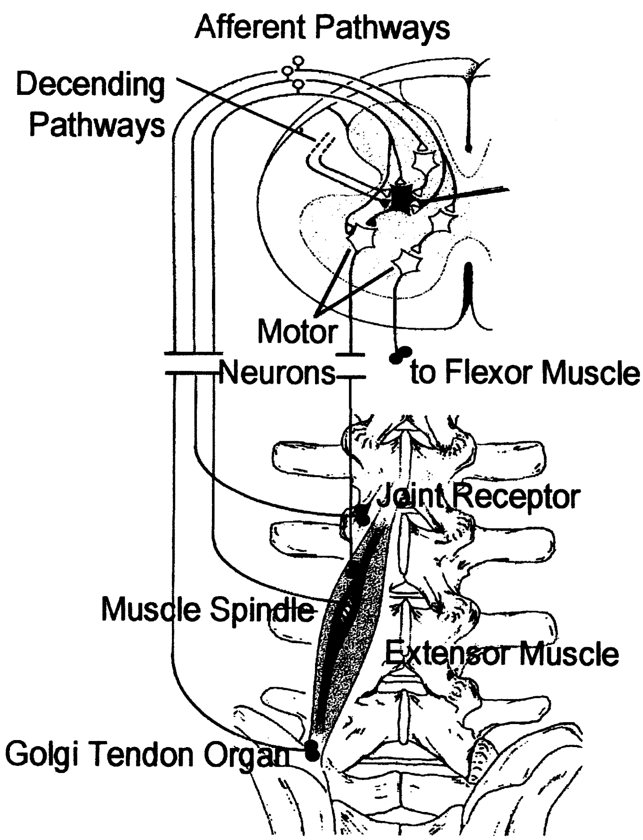

Fig. 22. Neuromuscular feedback system depicting the afferent sensory information from joint receptors, muscles spindles, and Golgi tendon organs for regulating muscle tension.

During pregnancy, many changes take place and with increased gestation time and there is increased loading as well as greater pelvic relaxation [41]. Relaxation of the pelvic joints can change the normal reference range for the mechanoreceptors and result in altered recruitment of stabilizing musculature. The change in walking pattern seen in pregnant women may be a result of such changes and reflect a functional adaptation to adjust for increased loading and result in comfortable walking with minimal use of energy. Failure to adapt a functional muscle strategy to redistribute the increased loading may result in a dysfunction capable of generating pain. The nature of sacroiliac dysfunction may rather be found within this framework than in mere mechanical failure.

Instability of a spinal motion segment, as a result of degeneration of the disc or zygapophysial joints, is believed to be manifested as ‘slipping’ as a result of laxity in the motion segment. Kaigle et al. [32] have shown that this kind of hypermobility does not seem to occur, but the motion pattern is greatly altered. The change in length and loading of the ligaments may rather result in altered firing patterns and changes in the coordination pattern of the muscles. When decreased disc height as a result of degeneration, adaptation of the surrounding nerve endings may be less efficient and thus result in less optimal neuromuscular reflexes. Better knowledge of the sensory function of the passive spinal structures should influence the manner in which these structures are clinically.

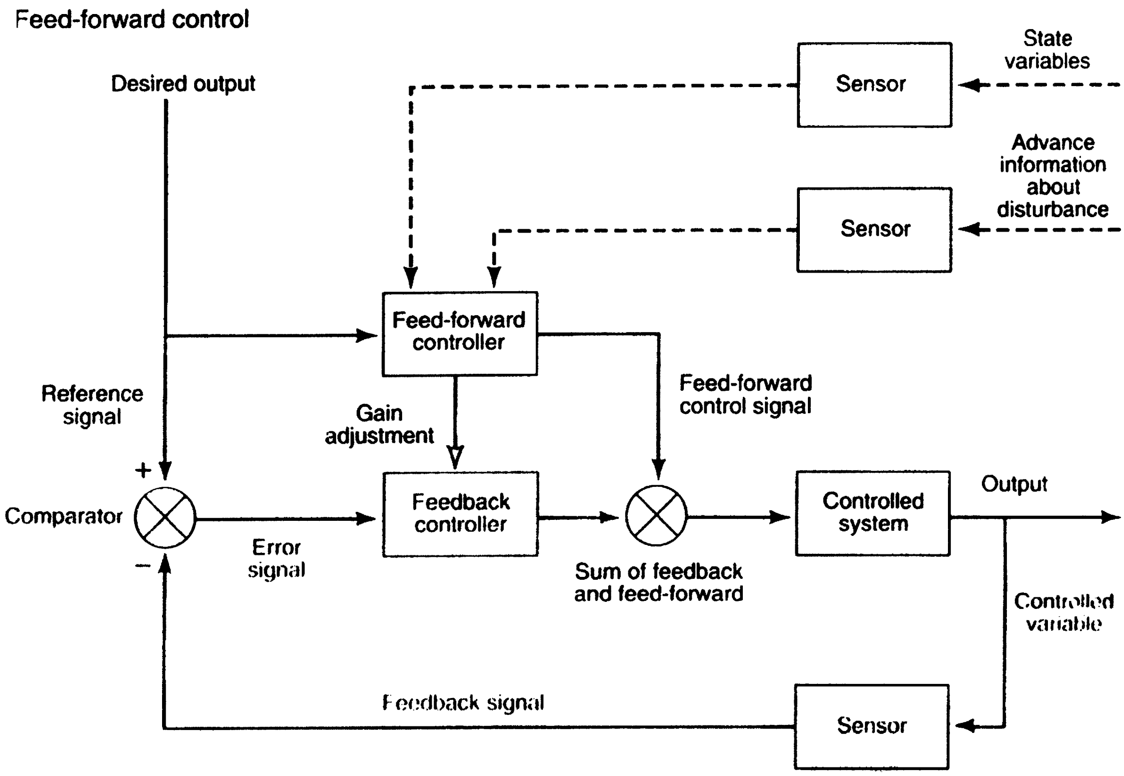

Fig. 23. Flowchart showing the functioning of the motor system involving a feed forward control in relation to feedback compensatory corrections.

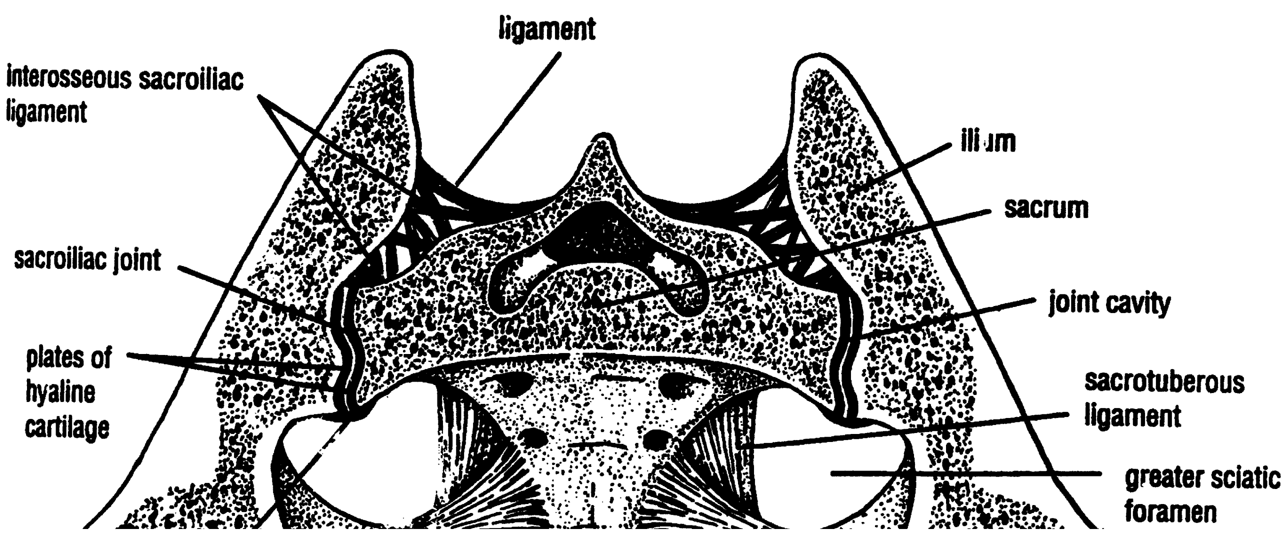

Fig. 24. Representation showing the sacroiliac joint and the stabilizing ligaments.

Acknowledgements

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