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Trunk muscle activation in low-back pain patients, an analysis of the literature

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Abstract

This paper provides an analysis of the literature on trunk muscle recruitment in low-back pain patients. Two models proposed in the literature, the pain–spasm–pain model and the pain adaptation model, yield conflicting predictions on how low- back pain would affect trunk muscle recruitment in various activities. The two models are outlined and evidence for the two from neurophsysiological studies is reviewed. Subsequently, specific predictions with respect to changes in activation of the lumbar extensor musculature are derived from both models. These predictions are compared to the results from 30 clinical studies and three induced pain studies retrieved in a comprehensive literature search. Neither of the two models is unequivocally supported by the literature. These data and further data on timing of muscle activity and load sharing between muscles suggest an alternative model to explain the alterations of trunk muscle recruitment due to low-back pain. It is proposed that motor control changes in patients are functional in that they enhance spinal stability.

Keywords: Low back pain; Electromyography; Diagnosis; Pain adaptation model; Pain spasm pain model; Trunk muscle coordination

1. Introduction

Low-back pain (LBP) is one of the most prevalent and costly health problems in western society [7]. In spite of extensive research efforts, the causes of LBP are still elusive and treatment effects are unsatisfactory. It is often suggested that the occurrence of LBP should be accepted as a fact of life and efforts of researchers and clinicians should focus on preventing LBP from becoming chronic rather than at prevention of first-time occurrence [7].

To design a rational program for secondary prevention, it needs to be established whether behavioral responses displayed by patients should be considered adaptive and supportive of recovery or as adverse and contributing to a vicious cycle leading to chronicity. An important debate in the literature on LBP in this respect focuses on the interpretation of changes in trunk muscle activity in LBP patients. On the one hand, these changes are interpreted in the context of the pain–spasm–pain model, postulating that pain results in increased muscle activity, which in turn will cause pain [110,129]. In contrast, the pain adaptation model postulates that pain reduces activation of muscles when active as agonists and increases activation of muscles when active as antagonists. This will reduce movement velocity and range of motion, which would prevent mechanical provocation of pain in damaged tissues and further damage of these tissues [78].

It is striking that proponents of both models have found evidence supporting their interpretation in reviews of the literature on electromyographically recorded back muscle activation in LBP patients [78,110]. The aim of the present paper therefore is to more systematically review the literature on effects of clinical and experimentally induced LBP on trunk muscle activation in terms of levels of activation, the timing of activation, and load sharing between muscles. First, the two models will be outlined and specific predictions on trunk muscle

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activation will be derived for each model. Second, studies on trunk muscle activation in LBP patients will be reviewed. The results from the studies reviewed will be compared to the model predictions. Finally, an alternative interpretation for the experimental results based on spinal instability as an important component of LBP will be proposed.

2. Pain–spasm–pain model

Pain occurring immediately after acute trauma is often accompanied by intense contractions of muscles surrounding the injured structures. This is thought to be functional, since it will prevent motion of the injured structures. In clinical practice, it is often assumed that similar muscular reactions occur following non-traumatic pain. In these cases, it is not the functional, adaptive nature of such a response that is emphasized but rather its possible adverse consequences. The pain–spasm–pain model was first formally proposed by Travell [129]. Travell suggested that pain would lead to muscular hyperactivity referred to as spasm, which in turn will cause pain. Treatment modalities based on this model involve relaxation and stretching of muscles.

Two distinct neural pathways have been proposed to form the basis of a vicious pain–spasm–pain cycle (Fig. 1). Most tissues in the low back, including, muscle, tendon, ligament, bone, endplate, and annulus fibrosus, are innervated by thin myelinated and unmyelinated fibers with free nerve endings. These afferents, which are considered to be nociceptors, enter the posterior horn, from where they project onto higher centers giving rise to the perception of pain. In addition, these fibers project via

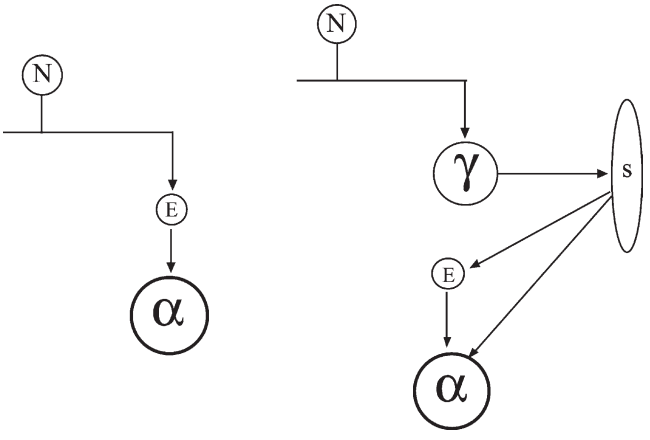

Fig. 1. Neural pathways suggested to form the basis of a pain-spasm-pain cycle. Left side: Feedback of nociceptive afferents (N) via excitatory interneurons (E) on the alpha motorneuron (α) will cause increased muscle activation (spasm). Right side: Feedback of nociceptive afferents (N) on the gamma motorneuron (γ) will increase the afference of the muscle spindles (s), which activates the alpha motorneurons directly and via excitatory interneurons (E), which will cause increased muscle activation.

interneurons onto alpha motorneurons at the segmental level. Wyke [138] has suggested that these projections form the basis of a reflexive hyperactivity of back muscles in response to pain. Johansson and Sojka [59] have proposed an alternative neural pathway to form the basis of a pain–spasm–pain loop. They proposed that nociceptors affect the output of muscle spindles via direct excitatory projections on the gamma motorneurons. The increased muscle spindle output will cause hyperexcitability of the alpha motorneuron pool. The common denominator in the two theories is the hyperexcitability of the alpha motorneuron pool, which could form the basis for more sustained and more intense muscle activity in pain patients as compared to controls. This sustained activity is in turn expected to cause pain due to the accumulation in the muscles of for example arachidonic acid, bradykinin, potassium and lactate. These substances have been shown to have strong excitatory effects on nociceptors in muscle tissue [66,91].

Several basic neurophysiological studies have been performed to verify the existence of the pathways proposed to underlie the pain–spasm–pain cycle. Evidence of hyperexcitability of the alpha motorneuron pool due to nociceptive stimuli was found in cats after mechanical or chemical noxious stimulation of facet joints, fascia, ligaments, periosteum, and muscle. The hyperexcitability was shown in the form of short erratic bursts of paraspinal muscle activity [56,101,118,119,123,137]. In pigs, paraspinal muscle activity was found to increase during electrical stimulation of the intervertebral disc and facet joints [54]. In rat, the flexion reflex was increased following induction of muscle pain [134]. Likewise resting EMG levels in rat masticatory muscles were found to be increased after induction of pain. This effect did however last only up to 10 min. Experimental results in humans are limited. The amplitude of the Hofmann's reflex, which is an indicator of alpha motorneuron excitability, was not increased after injection of hypertonic saline in the calf muscles [88]. However, resting EMG levels were increased in human masticatory muscles [124], although the effect was only short-lived.

Part of the neurophysiological studies inspired by the pain–spasm–pain model focused on the pathway proposed by Johansson and Sojka [59], by looking at the effects of nociceptive afference on muscle spindle activity. An increased output of muscle spindles in cat hind leg and neck muscles, as a consequence of nociceptive stimuli, was found in a range of experiments [30,76,102,103]. However, an experiment in which myositis was induced in the hind leg of the cat showed a decreased activity of gamma motorneurons [92]. Also in cat back muscles no increased gamma motorneuron activity was found after injection of bradykinin and capsaicin [65]. Inflammation of the knee joint produced mixed excitatory and inhibitory effects on gamma motorneurons in cats [42]. In human calf and masticat-

ory muscles, the stretch reflex, which is mediated by muscle spindle afferents, was found to be enhanced after injection of hypertonic saline [88,125]. However, in back muscles no enhancement of stretch reflexes was found after hypertonic saline injection [141]. In addition, during walking the stretch reflex in the calf muscles was unaffected by induced pain [87].

In conclusion, the studies reviewed do not unequivocally support nor clearly reject the mechanisms, which have been proposed to underlie the pain–spasm–pain model. In addition, most of this work involved animal experiments with anesthetized or decerebrate animals, greatly reducing or excluding any influence from the brain. The pain-adaptation model (see below) states that the effects of peripheral projections of nociceptors onto alpha motorneurons are modulated by central nervous influences related to voluntary activity. Furthermore, it has been suggested that higher levels of muscle activation can occur as part of a response to pain as a psychological stressor [57]. Therefore, testing the model with data obtained in studies of induced or clinical pain in awake humans may provide further information.

The basic tenet in the pain–spasm–pain model is that pain will result in more sustained and increased muscle activation. The model predicts that, in every submaximal task as well as in rest, muscle activation will be higher in patients as compared to healthy individuals. By mechanical necessity this implies that cocontraction of agonist and antagonist muscles occurs in the patients. During maximum efforts, patients are predicted to attain the same level of muscle activation as controls, with however a lower moment production due to the antagonistic cocontraction. These predictions (Table 1) will be tested below using data from clinical studies.

3. Pain-adaptation model

The pain-adaptation model was proposed by Lund et al. [78] to account for clinical findings on muscle activity in pain syndromes. The model states that pain decreases the activation of muscles when active as agonists and increases it when the muscle is active as antagonist [78]. The effects of such a control strategy would be that movement velocity is reduced and movement excursions are limited. These kinematic effects are believed to prevent pain provocation.

A neural pathway suggested to account for the recruitment changes in the pain-adaptation model consists of the nociceptors that project on the alpha motorneuron via both inhibitory and excitatory interneurons (Fig. 2). The excitability of these interneurons is controlled by the central nervous system, in such a way that depending

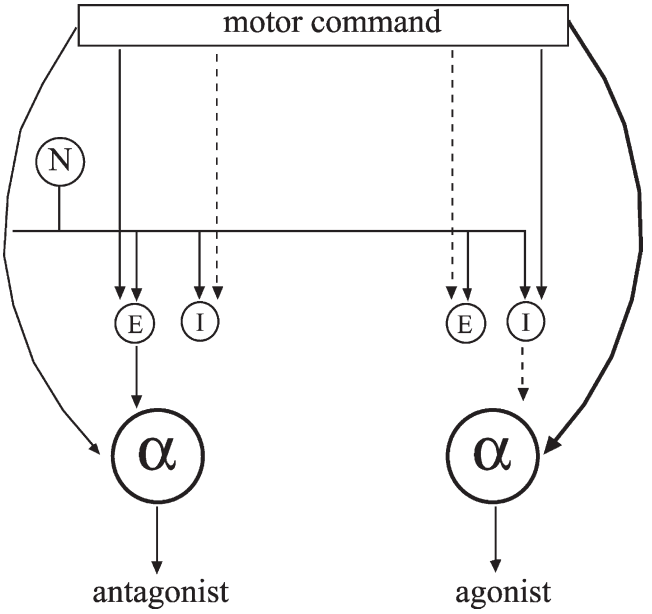

Fig. 2. Neural pathway suggested to form the basis of the pain-adaptation model. Feedback of nociceptive afferents (N) can via excitatory interneurons (E) or inhibitory interneurons (I) excite or inhibit the alpha motorneuron (α). Whether the output of inhibitory or excitatory interneurons dominates depends on the motor command. This nociceptive feedback suppresses the output of excitatory interneurons projecting on the agonists and of inhibitory interneurons projecting on the antagonists, resulting in a reduced activation of agonists and increased activation of antagonists. Dashed arrows refer to inhibitory inputs, solid arrows to excitatory inputs. Direct excitation of the motor neurons of the agonist and at a lower level of the antagonist by the motor command is illustrated by the thick and thin curved arrows respectively.

Table 1
Predictions of the pain–spasm–pain model regarding the difference in trunk muscle EMG amplitudes between patients and controls depending on the experimental task. The model predicts observations to lie in the ticked boxes (equal, higher, and lower refer to equal, higher, or lower EMG amplitudes in patients as compared to controls)

	Submaximal				Maximal		
	Rest (1)	Isometric (2)	Eccentric (3)	Concentric (4)	Isometric (5)	Eccentric (6)	Concentric (7)
Equal	—	—	—	—	✓	✓	✓
Higher	✓	✓	✓	✓	—	—	—
Lower	—	—	—	—	—	—	—

on the instantaneous motor command inhibition or excitation of the alpha motorneuron dominates.

The pain-adaptation model has motivated several neurophysiological studies addressing the effects of induced pain. Schwartz and Lund [113] found noxious pressure to cause changes in EMG and kinematics during cyclic jaw movements in decerebrate rabbits, which were consistent with the pain adaptation model. In an induced pain condition postural activity of the jaw muscles was increased with respect to baseline. However, it was not different from that in a sham pain condition [121]. Thus, in line with the model no consistent evidence for an effect of pain on resting activity of these muscles was found. Jaw kinematics were affected by pain in accordance with the model's predictions [120]. Clear evidence in support of the pain-adaptation model was found in a study by Graven-Nielsen et al. [41]. Induced pain in human gastrocnemius muscle was found to decrease the activity of this muscle during gait, while the activity of the tibialis anterior, its antagonist, increased. The reverse was found when pain was induced in the tibialis anterior muscle. Furthermore, motor unit firing rates were found to decrease in human forearm muscles after injection of hypertonic saline [14].

The pain-adaptation model predicts muscle activation to be reduced in agonists and increased in antagonists, irrespective of the level of exercise. The theory is somewhat aspecific as to what is meant by antagonists and agonists. In our interpretation, Lund et al. [78] used the term antagonists to refer to muscles that are lengthening and agonists to refer to muscles that are shortening. This interpretation is based on the predicted effects on kinematics. An alternative definition of antagonist and agonist roles is based on the sign of the moment produced by the muscle relative to the sign of the net moment [53]. This definition does clearly define agonists and antagonists in isometric as well as dynamic contractions. The implications of this definition may clearly deviate from the definition based on shortening versus lengthening. The fact that the originators of the model interpreted unchanged activity in static tasks as supportive of the model [121], is in line with our interpretation. Following this interpretation, the model predictions are given in Table 2.

4. Methods

This review was based on a literature search in PubMed using the search terms 'low-back pain and muscle activity' or 'low-back pain and electromyography' and several similar combinations. The references thus retrieved were screened on the basis of titles and abstracts and those papers apparently fulfilling our inclusion criteria (see below) were selected for further study. The literature retrieved in this way was supplemented with references from the authors' own databases, from previous reviews [31,110] and studies cited in the papers retrieved.

To be included studies had to be published in peer-reviewed journals in the English language. The study had to comprise a statistical comparison of trunk muscle activation (EMG amplitude, timing or recruitment pattern) between LBP patients and controls, or between an induced back pain condition and a no back pain condition.

To test the model predictions outlined above, the experimental tasks performed by the subjects were classified according to Tables 1 and 2. We further categorized the studies based on the characteristics of the patient group, the matching between patient and control group, and group size. In addition, details on data acquisition and analysis methods were noted. The most important issue in this respect was the normalization of EMG amplitudes. In general, absolute EMG amplitudes depend on many factors unrelated to the level of muscle activation, such as thickness of tissues overlying the muscle. Consequently, a systematic between-group difference may be expected when patients and controls are not carefully matched. Patients will tend to be less physically active than controls, which could cause a difference in thickness of the subcutaneous fat layer. To obtain a signal independent of such factors, normalization of EMG amplitudes to the amplitudes obtained in maximum voluntary contractions (MVC) is often used. However, in patients this procedure is considered unreliable, since patients are usually unwilling or unable to perform maximum contractions [12]. Normalization to submaximal contractions does not provide a solution, because in patients the EMG amplitudes during these submaximal tests can be expected to be similarly affected as the levels during the experimental task. Therefore, non-normalized EMG amplitudes are to be preferred in clinical studies. However, this will result in a low power due to the random variance in raw EMG amplitudes, where group size is inadequate and may even result in a bias where subject groups are not matched with respect to factors like body mass, body mass index, or skinfold thickness.

A relatively small number of studies was retrieved that dealt with timing of muscle activation and patterns of load sharing between muscles. These were separately reviewed in a more qualitative way.

5. Results

5.1. Effects of clinical pain on EMG amplitudes

Of the total number of 44 studies retrieved fulfilling the inclusion criteria, 30 reported a comparison of lumbar erector spinae (LES) EMG amplitudes between

Table 2
Predictions of the pain-adaptation model regarding the difference in trunk muscle EMG amplitudes between patients and controls depending on the experimental task. The model predicts observations to lie in the double ticked boxes (equal, higher, and lower refer to equal, higher, or lower EMG amplitudes in patients as compared to controls). Note that predictions on isometric tasks are not made explicit in the theory, hence the single tick boxes

	Submaximal				Maximal		
	Rest (1)	Isometric (2)	Eccentric (3)	Concentric (4)	Isometric (5)	Eccentric (6)	Concentric (7)
Equal	✓✓	✓			✓		
Higher			✓✓			✓✓	
Lower				✓✓			✓✓

patients and controls (Table 3). This material was used for testing the predictions of the pain–spasm–pain model and the pain-adaptation model. The 30 studies together described 101 separate experimental tasks, which were classified according to Tables 1 and 2. This classification made it necessary to include two extra categories. A number of studies reported on EMG of the LES in or around full trunk flexion. This posture did not fit our classification scheme and was thus added as a separate class (task 8). Furthermore, a number of studies reported on EMG amplitudes averaged over movement cycles, comprising an eccentric and concentric part. These tasks would comprise two separate classifications in Tables 1 and 2, for which the predictions of the pain-adaptation model diverged. Therefore, these studies were also summarized as a separate class (task 3/4).

The classifications of the experimental tasks in the 30 studies and the outcomes have been summarized in Table 4. The majority of studies involved measurements of EMG in rest postures, mainly standing and sitting. Very few studies dealt with maximal exertions.

Neither of the two models is consistently supported by the literature summarized in Table 4. Most clearly diverging predictions by the two models are made regarding rest postures and submaximal concentric contractions. For rest postures, the pain adaptation model predicts no change in activation and the pain–spasm–pain model predicts an increased activation. Regarding submaximal concentric contractions, the pain-adaptation model predicts decreased activation and the pain–spasm–pain model predicts increased activation. Focusing on the first of these tasks, the results appear more in line with the predictions of the pain-adaptation model. Regarding concentric contractions no clear trend is discernible. The trend in studies on submaximal isometric exertions appears in line with the general idea behind the pain-adaptation model that activation of agonistic muscles is reduced, though not when using a definition of agonistic activity based on movement direction. Most of these studies involved generating an extensor moment against gravity. The magnitude of the external load was usually not strictly controlled. Hence the reduced activation in the patient group could be due to slight postural adjustments.

It is important to note that part of the support for the pain-adaptation model relies on predictions of equal muscle activation in patients and controls. Of course, this equality cannot be statistically assessed. Many of the results on the first row (equal EMG amplitudes) in Table 4 could be due to a lack of discriminative power of the studies concerned. We therefore omitted studies with a lack of power due to low subject numbers. The cut-off points for low power were set at 25 subjects per group, when groups were not matched and 15 subjects per group when groups were matched (11 studies were omitted). The former figure was based on a conservative estimate for the coefficient of variation between subjects of 25%, a power of 0.80 and a difference between groups to be detected of 20% [86]. Normalization may lead to biased results, therefore also studies in which normalization of EMG or exertion levels might account for the (lack of) effects were omitted (six studies of which three also had a low number of subjects). The results of the remaining studies are shown in Table 5.

The overview in Table 5 shows two classes of tasks for which fairly strong evidence exists that increased muscle activation can occur in the patient group, i.e. in rest postures and in full flexion of the trunk. In rest postures, this finding appears dependent on the type of posture even within the same population (see studies 4 and 5). In full flexion, most healthy subjects show complete electromyographical silence of the LES. This phenomenon is called flexion–relaxation. In many patients flexion–relaxation is absent. These findings appear to contradict the pain-adaptation model.

5.2. Effects of induced pain on EMG amplitudes

An early study on the effects of pain induced by the injection of hypertonic saline in the LES reported an increase of the EMG amplitude of this muscles with increasing pain intensity and a subsequent reduction in EMG amplitude with diminishing pain [21]. These results were obtained in subjects sitting with back sup-

Table 3
Overview of studies comparing LES EMG amplitude between LBP patients and controls. The symbols =, +, and − refer to equal, higher, or lower EMG amplitudes in patients as compared to controls

Nr.	1st author	Year	Subjects			Diagnosis			Task class instruction		EMG normalisation		Results amplitude		
			# Pts	# Con	Matching yes/no	Specifici diagnosis yes/no	Diagnosis specific diagnosis yes/no	Duration acute/chronic	Class instruction	Instruction	Yes/no	MVC/submax	+	-	=
3a	Ahern [3]	1988	40	40	Yes	No		Chronic (> 6 months)	3/4	Full flexion/full extension	No			1	
3b									2	Full rotation to left and right				1	
3c									1	Standing					1
5a	Alexiev [5]	1994	40	40	No	No		Acute (< 1 month)	5	isometric extension 100% MVC	No				1
5b									2	Isometric extension 50% MVC					1
5c									2	Isometric extension 30% MVC					1
6a	Ambroz [6]	2000	30	30	Yes	No		Chronic (> 6 months)	1	Standing	No		1		
6b									8	Full flexion	Yes	Submax	1		
8a	Arena [8]	1989	20	29	No	Yes	Spondyloarthritis		1	Standing	No		1		
8b			52				Intervertebral disk disorders	3	Flex to 30 degrees					1	
8c			66				Unspecified musculoskeletal backache	4	Re-extend					1	
8d			17				Combined		1	Sitting supported			1		
8e			23				Other back pain		1	Sitting unsupported					1
8f									1	Prone					1
9a	Arena [9]	1991	46	20	No	Yes	21 Intervertebral disk disorder	1	Standing	No		1			
9b							25 Unspecified musculoskeletal pain	3	Flexion to 30 degrees					1	
9c									4	Re-extension					1
9d									1	Sitting supported			1		
9e									1	Sitting unsupported					1
9f									1	Prone					1
10a	Arendt-Nielsen [10]	1995	10	10	Yes	No		Chronic (2–14 yr)	2	Walking 4 km/h on treadmill	no		1		

(*continued on next page*)

Table 3 (*continued*)

Nr.	1st author	Year	Subjects			Diagnosis			Task class instruction		EMG normalisation		Results amplitude		
			# Pts	# Con	Matching yes/no	Specifici diagnosis yes/no	Diagnosis specific diagnosis yes/no	Duration acute/chronic	Class instruction	Instruction	Yes/no	MVC/submax	+	-	=
16a	Cassisi [16]	1993	21	12	No	Yes	7 Neurocompression	Chronic (mean 2.8 yr)	5	100% Isometric extension	no			1	
16b							6 Spondylolisthesis 3 Myofascial syndrome 5 Combined pain syndrome		1	Rest				1	
17a	Chiou [17]	1999	47	40	No	No		Unknown	1	Upright holding light load	no		1		
17b									2	Upright holding moderate load					1
17c									2	Upright holding heavy load				1	
17d									2	Flexed holding light load				1	
17e									2	Flexed holding moderate load				1	
17f									2	Flexed holding heavy load				1	
22a	Cohen [22]	1986	13	13	Yes	No		Chronic (> 6 months)	1	Standing	no				1
22b									2	45° Flexion					1
22c									8	Full flexion					1
22d									2	Forward pelvic tilt					1
22e									2	Backward pelvic tilt					1
22f									1	Unsupported sitting					1
22g									1	Supine knees bent					1
23a	Collins [23]	1982	11	11	No	No		Chronic	1	Sitting	No				1
23b									1	standing					1
23c									2	45°Flexion				1	
23d									8	Maximum bend					1
23e									2	Forward pelvic tilt				1	
23f									2	Backward pelvic tilt					1
23g									1	Unsupported sitting					1
23h									1	Supine knees bent					1

Table 3 (*continued*)

Nr.	1st author	Year	Subjects			Diagnosis			Task class instruction		EMG normalisation		Results amplitude		
			# Pts	# Con	Matching yes/no	Specifici diagnosis yes/no	Diagnosis specific diagnosis yes/no	Duration acute/chronic	Class instruction	Instruction	Yes/no	MVC/submax	+	-	=
24a	Cram [24]	1983	9	14	No	No		Unknown	1	Sitting	No				1
24b									1	Standing					1
25a	Danneels [25]	2002	75	77	Yes	No		51 Chronic (> 1 yr)	2	Holding lordosis	Yes	MVC		1	
25b								24 Subacute (< 1 yr)	2	Holding lordosis, moving extr					1
25c									3/4	Holding lordosis moving trunk					1
25d									3	Trunk/leg lifting				1	
32a	Fischer [32]	1985	9	12	No	Yes	Palpable muscle spasm	Chronic (>1 yr)	1	Sleeping	No		1		
35a	Flor [35]	1985	17	34	Yes	No		Chronic (> 3 months)	1	Sitting	No		1		
35b									1	Stress			1		
38a	Grabel [38]	1974	30	30	Yes	No		Chronic (> 6 months)	1	Relaxed	No		1		
43a	Hemborg [43]		20	20	Yes	No		Chronic	4	10 kg Lifting	Yes	MVC	1		
43b									4	25 kg Lifting			1		
43c									4	40 kg Lifting			1		
43d									3	10 kg Lowering			1		
43e									3	25 kg Lowering			1		
43f									3	40 kg Lowering			1		
52a	Hoyt [52]	1981	40	40	No	No		Chronic (> 1 yr)	1	Semi Fowler's	No				1
52b									1	Sitting					1
52c									1	Standing			1		
64a	Kaigle [64]	1998	7	6	No	No		Chronic (> 3 yr)	8	Full flexion	Yes	Submax	1		
64b									1	Standing					1
67a	Kravitz [67]	1981	22	17	No	Yes	Palpable muscle spasm	Intermittent	1	Prone	No				1
67b									1	Prone, contract other muscle					1
67c									1	Prone, minimally contract ES			1		

Table 3 (*continued*)

Nr.	1st author	Year	Subjects			Diagnosis			Task class instruction		EMG normalisation		Results amplitude		
			# Pts	# Con	Matching yes/no	Specifici diagnosis yes/no	Diagnosis specific diagnosis yes/no	Duration acute/chronic	Class instruction	Instruction	Yes/no	MVC/submax	+	-	=
68a	Lariviere [68]	2000	15	18	Yes	No		Chronic	3/4	Flexion/extension unloaded	No				1
68b									3/4	Lateral bending unloaded					1
68c									3/4	Flexion/extension loaded (12 kg)					1
68d									3/4	Lateral bending loaded (12 kg)					1
75a	Lisinski [75]	2000	62	31	No	No		Chronic	2	Sorensen test	No			1	
93a	Miller [93]	1985	11	12	Yes	No		Chronic (> 6 months)	1	Sitting supported	Yes	Submax			1
93b									1	Standing					1
93c									2	Sitting unsupported + manual task					1
94a	Ng [94]	2002	12	12	Yes	No			5	Isometric rotation 100% MVC	Yes/no	MVC			1
94b									2	Isometric rotation 70% MVC					1
94c									2	Isometric rotation 50% MVC					1
94d									2	Isometric rotation 30% MVC					1
96a	Nouwen [96]	1987	20	20	No	No		Chronic (> 1 yr)	3	Flexion	No				1
96b									4	Extension					1
96c									3/4	Lateral bending					1
96d									2	Rotation					1
100a	Paquet [100]	1994	10	10	No	Yes	Ligament injury	Acute (7 days –7 weeks)	8	Flexion	No			1	

Table 3 (*continued*)

Nr.	1st author	Year	Subjects			Diagnosis			Task class instruction		EMG normalisation		Results amplitude		
			# Pts	# Con	Matching yes/no	Specifici diagnosis yes/no	Diagnosis specific diagnosis yes/no	Duration acute/chronic	Class instruction	Instruction	Yes/no	MVC/submax	+	-	=
100b									4	Extension					1
109a	Robinson [109]	1992	19	12	No	Yes	6 Neurological complaint 6 Mechanical instability 3 Musculoligamentous 4 Neurological and mechanical	3,4	Flexion/extension	No			1		
115a	Sherman [115]	1985	83	15	No	No		Unknown	1	Standing	No				1
115b									3	Flexion to 30°					1
115c									4	Re-extension					1
115d									1	Sitting supported					1
115e									1	Sitting unsupported					1
115f										Above combined			1		
116a	Sihvonen [116]	1991	87	25	Yes	No	No back surgery	Chronic (> 0.8 yr)	8	Full flexion	No		1		
116b									3	Flexion			1		
116c									1	Standing			1		
116d									4	Extension				1	
131a	Triano [131]	1987	41	7	No	No		Mixed	8	Full flexion	No		1		
135a	Watson [135]	1997	70	20	No	No		Chronic (> 6 months)	1	Standing	No				1
135b									8	Full flexion			1		
135c									3	Flexion				1	
135d									4	Extension				1	

Table 4
Classification of the experimental tasks and outcomes of the 30 studies included. Numbers refer to the numbers of the studies in Table 3 and the letters refer to the experimental task in the study also given in Table 3. Equal, higher, and lower refer to equal, higher, or lower EMG amplitudes in patients as compared to controls

	Submaximal					Maximal			Full
	Rest (1)	Isometric(2)	Eccentric(3)	Concentric(4)	Ecc/conc(3/4)	Isometric(5)	Eccentric(6)	Concentric(7)	Flexion(8)
Equal	3c 8e 8f 9e 9f 22a 22f 23a 23b 23g 23h 24a 24b 38a 52a 52b 64b 67a 67b 93a 93b 115a 115d 115e 135a	5b 5c 17b 22b 22d22e 23f 25b 93c 94b 94c 94d 96d	8b 9b 96a 115b	8c 9c 96b 100b 115c	25c 68a 68b 68c 68d 96c	5a 94a			22c 23d
Higher	6a 8a 8d 9a 9d 17a 35a 35b 52c 67c 116c	10a	43d 43e 43f 116b	43a 43b 43c					3a 6b 64a 100a 116a 131a 135b
Lower	16b	3b 17c 17d 17e 17f 23c 23e 25a 75a	25d 135c	116d 135d	3a 109a	16a			

Table 5
Classification of the experimental tasks and outcomes of the 26 studies remaining after selection. Studies with low discriminative power due to low subject numbers (<25 per group, when groups were not matched; <15 per group when groups were matched; 11 studies), and studies in which normalization of EMG or exertion levels might account for the effects (six studies, three also had a low number of subjects) were omitted. Numbers refer to the numbers of the studies in Table 3 and the letters refer to the experimental task in the study also given in Table 3

	Submaximal					Maximal			Full
	Rest(1)	Isometric(2)	Eccentric(3)	Concentric(4)	Ecc/conc(3/4)	Isometric(5)	Eccentric(6)	Concentric(7)	Flexion(8)
Equal	3c 8e 8f 9e 9f 38a 52a 52b 64b 67a 67b 115a 115d 115e 135a	17b	8b 9b 115b	8c 9c 115c	68a 68b 68c 68d				
Higher	6a 8a 8d 9a 9d 17a 35a 35b 52c 67c 116c		116b						3a 6b 116a 131a 135b
Lower		3b 17c 17d 17e 17f 75a	135c	116d 135d	3a 109a				

port. The results were not statistically tested and judging from the graphs appear very weak in some subjects. Arendt-Nielsen et al. [10] studied the effects of induced pain in the lumbar erector spinae during walking. They found a significant increase in activation, which was most pronounced during the ipsilateral swing phase. During the double-support phase LES activation was found to be reduced. Zedka et al. [141] tested subjects in trunk flexion and extension movements. During flexion, the LES activation appeared unchanged after injection of hypertonic saline. In full flexion, increased activity was found on the injected painful and non-injected painless side, which was associated with a reduced range of motion. However, when subjects overcame this limitation of the range of motion by instruction and feedback, the increase in activity on the painful side remained. During re-extension the LES activation was found to be reduced, this effect also remained on the painful side when the subject was guided to perform the movement identical to the painless control movement.

The results of the studies showed no consistent support for either of the two models.

5.3. Effects of clinical pain on timing of muscle activation

Nine studies were retrieved that focused on effects of clinical pain on timing of trunk muscle activation. Hemborg and Moritz [43] found a longer period of LES activation during lifting and lowering of loads. This effect may be associated with the absence of flexion–relaxation in patients. Leinonen et al. [73], studied the recruitment order of the LES and the hip extensors (gluteus maximus and hamstrings) during flexion–extension movements. The recruitment order was the same in patients and controls. Only the duration of gluteus activity was reduced in patients.

Hodges and coworkers performed a number of studies on timing of trunk muscle activation prior to and shortly after rapid voluntary movements of the limbs [46,47,49,50,142]. Anticipatory activation of the transversus abdominus muscle was a consistent finding in the controls, but was absent in the patients. In general trunk muscle activation in the patients was delayed.

Sudden perturbations of trunk posture showed delayed responses of the trunk muscles both in a sudden release experiment [106] and sudden loading experiments [82]. The amplitude of the LES response to sudden loading also was reduced. Short latency reflexes in the LES in response to sudden upper limb loading appeared unaffected, but in contrast to controls the latency was not shortened when subjects anticipated the perturbation [74].

Again these studies do not consistently support either of the two models. The results found by Hemborg and Moritz [43] appear more in line with the pain–spasm–pain model, whereas the results of the perturbation experiment could be considered more in line with the pain-adaptation model in the sense that reduced excitability was found.

5.4. Effects of clinical pain on load sharing between muscles

Load sharing between muscles was studied in nine of the studies retrieved. Seven of these focused on left/right differences in LES activation. Alexiev [5] reported a significant difference between painful and painless sides in patients, with lower activation on the painful side, when exerting isometrically against resistance. No asymmetry in the controls was found, but groups were not directly compared. Hoyt et al. [52] reported significantly more asymmetry in muscle activation during standing in patients as compared to controls. A similar trend, which did not reach significance, was reported by Cram and Steger [24]. Triano and Luttges [130] reported significantly more asymmetry of paraspinal EMG amplitudes in LBP patients than in controls during several static tasks (standing, sitting, trunk flexion). No significant differences were found in a study on standing [3] and a study on gait [10]. Also with unilaterally induced pain no left/right difference in LES activation during gait emerged [10]. Grabiner et al. [39] found more asymmetric activation in patients than in controls during ramp contractions. In the patients, the temporal pattern of activation on each side was highly irregular without covariation of the moment produced. This suggests that load sharing was variable over time in the patient group.

O'Sullivan et al. [97] studied load sharing between abdominal muscles during an exercise called 'abdominal hollowing'. Although activation levels of the internal oblique and rectus abdominus muscles were not different between patients and controls, the ratio of the two was different with a higher activation of rectus abdominus relative to the internal oblique in the patient group. We recently studied the same ratio during free planar motions through the upright posture and during ramp contractions [26]. No differences between patients and matched controls were found. In contrast, we did find consistent evidence in the ramp contractions and planar motions that the LES was more active relative to the thoracic erector spinae in the patient group as compared to the control group. In the free motions ratios of antagonist over agonist EMG amplitudes were higher in the patients, indicating more cocontraction. Simulation with a mechanical model suggested that these changed recruitment patterns would enhance spinal stability in the patient group.

6. Discussion

The literature reviewed here reveals that neither one of the two models adequately predicts the effects of back pain on trunk muscle activation. In some cases evidence for reduced activation is found in line with the pain-adaptation model and in conflict with the pain–spasm–pain model. These changes appear to be adaptive in that heavy exertion of painful muscles and high accelerations that may impose a risk of pain provocation are avoided. In line with this Marras et al. [84,85] reported that LBP patients generally perform movements relatively slow. However, in other cases increased activation is found, conflicting with the prediction of the pain-adaptation model. Moreover, the wide variance in results within and between subjects appears irreconcilable with the hard-wired neural pathway proposed in the pain-adaptation model. In addition, some of the data reviewed indicate that pain may in some cases lead to disturbances of motor control, which are not likely to be adaptive, such as for instance left/right asymmetry of LES activation and the erratic temporal pattern of LES activation reported by Grabiner et al. [39]. Also the delayed

responses to perturbations of trunk equilibrium might fall into this category. As we will argue below, a loss of control may actually be one of the reasons why muscle activation patterns are adapted.

We suggest that instances of increased activation may be adaptive in nature. The nature of these adaptations however appears much more complicated than suggested by the pain-adaptation model. First the changes may be tuned to the individual problem probably through learning, as is suggested by the substantial variance between subjects. In support of this Arena et al. [8] found erector spinae activity to differ between groups diagnosed to have LBP of different origins. However, too few studies have attempted to discriminate between different diagnostic groups to allow more definitive conclusions. It is also conceivable that between-subject variation may occur due to differences between patients in the developmental stage of their low-back disorder. However, most of the studies reviewed have included only patients with chronic LBP. Second, the changes appear tuned to the mechanical circumstances, or in other words to the task at hand. Both this and the former point are illustrated in a study by Sherman [115]. In this study each LBP patient was found to show increased LES activation during at least one of the five experimental tasks, but when analyzing the data per task no differences between patients and controls were found.

We surmise that the changes observed due to pain in general are aimed at avoiding noxious tensile stresses in injured structures. Our interpretation is in line with the hypothesis on stability as a cause of LBP put forward by Panjabi [98,99,99a]. In rest postures, stability of the spine can be critical [19]. If the spine becomes unstable, excessive rotations of segments will occur and pain may be provoked [98,99]. The increased activation in rest postures, which reflects increased cocontraction and the changes in load sharing between lumbar and thoracic erector spinae may be aimed at enhancing stability. Absence of flexion-relaxation is associated with limited segmental rotations [64]. It prevents excessive lengthening of muscle, ligaments, and posterior annulus by supporting the upper body against gravity through active muscle force, rather than with elastic forces in the aforementioned structures.

Three reasons could be put forward, as to why back pain patients would need additional muscular stabilization of their spines:

- the passive stiffness of the spine is reduced as a consequence of damage to disc or ligaments;
- muscle force and consequently the capacity to correct perturbations is reduced;
- sensorimotor integration is disturbed, hampering corrective responses.

Below, each of these possible reasons will be discussed.

LBP in many cases appears to be caused by injury to the ligaments or discs as a consequence of mechanical overloading [1,29,104]. These injuries and especially those of the intervertebral discs have a substantial effect on the mechanical behavior of the spine. In relation to spinal stability the main effects are:

- an increase of the neutral zone, the part of the movement trajectory where stiffness is minimal [62,63].
- a reduction of stiffness outside the neutral zone [62,63,105].
- an increase of the range of motion [62,63,105].

Although it is difficult to verify the existence of such changes in living subjects, there is ample evidence that in many LBP patients the stiffness of affected spinal motion segments is decreased and the range of motion increased [36,69,112,114]. These changes will lead to a reduced spinal stability as evidenced by the fact that injured spinal motion segments buckle under lower compression forces as compared to intact specimens [136]. This buckling occurs at a very high velocity, which might preclude muscular corrections. Consequently, adaptations of muscular activation might be required to compensate for the reduced stiffness. This could involve increased cocontraction [18,20,40] and adaptations in load sharing between extensor muscles [26]. With respect to the increased LES activation found in patients in full trunk flexion, it seems likely that this is explained by the need to limit the segmental range of motion [64,116]. It could limit the excursion of the vertebrae with respect to each other where the passive stiffness is insufficient to do so.

Many studies have shown a reduced trunk muscle force in LBP patients as compared to healthy controls [72,90,126,127]. This is not only caused by a lack of maximal activation during the test, since wasting of the extensor muscle mass [44,45] and a loss of type II fibers [83] have been demonstrated. Furthermore, several studies have shown that LBP patients are characterized by a reduced endurance of the trunk extensor muscles [13,79,89,95,111]. On the basis of these findings it is likely that LBP patients will be less able to rapidly develop trunk muscle force. This would limit their capacity to correct perturbations of trunk equilibrium and prevent spinal instability.

Injuries of spinal ligaments are likely to cause a disturbance of the control of trunk equilibrium, since ligaments have been shown to have an important sensory function in feedback control of joint position [58,117]. This function probably also holds for injuries of the annulus fibrosus, which is richly supplied with mechanoreceptors [108,139]. A number of animal experiments and limited experimentation in humans has indeed shown a reflexive coupling between damage to the annulus and ligaments on one hand and the activity of the

multifidus and longissimus muscles on the other hand [51,54,55,119,123,119a]. In addition, reduced proprioception [15,37] and disturbances of postural control [80] have been found in back pain patients. The same study demonstrated increased reaction times in LBP patients. Impaired postural control in back pain patients is associated with a reduced capacity to react to perturbations of trunk equilibrium [107].

In summary, all three subsystems that subserve spinal stability (the passive system, the control system and the muscular system [98]) appear to be affected in LBP, though not necessarily all at the same time. The reduction in passive spinal stiffness would increase the risk of buckling under perturbations, whereas the changes in the control system and muscular system would impair the capacity to respond to perturbations. Recent studies have indeed confirmed that LBP patients respond less adequately to trunk perturbations [82,106]. Alterations of trunk muscle recruitment patterns might be functional in LBP patients, since they would stiffen the trunk, thus precluding the probability of perturbations to which the patient could not adequately respond.

The above rationale might also account for the disappearance of anticipatory muscle activation and the consequent anticipatory postural adjustments in back pain patients preceding fast limb movements [46,47,49,50]. Healthy subjects generally use specific anticipatory control to counteract ensuing perturbations of trunk equilibrium. For instance when raising the arms forward, the back muscles are activated before the arm muscles to counteract the flexing torque on the trunk imposed by the arm movement [11,33,48,140]. This anticipatory activation is specific with respect to the magnitude and direction of the ensuing perturbation and antagonistic cocontractions appear to be absent [11,33,48,140]. When moving the arms, specific anticipation is possible since the perturbation is entirely internal. However, it turns out that this can be generalized to situations in which the mechanical nature of the perturbation is to a lesser extent under the control of the subject. A good example of an everyday activity in which the mechanical nature of the perturbation is a priori unknown to the subject is the lifting of a load. The mass and the center of mass position can only be known when the object has already been lifted [60,61]. It appears that also when lifting loads the trunk extensor musculature is activated in an anticipatory fashion, whereas no anticipatory activation of the antagonistic abdominal muscles is found [27]. In addition, this anticipatory activation was shown to be specific with respect to load magnitude [77] and center of mass position [27]. With an incorrect expectation of the ensuing perturbation, anticipatory actions will actually further disturb equilibrium [128]. Therefore, in cases where the possibilities for corrections are limited, such as appears to be the case in LBP patients [81,106], specific anticipation might impose a risk. For this reason, patients might prepare by cocontracting muscles, as healthy subjects do when specific anticipation is impossible [71]. Furthermore, anticipatory postural adjustments might be suppressed as was found in healthy subjects when the risk associated with balance loss was high [2] and in patient groups with problems in maintaining whole-body equilibrium [70]. Finally it is conceivable that anticipatory adjustments are less relevant in patients, since the muscle activation patterns, such as elucidated in the present study, provide them with adequate stability to deal with ensuing perturbations [50a]. Increased coactivation could even account for the delayed and reduced responses after a perturbation [81,106], since prior activation of muscles reduces the amplitude of responses in these muscles to perturbations [122].

Besides the positive effect of pain-related changes in muscular recruitment, some negative consequences are likely to occur. Hyperactivity could cause pain in the muscles themselves, as proposed in the pain–spasm–pain model. In addition, increased cocontraction will increase the forces acting on the spine [28,40]. However, the increased activation reported in the literature review is generally only small or moderate. Maybe more importantly, both cocontraction and selective derecruitment of muscles will limit functional abilities of patients. It is possible that changes in recruitment of trunk musculature remain present after their functional significance has disappeared, when injured structures have recovered. In chronic LBP, aspects of pain behavior in many cases appear to remain, whereas the physiological cause may no longer be present [34,132]. The fear of movement and re-injury, which characterizes many LBP patients [133], could underlie the remaining changes. This assumption is supported by the relationship found between hyperactivity and other aspects of pain behavior [4] and by a study indicating that mental and hence mechanically irrelevant stressors may trigger the responses observed in muscle activation [35]. However, these effects of mental stressors were not generally observed [22,23,38]. Nevertheless, caution should be exercised when rehabilitating LBP patients with the sole purpose of restoring a normal muscle recruitment pattern. The ‘abnormalities’ may represent compensation mechanisms to stabilize the spine. The problem a clinician is facing then, is to differentiate between adaptive changes and unwanted residuals of former injury. Further scientific research may provide the knowledge and methods to support clinical decision-making.

7. Conclusion

Findings on trunk muscle recruitment in LBP patients fit neither the pain–spasm–pain model, nor the pain-

adaptation model. The changes observed are task-dependent, related to the individual problem and hence highly variable between and probably within individuals. We propose that the alterations in trunk muscle recruitment in patients are functional in that they reduce the probability of noxious tissue stresses by limiting range of motion and providing stabilization of the spine. This explanation should be tested in future experiments specifically designed to refute hypotheses derived from it.

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References

[1] M.A. Adams, P. Dolan, Could sudden increases in physical activity cause degeneration of intervertebral discs?, The Lancet 350 (1997) 734–735.
[2] A.L. Adkin, J.S. Frank, M.G. Carpenter, G.W. Peysar, Fear of falling modifies anticipatory postural control, Exp. Brain Res. 143 (2002) 160–170.
[3] D.K. Ahern, M.J. Follick, J.R. Council, N. Laser-Wolston, H. Litchman, Comparison of lumbar paravertebral EMG patterns in chronic low back pain patients and non-patient controls, Pain 34 (1988) 153–160.
[4] D.K. Ahern, D.J. Hannon, A.J. Goreczny, M.J. Follick, J.R. Parziale, Correlation of chronic low-back pain behavior and muscle function examination of the flexion-relaxation response, Spine 15 (1990) 92–95.
[5] A.R. Alexiev, Some differences of the electromyographic erector spinae activity between normal subjects and low back pain patients during the generation of isometric trunk torque, Electromyogr. Clin. Neurophysiol. 34 (1994) 495–499.
[6] C. Ambroz, A. Scott, A. Ambroz, E.O. Talbott, Chronic low back pain assessment using surface electromyography, J. Occup. Environ. Med. 42 (2000) 660–669.
[7] G.B.J. Andersson, Epidemiological features of chronic low-back pain, The Lancet 354 (1999) 581–585.
[8] J.G. Arena, R.A. Sherman, G.M. Bruno, T.R. Young, Electromyographic recordings of 5 types of low back pain subjects and non-pain controls in different positions, Pain 37 (1989) 57–65.
[9] J.G. Arena, R.A. Sherman, G.M. Bruno, T.R. Young, Electromyographic recordings of low back pain subjects and non-pain controls in six different positions: effect of pain levels, Pain 45 (1991) 23–28.
[10] L. Arendt-Nielsen, T. Graven-Nielsen, H. Svarrer, P. Svensson, The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study, Pain 64 (1995) 231–240.
[11] A.S. Aruin, M.L. Latash, Directional specificity of postural muscles in feed-forward postural reactions during fast voluntary arm movements, Exp. Brain Res. 103 (1995) 323–332.
[12] D.S. Beimborn, M.C. Morrissey, A review of the literature related to trunk muscle performance, Spine 13 (1988) 661.
[13] H.J. Biedermann, G.L. Shanks, W.J. Forrest, J. Inglis, Power spectral analyses of electromyographic activity. Discriminators in the differential assessment of patients with chronic low back pain, Spine 16 (1991) 1179–1184.
[14] L. Birch, H. Christensen, L. Arendt-Nielsen, T. Graven-Nielsen, K. Sogaard, The influence of experimental muscle pain on motor unit activity during low-level contraction, Eur. J. Appl. Physiol. 83 (2000) 200–206.
[15] S. Brumagne, P. Cordo, R. Lysens, S. Verscueren, S. Swinnen, The role of paraspinal muscle spindles in lumbosacral position sense in individuals with and without low back pain, Spine 25 (2000) 989–994.
[16] J.E. Cassisi, M.E. Robinson, P. O'Conner, M. MacMillan, Trunk strength and lumbar paraspinal muscle activity during isometric exercise in chronic low-back pain patients and controls, Spine 18 (1993) 245–251.
[17] W.-K. Chiou, Y.-H. Lee, W.-J. Chen, Use of the surface EMG coactivational pattern for functional evaluation of trunk muscles in subjects with and without low-back pain. Int, J. Indust. Ergon. 23 (1999) 51–60.
[18] J. Cholewicki, K. Juluru, S.M. McGill, Intra-abdominal pressure mechanism for stabilizing the lumbar spine, J. Biomech. 32 (1999) 13–17.
[19] J. Cholewicki, S.M. McGill, Mechanical stability of the in-vivo lumbar spine—implications for injury and chronic low-back-pain, Clin. Biomech. 11 (1996) 1–15.
[20] J. Cholewicki, M.M. Panjabi, A. Khatchatryan, Stabilizing function of trunk flexor/extensor muscles around a neutral spine posture, Spine 22 (1997) 2207–2212.
[21] C.R. Cobb, H.A. DeVries, R.T. Urban, C.A. Luekens, R.J. Bagg, Electrical activity in muscle pain, Am. J. Phys. Med. 54 (1975) 80–87.
[22] M.J. Cohen, G.A. Swanson, B.D. Naliboff, S.L. Schandler, D.L. McArthur, Comparison of electromyographic response patterns during posture and stress tasks in chronic low back pain patterns and control, J. Psychosomat. Res. 30 (1986) 135–141.
[23] G.A. Collins, M.J. Cohen, B.D. Naliboff, S.L.C. Schandler, omparative analysis of paraspinal and frontalis EMG, heart rate and skin conductance in chronic low back pain patients and normals to various postures and stress, Scand. J. Rehab. Med. 14 (1982) 39–46.
[24] J.R. Cram, J.C. Steger, EMG scanning in the diagnosis of chronic pain, Biofeedback Self-Reg 8 (1983) 229–241.
[25] L.A. Danneels, P.L. Coorevits, A.M. Cools, G.G. Vanderstraeten, D.C. Cambier, E.E. Witvrouw, C.H. De, Differences in electromyographic activity in the multifidus muscle and the iliocostalis lumborum between healthy subjects and patients with sub-acute and chronic low back pain, Eur. Spine J. 11 (2002) 13–19.
[26] J.H. van Dieën, J. Cholewicki, A. Radebold, Trunk muscle recruitment patterns in low back pain patients enhance the stability of the lumbar spine, in press.
[27] J.H. van Dieën, M.P. de Looze, Directionality of anticipatory activation of trunk muscles in a lifting task depends on load knowledge, Exp. Brain Res. 128 (1999) 397–404.
[28] J.H. van Dieën, M.P. de Looze, Sensitivity of single-equivalent trunk extensor muscle models to anatomical and functional assumptions, J. Biomech. 32 (1999) 195–198.
[29] J.H. van Dieën, H. Weinans, H.M. Toussaint, Fractures of the lumbar vertebral endplate in the etiology of low back pain. A hypothesis on the causative role of spinal compression in a-specific low back pain, Med. Hypotheses 53 (1999) 246–252.
[30] M. Djupsjöbacka, H. Johansson, M. Bergenheim, Influences on the gamma-muscle-spindle system from muscle afferents stimulated by increased intramuscular concentrations of arachidonic acid, Brain Res. 663 (1994) 293–302.
[31] J.J. Dolce, J.M. Raczynski, Neuromuscular activity and electromyography in painful backs: psychological and biomechanical

models in assessment and treatment, Psychol. Bull. 97 (1985) 502–520.
[32] A.A. Fischer, C.H. Chang, Electromyographic evidence of paraspinal muscle spasm during sleep in patients with low back pain, Clin. J. Pain 1 (1985) 147–154.
[33] I.B.M. van der Fits, A.W.J. Klip, L.A. van Eykern, M. Hadders-Algra, Postural adjustments accompanying fast pointing movements in standing, sitting and lying adults, Exp. Brain Res. 120 (1998) 202–216.
[34] H. Flor, D.C. Turk, Etiological theories and treatments for chronic back pain. I. Somatic models and interventions, Pain 19 (1984) 105–121.
[35] H. Flor, D.C. Turk, N. Birbaumer, Assessment of stress-related psychophysiological reactions in chronic back pain patients, J. Consult. Clin. Psychol. 53 (1985) 354–364.
[36] S.D. Gertzbein, Segmental instability of the lumbar spine: in vitro and in vivo studies, Sem. Spine Surg. 3 (1991) 130–135.
[37] K.P. Gill, M.J. Callaghan, The measurement of lumbar proprioception in individuals with and without low back pain, Spine 23 (1998) 371–377.
[38] Grabel J.A. Electromyographic study of low back muscle tension in subjects with and without chronic low back pain. Diss. Abstr. Int. B 1073; 34:2929-2930
[39] M.D. Grabiner, T.J. Koh, A. El Ghazawi, Decoupling of bilateral paraspinal excitation in subjects with low back pain, Spine 17 (1992) 1219–1223.
[40] K.P. Granata, W.S. Marras, Cost-benefit of muscle cocontraction in protecting against spinal instability, Spine 25 (2000) 1398–1404.
[41] T. Graven-Nielsen, P. Svensson, L. Arendt-Nielsen, Effects of experimental muscle pain on muscle activity and co-ordination during static and dynamic motor function, Electroenceph. Clin. Neurophysiol. 105 (1997) 156–164.
[42] X. He, U. Proske, H.G. Schaible, R.F. Schmidt, Acute inflammation of the knee joint in the cat alters responses of flexor motorneurons to leg movements, J. Neurophysiol. 59 (1988) 326–340.
[43] B. Hemborg, U. Moritz, Intra-abdominal pressure and trunk muscle activity during lifting. II. Chronic low-back patients, Scand. J. Rehab. Med. 17 (1985) 5–13.
[44] J.A. Hides, C.A. Richardson, G.A. Jull, Multifidus muscle recovery is not automatic after resolution of acute, first-episode low-back-pain, Spine 21 (1996) 2763–2769.
[45] J.A. Hides, M.J. Stokes, M. Saide, G.A. Jull, D.H. Cooper, Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain, Spine 19 (1994) 165–172.
[46] P.W. Hodges, Changes in motor planning of feedforward postural responses of the trunk muscles in low back pain, Exp. Brain Res. 141 (2001) 261–266.
[47] P.W. Hodges, C.A. Richardson, Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis, Spine 21 (1996) 2640–2650.
[48] P.W. Hodges, C.A. Richardson, Feedforward contraction of transversus abdominis is not influenced by the direction of arm movement, Exp. Brain Res. 114 (1997) 362–370.
[49] P.W. Hodges, C.A. Richardson, Delayed postural contraction of transversus abdominis in low back pain associated with movement of the lower limb, J. Spinal Disord. 11 (1998) 46–56.
[50] P.W. Hodges, C.A. Richardson, Altered trunk muscle recruitment in people with low back pain with upper limb movement at different speeds, Arch. Phys. Med. Rehab. 80 (1999) 1005–1012.
[50a] P.W. Hodges, G.L. Moseley, Pain and motor control of the lumbopelvic region: effect and possible mechanisms, J. Electromyogr. Kinesiol. 13 (2003)
[51] S. Holm, A. Indahl, M. Solomonow, Sensorimotor control of the spine, J. Electromyogr. Kinesiol. 12 (2002) 219–234.
[52] W.H. Hoyt, H.H. Hunt Jr., M.A. De Pauw, D. Bard, F. Shaffer, J.N. Passias, D.H. Robbins Jr., D.G. Runyon, S.E. Semrad, J.T. Symonds, K.C. Watt, Electromyographic assessment of chronic low-back pain syndrome, J. Am. Osteopath. Assoc. 80 (1981) 728–730.
[53] R.E. Hughes, J.C. Bean, D.B. Chaffin, Evaluating the effect of co-contraction in optimization models, J. Biomech. 28 (1995) 875–878.
[54] A. Indahl, A. Kaigle, O. Reikeras, S. Holm, Electromyographic response of the porcine multifidus musculature after nerve stimulation, Spine 20 (1995) 2652–2658.
[55] A. Indahl, A.M. Kaigle, O. Reikeras, S.H. Holm, Interaction between the porcine lumbar intervertebral disc, zygapophysial joints, and paraspinal muscles, Spine 22 (1997) 2834–2840.
[56] M. Jackson, M. Solomonow, B. Zhou, R.V. Baratta, M. Harris, Multifidus EMG and tension-relaxation recovery after prolonged static lumbar flexion, Spine 26 (2001) 715–723.
[57] E. Jacobson, Progressive Relaxation; A Physiological and Clinical Investigation of Muscular States and their Significance in Psychology and Medical Practice, University of Chicago Press, Chicago, IL, 1944.
[58] H. Johansson, P. Sjölander, Neurophysiology of joints, in: V. Wright, E.L. Radin (Eds.), Mechanics of Human Joints, Marcel Dekker, New York, 1993, pp. 243–290.
[59] H. Johansson, P. Sojka, Pathophysiological mechanisms involved in genesis and spread of muscular tension in occupational muscle pain and in chronic musculoskeletal pain syndromes. A hypothesis, Med. Hypotheses 35 (1991) 196–203.
[60] R.S. Johansson, B.B. Edin, Predictive feed-forward sensory control during grasping and manipulation in man, Biomed. Res. 14 (Suppl. 4) (1993) 95–106.
[61] R.S. Johansson, G. Westling, Coordinated isometric muscle commands adequately and erroneously programmed for the weight during lifting tasks with precision grip, Exp. Brain Res. 71 (1988) 59–71.
[62] A.M. Kaigle, S.H. Holm, T.H. Hansson, Experimental instability in the lumbar spine, Spine 20 (1995) 421–430.
[63] A.M. Kaigle, S.H. Holm, T.H. Hansson, Kinematic behavior of the porcine lumbar spine: a chronic lesion model, Spine 22 (1997) 2796–2806.
[64] A.M. Kaigle, P. Wessberg, T.H. Hansson, Muscular and kinematic behavior of the lumbar spine during flexion-extension, J. Spinal Disord. 11 (1998) 163–174.
[65] Y.M. Kang, J.D. Wheeler, J.G. Pickar, Stimulation of chemosensitive afferents from multifidus muscle does not sensitize multifidus muscle spindles to vertebral loads in the lumbar spine of the cat, Spine 26 (2001) 1528–1536.
[66] M.P. Kaufman, J.C. Longhurst, K.J. Rybicki, J.H. Wallach, J.H. Mitchel, Effects of static muscular contraction on impulse activity of groups III and IV afferents in cats, J. Appl. Physiol. 55 (1983) 105–112.
[67] E. Kravitz, M.E. Moore, A. Glaros, Paralumbar muscle activity in chronic low back pain, Arch. Phys. Med. Rehab. 62 (1981) 172–176.
[68] C. Lariviere, D. Gagnon, P. Loisel, The comparison of trunk muscles EMG activation between subjects with and without chronic low back pain during flexion-extension and lateral bending tasks, J. Electromyogr. Kinesiol. 10 (2001) 79–91.
[69] H. LaRocca, Segmental spinal instability. Evolution of a concept, Sem. Spine Surg. 3 (1991) 94–104.
[70] M.L. Latash, J.G. Anson, What are 'normal movements' in atypical populations?, Behav. Brain Sci 19 (1996) 55–106.
[71] S.A. Lavender, W.S. Marras, R.A. Miller, The development of response strategies in preparation for sudden loading to the torso, Spine 18 (1993) 2097–2105.

[72] J.H. Lee, Y. Ooi, K. Nakamura, Measurement of muscle strength of the trunk and the lower-extremities in subjects with history of low-back-pain, Spine 20 (1995) 1994–1996.
[73] V. Leinonen, M. Kankaanpaa, O. Airaksinen, O. Hanninen, Back and hip extensor activities during trunk flexion/extension: effects of low back pain and rehabilitation, Arch. Phys. Med. Rehab. 81 (2000) 32–37.
[74] V. Leinonen, M. Kankaanpaa, M. Luukkonen, O. Hanninen, O. Airaksinen, S. Taimela, Disc herniation-related back pain impairs feed-forward control of paraspinal muscles, Spine 26 (2001) E367–E372.
[75] P. Lisinski, Surface EMG in chronic low back pain, Eur. Spine J. 9 (2000) 559–562.
[76] M. Ljubisavljevic, K. Jovanovic, R. Anastasivic, Changes in discharge rate of fusimotor neurons provoked by fatiguing contractions of cat triceps surae muscles, J. Physiol. Lond. 445 (1992) 499–513.
[77] M.P. de Looze, M.C. Boeken-Kruger, S. Steenhuizen, C.T.M. Baten, I. Kingma, J.H. van Dieën, Trunk muscle activation and low back loading in lifting in the absence of load knowledge, Ergonomics 43 (2000) 333–344.
[78] J.P. Lund, R. Donga, C.G. Widmer, C.S. Stohler, The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity, Can. J. Physiol. Pharmacol. 69 (1991) 683–694.
[79] S. Luoto, M.H. Heliovaara, H. Hurri, H.S. Alaranta, tatic back endurance and the risk of low-back-pain, Clin. Biomech. 10 (1995) 323–324.
[80] S. Luoto, S. Taimela, H. Hurri, H. Aalto, I. Pyykko, H. Alaranta, Psychomotor speed and postural control in chronic low-back-pain patients—a controlled follow-up-study, Spine 21 (1996) 2621–2627.
[81] M.L. Magnusson, A. Aleksiev, D.G. Wilder, M.H. Pope, K. Spratt, S.H. Lee, V.K. Goel, J.N. Weinstein, Unexpected load and asymmetric posture as etiologic factors in low back pain, Eur. Spine J. 5 (1996) 23–35.
[82] M.L. Magnusson, A.R. Aleksiev, D.G. Wilder, M.H. Pope, K.F. Spratt, S.H. Lee, V.K. Goel, J.N. Weinstein, Unexpected load and asymmetric posture as etiologic factors in low back pain, Eur. Spine J. 5 (1996) 23–35.
[83] A.F. Mannion, Fibre type characteristics and function of the human paraspinal muscles: normal values and changes in association with low back pain, J. Electromyogr. Kinesiol. 9 (1999) 363–377.
[84] W.S. Marras, S.A. Ferguson, P. Gupta, S. Bose, M. Parnianpour, J.Y. Kim, R.R. Crowell, The quantification of low back disorder using motion measures. Methodology and validation, Spine 24 (1999) 2091–2100.
[85] W.S. Marras, M. Parnianpour, S.A. Ferguson, J.Y. Kim, R.R. Crowell, S. Bose, S.R. Simon, The classification of anatomic- and symptom-based low back disorders using motion measure models, Spine 20 (1995) 2531–2546.
[86] S.E. Mathiassen, Burdorf A. and van der Beek A.J. Statistical power and measurement allocation in ergonomic intervention studies assessing upper trapezius EMG amplitude. A case study of assembly work, J. Electromyogr. Kinesiol. 12 (2002) 45–57.
[87] D.A. Matre, T. Sinkjaer, S. Knardahl, J.B. Andersen, L. Arendt-Nielsen, The influence of experimental muscle pain on the human soleus stretch reflex during sitting and walking, Clin. Neurophysiol. 110 (1999) 2033–2043.
[88] D.A. Matre, T. Sinkjaer, P. Svensson, L. Arendt-Nielsen, Experimental muscle pain increases the human stretch reflex, Pain 75 (1998) 331–339.
[89] T.G. Mayer, G. Kondraske, V. Mooney, T.W. Carmichael, R. Butsch, Lumbar myoelectric spectral analysis for endurance assessment: a comparison of normals with deconditioned patients, Spine 14 (1989) 986–991.
[90] T.G. Mayer, S.S. Smith, J. Keeley, V. Mooney, Quantification of lumbar function; part 2. Sagittal plane trunk strength in chronic low-back pain patients, Spine 10 (1985) 765–772.
[91] S. Mense, Nociception from skeletal muscle in relation to clinical muscle pain, Pain 54 (1993) 241–289.
[92] S. Mense, P. Skeppar, Discharge behaviour of feline gamma-motoneurons following induction of an artificial myositis, Pain 46 (1991) 201–210.
[93] D.J. Miller, Comparison of electromyographic activity in the lumbar paraspinal muscles of subjects with and without chronic low back pain, Phys. Ther. 65 (1985) 1347–1354.
[94] J.K. Ng, C.A. Richardson, M. Parnianpour, V. Kippers, EMG activity of trunk muscles and torque output during isometric axial rotation exertion: a comparison between back pain patients and matched controls, J. Orthop. Res. 20 (2002) 112–121.
[95] T. Nicolaisen, K. Jørgensen, Trunk strength, back muscle endurance and low back trouble, Scand. J. Rehab. Med. 17 (1985) 121–127.
[96] A. Nouwen, P.F. Van Akkerveeken, J.M. Versloot, Patterns of muscular activity during movement in patients with chronic low-back pain, Spine 12 (1987) 777–782.
[97] P. O'Sullivan, L. Twomey, G. Allison, J. Sinclair, K. Miller, Altered patterns of abdominal muscle activation in patients with chronic low back pain, Aust. J. Physiother. 43 (1997) 91–98.
[98] M.M. Panjabi, The stabilizing system of the spine. Part I. Function, dysfunction, and enhancement, J. Spinal Disord. 5 (1992) 383–389.
[99] M.M. Panjabi, The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis, J. Spinal Disord. 5 (1992) 390–397.
[99a] M.M. Panjabi, Clinical spinal instability and low back pain. J. Electromyogr. Kinesiol. 13 (2003).
[100] N. Paquet, F. Malouin, C.L. Richards, Hip-spine movement interaction and muscle activation patterns during sagittal trunk movements in low back pain patients, Spine 19 (1994) 596–603.
[101] H.E. Pedersen, C.F.J. Blunck, E. Gardner, The anatomy of lumbosacral posterior rami and meningeal branches of spinal nerves (sinu-vertebral nerves). With an experimental study of their functions, J. Bone Joint Surg. A 38 (1956) 377–391.
[102] J. Pedersen, M. Ljubisavljevic, M. Bergenheim, H. Johansson, Alterations in information transmission in ensembles of primary muscle spindle afferents after muscle fatigue in heteronymous muscle, Neuroscience 84 (1998) 953–959.
[103] J. Pedersen, P. Sjolander, B.I. Wenngren, H. Johansson, Increased intramuscular concentration of bradykinin increases the static fusimotor drive to muscle spindles in neck muscles of the cat, Pain 70 (1997) 83–91.
[104] M.H. Pope, J.E. Novotny, Spinal biomechanics, J. Biomed. Engng 115 (1993) 509–574.
[105] I. Posner, A.A. White, W.T. Edwards, W.C. Hayes, A biomechanical analysis of the clinical stability of the lumbar and lumbosacral spine, Spine 7 (1982) 374–389.
[106] A. Radebold, J. Cholewicki, M.M. Panjabi, T.C. Patel, Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain, Spine 25 (2000) 947–954.
[107] A. Radebold, J. Cholewicki, G.K. Polzhofer, H.S. Greene, Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain, Spine 26 (2001) 724–730.
[108] S. Roberts, S.M. Eisenstein, J. Menage, E.H. Evans, K. Ashton, Mechanoreceptors in intervertebral discs. Morphology, distribution, and neuropeptides, Spine 20 (1995) 2645–2651.
[109] M.E. Robinson, J.E. Cassisi, P.D. O'Connor, M. MacMillan, Lumbar iEMG during isotonic exercise: chronic low back pain patients versus controls, J. Spinal Disord. 5 (1992) 8–15.
[110] M.O. Roland, A critical review of the evidence for a pain-

spasm-pain cycle in spinal disorders, Clin. Biomech. 1 (1986) 102–109.
[111] S.H. Roy, C.J.D. Luca, D.A. Casavant, Lumbar muscle fatigue and chronic lower back pain, Spine 14 (1989) 992.
[112] T.A. Schmidt, H.S. An, T.H. Lim, B.H. Nowicki, V.M. Haughton, The stiffness of lumbar spinal motion segments with a high-intensity zone in the anulus fibrosus, Spine 23 (1998) 2167–2173.
[113] G. Schwartz, J.P. Lund, Modification of rhythmical jaw movements by noxious pressure applied to the periosteum of the zygoma in decerebrate rabbits, Pain 63 (1995) 153–161.
[114] W.O. Shaffer, J. Weinstein, Segmental spinal instability. A survey of measurement techniques, Sem. Spine Surg. 3 (1991) 124–129.
[115] R.A. Sherman, Relationships between strength of low back muscle contraction and reported intensity of chronic low back pain, Am. J. Phys. Med. 64 (1985) 190–200.
[116] T. Sihvonen, J. Partanen, O. Hanninen, S. Soimakallio, Electric behavior of low back muscles during lumbar pelvic rhythm in low back pain patients and healthy controls, Arch. Phys. Med. Rehab. 72 (1991) 1080–1087.
[117] P. Sjölander, H. Johansson, M. Djupsjöbacka, The sensory function of ligaments, J. Electromyogr. Kinesiol. 12 (2002) 167–176.
[118] M. Solomonow, B.H. Zhou, R.V. Baratta, Y. Lu, M. Harris, Biomechanics of increased exposure to lumbar injury caused by cyclic loading: Part 1. Loss of reflexive muscular stabilization, Spine 24 (1999) 2426–2434.
[119] M. Solomonow, B.H. Zhou, M. Harris, Y. Lu, R.V. Baratta, The ligamento-muscular stabilizing system of the spine, Spine 23 (1998) 2552–2562.
[119a] M. Solomonow, R.V. Baratta, B.-H. Zhou, E. Burger, A. Zieske, A. Gedalia, Muscular dysfunction elicited by creep of lumbar viscoelastic tissues, J. Electromyogr. Kinesiol. 13 (2003).
[120] C.S. Stohler, J.P. Lund, Effects of noxious stimulation of the jaw muscles on the sensory experience of human volunteers, in: C.S. Stohler, J.P. Lund (Eds.), Biological and psychological aspects of orofacial pain, Center for Human Growth and Development, University of Michigan, Ann Arbor, MI, 1994, pp. 55–73.
[121] C.S. Stohler, X. Zhang, J.P. Lund, The effect of experimental jaw muscle pain on postural muscle activity, Pain 66 (1996) 215–221.
[122] I.A.F. Stokes, M. Gardner-Morse, S.M. Henry, G.J. Badger, Decrease in trunk muscular response to perturbation with preactivation of lumbar spinal musculature, Spine 25 (2000) 1957–1964.
[123] M. Stubbs, M. Harris, M. Solomonow, B. Zhou, Y. Lu, R.V. Baratta, Ligamento-muscular protective reflex in the lumbar spine of the feline, J. Electromyogr. Kinesiol. 8 (1998) 197–204.
[124] P. Svensson, T. Graven-Nielsen, D. Matre, L. Arendt-Nielsen, Experimental muscle pain does not cause long-lasting increases in resting electromyographic activity, Muscle Nerve 21 (1998) 1382–1389.
[125] P. Svensson, G.M. Macaluso, A. De Laat, K. Wang, Effects of local and remote muscle pain on human jaw reflexes evoked by fast stretches at different clenching levels, Exp. Brain Res. 139 (2001) 495–502.
[126] S. Taimela, K. Harkapaa, Strength, mobility, their changes, and pain reduction in active functional restoration for chronic low-back disorders, J. Spinal Disord. 9 (1996) 306–312.
[127] R. Takemasa, H. Yamamoto, T. Tani, Trunk muscle strength in and effect of trunk muscle exercises for patients with chronic low-back-pain—the differences in patients with and without organic lumbar lesions, Spine 20 (1995) 2522–2530.
[128] H.M. Toussaint, M.N. Faber, Y.M. Michies, Commissaris D.A.C.M. and van Dieën J.H. Scaling anticipatory postural adjustments dependent on confidence of load estimation in a bimanual whole body lifting task, Exp. Brain Res. 120 (1998) 85–94.
[129] J. Travell, S. Rinzter, M. Herman, Pain and disability of the shoulder and arm, JAMA 120 (1942) 417–422.
[130] J.J. Triano, M. Luttges, Myoelectric paraspinal response to spinal loads: potential for monitoring low back pain, J. Manip. Physiol. Therap. 8 (1985) 17–145.
[131] J.J. Triano, A.B. Schultz, Correlation of objective measure of trunk motion and muscle function with low-back disability ratings, Spine 12 (1987) 561–565.
[132] D.C. Turk, H. Flor, Etiological theories and treatments for chronic back pain II. Psychological models and interventions, Pain 19 (1984) 209–233.
[133] J.W.S. Vlaeyen, A.M.J. Kole-snijders, R.G.B. Boeren, H. van Eek, Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance, Pain 62 (1995) 363–372.
[134] P.D. Wall, C.J. Woolf, Muscle but not cutaneus C-afferent input produces prolonged increases in the excitability of the flexion reflex in the rat, J. Physiol. 356 (1984) 443–458.
[135] P.J. Watson, C.K. Booker, C.J. Main, A.C.N. Chen, Surface electromyography in the identification of chronic low-back-pain patients—the development of the flexion relaxation ratio, Clin. Biomech. 12 (1997) 165–171.
[136] D.G. Wilder, M.H. Pope, J.W. Frymoyer, The biomechanics of lumbar disc herniation and the effect of overload and instability, J. Spinal Disord. 1 (1988) 16–32.
[137] M. Williams, M. Solomonow, B.H. Zhou, R.V. Baratta, M. Harris, Multifidus spasms elicited by prolonged lumbar flexion, Spine 25 (2000) 2916–2924.
[138] B.D. Wyke, The neurology of back pain, in: M.I.V. Jayson (Ed.), The Lumbar Spine and Back Pain, Churchill Livingstone, Edinburgh, 1987.
[139] H. Yoshizawa, J.P. O'Brien, W.T. Smith, M. Trumper, The neuropathology of intervertebral discs removed for low-back pain, J. Pathol. 132 (1980) 95–104.
[140] M. Zattara, S. Bouisset, Posturo-kinetic organisation during the early phase of voluntary upper limb movement. 1 Normal subjects, J. Neurol. Neurosurg. Psychiat. 51 (1988) 956–965.
[141] M. Zedka, A. Prochazka, B. Knight, D. Gillard, M. Gauthier, Voluntary and reflex control of human back muscles during induced pain, J. Physiol. 520 (1999) 591–604.

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