

Influence of chronic back pain on kinematic reactions to unpredictable arm pulls



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ABSTRACT

Background: There is evidence that muscle reflexes are delayed in patients with chronic low back pain in response to perturbations. It is still unrevealed whether these delays accompanied by an altered kinematic or compensated by adaption of other muscle parameters. The aim of this study was to investigate whether chronic low back pain patients show an altered kinematic reaction and if such data are reliable for the classification of chronic low back pain.

Method: In an experiment involving 30 females, sudden lateral perturbations were applied to the arm of a subject in an upright, standing position. Kinematics was used to distinguish between chronic low back pain patients and healthy controls.

Findings: A calculated model of a stepwise discriminant function analysis correctly predicted 100% of patients and 80% of healthy controls. The estimation of the classification error revealed a constant rate for the classification of the healthy controls and a slightly decreased rate for the patients.

Interpretation: Observed reflex delays and identified kinematic differences inside and outside the region of pain during impaired movement indicated that chronic low back pain patients have an altered motor control that is not restricted to the lumbo-pelvic region. This applied paradigm of external perturbations can be used to detect chronic low back pain patients and also persons without chronic low back pain but with an altered motor control. Further investigations are essential to reveal whether healthy persons with changes in motor function have an increased potential to develop chronic back pain.

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1. Introduction

Chronic low back pain (CLBP) is one of the most frequent musculoskeletal ailments with a higher prevalence in developed countries and is linked to an escalating rate of health care services (Friedly et al., 2010). The majority of people experience back pain at least once in their life and 10% to 30% of them develop chronic symptoms (Balagué et al., 2012). The etiology of CLBP is complex and depends on several factors such as psychosocial conditions, anatomy of the body including the spine and system control such as sensorimotor interactions and nociceptive stimulations. The diagnosis is related to anatomical factors only in a few cases (Hicks et al., 2002), while for the majority of the other cases, the etiological factors are ambiguous. Diagnostic methods such as monitoring the response to disturbances may help resolve this ambiguity.

In human movement, different motor tasks require different levels of processing, ranging from controls based on reflexes and

pure mechanical interaction (self-stability) to controls that are processed in the central nervous system (Taylor and Ivry, 2012). Various studies have compared CLBP patients and healthy controls (HC) during either voluntary motions or those movements provoked by external perturbations. For voluntarily controlled motions, it could be shown that patients with CLBP have a reduced range of movement (Marras et al., 1995, 2001; Mayer et al., 1984; Mellin, 1990; Shum et al., 2005). For example, it was shown that the mobility of spine and hip is significantly reduced and that the lumbar spine–hip joint coordination is altered in sit-to-stand and stand-to-sit exercises in back pain subjects (Shum et al., 2005). The reduced range of motion in voluntarily controlled motions might be a result of compensatory responses used to reduce the pain and to protect injured tissues.

In contrast to voluntarily controlled movements, reactions to perturbations are predominantly induced by muscle reflexes (Nashner, 1976). Altered muscular reflex behavior can cause local instabilities or even provoke injuries. In the context of CLBP, delayed trunk muscle reflexes were identified in pain patients during experiments with perturbations tasks (Liebetrau et al., 2013; Magnusson et al., 1996; Radebold et al., 2000, 2001; Wilder et al., 1996). During trunk flexion, extension and

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lateral bending, delayed reflex response latencies of up to 30 ms for the switch-off of agonistic muscles and about 20 ms for the switch-on of antagonistic muscles were observed in the erector spine muscle group in individuals with CLBP (Reeves et al., 2005). In another study involving sudden trunk load, longer reaction times of trunk muscles (8 ms to 20 ms) were identified (Radebold et al., 2000). Delayed muscle responses can be interpreted as a result of tissue damage that disturbs the innervations and results in a modified motor control strategy to stabilize the lumbar spine or it may be a reaction to central pain signals (Radebold et al., 2000; Reeves et al., 2005; Giesecke et al., 2004). To avoid instability of the lumbar spine in face of delayed reflexes other muscle-skeletal parameters (e.g., activation and co-activation, reflex gain and amplitude) have to be adapted. A mismatch of these parameters due to poor proprioception (Magnusson et al., 1996; Radebold et al., 2000; Wilder et al., 1996; Hodges and Richardson, 1996) are a major cause of CLBP.

The goal of this study was to investigate whether the kinematic reaction to unpredictable perturbations can be used as an accurate objective method to distinguish CLBP from pain-free control subjects in addition to the observed reflex delays in patients with CLBP (Liebetrau et al., 2013).

2. Methods

We investigated how chronic low back pain (CLBP) influences the kinematic reaction to specific mechanical disturbances. The disturbances were provoked by a servomotor and applied on the hands of the standing subjects (Fig. 1A).

2.1. Subjects

Thirty female subjects participated in this study—twenty CLBP patients, which were separated into two groups (CLBP I and CLBP II), and ten HC subjects. An appropriate HC could be assigned to each member of CLBP I (matched according to age, weight, stand width, hip width,

Table 1

Subject characteristics by group mean (standard deviation). No matching could be achieved for CLBP II members with respect to both HC and CLBP I for the parameters age, weight and hip width (significant differences indicated by * $P < 0.05$; N —number of participants).

Group	HC	CLBP I	CLBP II
N	10	10	10
Age (years)	39.7 (14)	40.6 (11.6)	51.2 (2.8)*
Weight (kg)	61.7 (7.8)	62.3 (6.8)	75.9 (11.1)*
Stand width (mm)	254 (68)	248 (30)	229 (24)
Hip width (mm)	381 (16)	375 (21)	412 (27)*
Shoulder height (mm)	1333 (40)	1335 (44)	1364 (97)
Shoulder width (mm)	336 (28)	338 (17)	335 (29)

shoulder height and shoulder width, see Table 1). However, no control person could be assigned to the other patients (CLBP II).

Participants in the pain groups (CLBP I and CLBP II) had been seen by specialists (radiologist and pathophysiological) and were only selected if they suffered from low back pain for a minimum duration of 2 years, had not taken any analgesic medications before the experiment (for at least 48 hours), had no spinal alignment or disc pathology and did not have any symptoms of nerve root problems (pain radiating to leg, numbness and/or paresthesia). Furthermore, CLBP patients reported their current level of low back pain on a visual analog scale ranked from “no pain” (0) to “maximum pain” (10). Subjects with CLBP indicated a pain intensity of 2.9 (standard deviation = 2.1) at the beginning of the experiment and 3.5 (standard deviation = 1.4) over the last 4 weeks. All patients with CLBP were administered in an abbreviated version of the health survey questionnaire (SF-36), a compilation of 149 health status questions (Table 2; Tarlov et al., 1989). For the healthy controls (HC), a medical anamnesis was obtained. They were eligible if they had not experienced any low back pain during the previous 12 months, had no spinal alignment in the past, did not have any symptoms of nerve root problems and matches with a specific person in the CLBP I group.

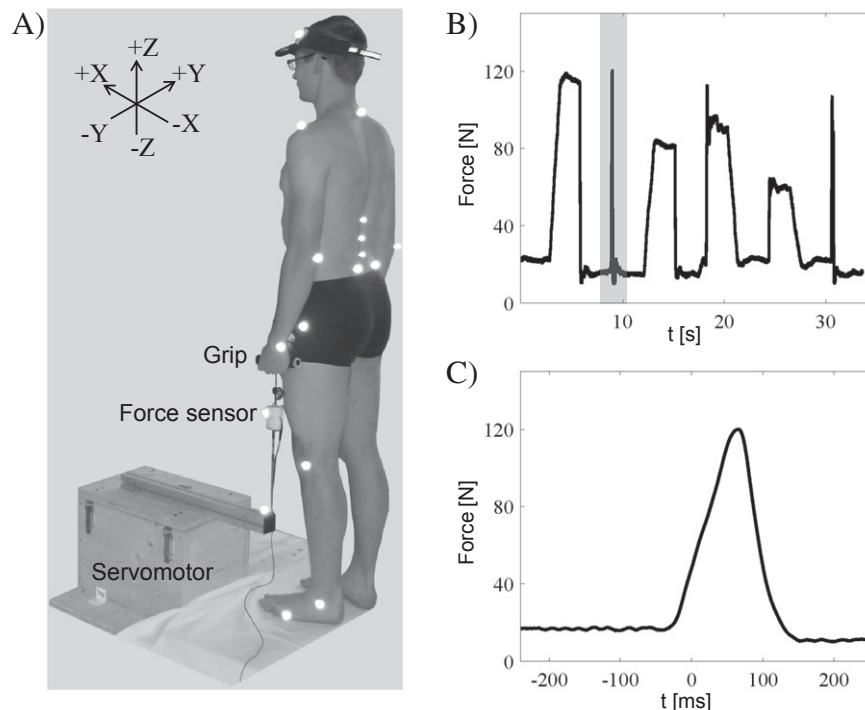


Fig. 1. Illustration of the setup and forces. (A) The setup (servomotor, force sensor and grip) and the marker setup on subjects. X represented the anteroposterior direction, Y the mediolateral and Z the vertical direction. (B) The force sensor signal shows the 6 different perturbations (4 ramps like shapes and 2 short pulses of about 100 ms and 200 ms each) for a typical trial lasting 35 s. For every single trial, the sequence was randomized by the computer control. (C) Here the response to the impact perturbation (100 ms short pulse) was extracted and analyzed for further investigation. The fine vertical line marks the end of the initial loading phase.

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