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Changes in coordination of postural control during dynamic stance in chronic low back pain patients

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Abstract

The human postural system operates on the basis of integrated information from three independent sources: vestibular, visual and somatosensory. It is conceivable that a derangement of any of these systems will influence the overall output of the postural system. The peripheral proprioceptive system or the central processing of proprioceptive information may be altered in chronic low back pain (CLBP). We therefore investigated whether patients with CLBP exhibited an altered postural control during quiet standing. Dynamic posturography was performed by 12 CLBP patients and 12 age-matched controls. Subject's task was to stand quietly on a computer-controlled movable platform under six sensory conditions that altered the available visual and proprioceptive information. While the control of balance was comparable between the two groups across stabilized support surface conditions (1–3), CLBP patients oscillated much more than controls in the anterior–posterior (AP) direction in platform sway-referenced conditions (4–6). Control experiments ruled out that increased sway was due to pain interference. In CLBP patients, postural stability under challenging conditions is maintained by an increased sway in AP direction. This change in postural strategy may underlie a dysfunction of the peripheral proprioceptive system or the central integration of proprioceptive information.

Keywords: Posturography; Balance; Back pain; Proprioception

1. Introduction

The maintenance and control of balance, whether under static or dynamic conditions, is an essential requirement for physical and daily activities. In humans, the balance-controlling system is believed to be phylogenetically old, and to operate relatively autonomously through the spinal and brainstem reflex networks [1]. However, there is evidence that standing, rather than rely on mechano-reflex mechanisms [2,3], may require activity of higher order structures [4,5]. Therefore, the control of erect posture may be more integrated into the movement control scheme than has been previously considered.

The human postural system operates on the basis of the integrated information from three independent sensory sources: somatosensory, vestibular and visual inputs [6]. This information, which allows to assess the position and motion of the body in space, is constantly reweighted so as to generate the appropriate forces to control and maintain balance in a wide range of situations [6]. It is thus conceivable that a derangement to any of the three sensory systems will influence the overall output of the postural system.

The coordination of postural control may be affected in subjects with chronic low back pain (CLBP) [7–12]. The cause of this disturbance is not known. Specifically, it is not clear whether changes in postural control are related to pain itself and to its stressful nature, so-called ''pain interference'' [9,10]. In humans, discharge from high-threshold nociceptive afferents interacts with spinal motor pathways [13–15] as well as with primary somatosensory and motor

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cortex [16,17]. These complex actions are likely to contribute to adaptive changes in postural control [18,19]. In addition, proprioceptive impairment has been suspected as one of the possible causes for balance impairments in CLBP [20–22]. The majority of studies have focused on the lumbar spine, while little attention has been paid to the possibility that a reduced proprioception from the lower limbs may contribute to an altered control of balance in CLBP patients. In normal adults, postural adjustments during quiet standing are generally achieved using an "ankle strategy" [23], in which the ankle torque maintains the center of force over the base of support. In this strategy, the muscles groups acting at the ankle joint are considered as the main musculature for the control of stability during quiet standing [24]. Patients with CLBP due to spondylolisthesis have been found to have abnormalities of the soleus H-reflex [25,26] which depends on the activation of large-diameter mechano-receptive afferents (group Ia fibres) in the muscle [27]. It is known that changes in Ia input may result in altered proprioception [28] and distortion of sensory maps [29]. In addition, altered processing of non-noxious afferent information from large-diameter afferent fibres has been suspected to contribute to some aspects of pain [30,31].

The purpose of the present experiments was to investigate the possibility that patients with CLBP exhibited an altered postural control during quiet standing with respect to a control population, as assessed by dynamic posturography [32]. This method measures whole body performance during quiet standing and allows to differentiate the contribution of the vestibular, proprioceptive and visual system to balance control. The hypothesis was that changes in postural control associated with CLBP, rather than being caused by pain interference, probably reflect an altered postural strategy underlying a dysfunction of the peripheral proprioceptive system or of the central processing of proprioceptive information.

2. Methods

2.1. Subjects

Twelve CLBP patients participated in this study. Inclusion criteria were: (1) localized back pain, lasting more than 6 months and radiating no further than the buttock, (2) no previous history of sciatica or other radicular involvement and (3) normal neurological examination. Self-reported disability was assessed with the Oswestry Low Back Pain Disability Questionnaire [33]. None of the patients had any history of vestibular and neurological disease nor of hip/knee/ankle/foot problems. The patients underwent medical and neurological examinations. Clinical data were recorded according to standardized protocols. Special attention was given to symptoms and signs relevant to the dysfunction of the nerve roots (i.e., pain, changes in muscle strength, sensory changes and problems with bladder or bowel function). The duration of pain was registered. When possible, the patients were examined with plain radiographs, computed tomographic scans and magnetic resonance imaging of the lumbosacral spinal tract. The radiographs were read by experienced neuroradiologist who had no clinical information available. The clinical characteristics and radiological findings are summarized in Table 1. Twelve age-matched controls participated in this study. Control subjects had not experienced any low back pain 6 months prior to testing and had no evidence of gait,

Table 1
Clinical and radiological findings

Patient no.	Age (years)	Sex	Height (cm)	Back pain (years)	Leg pain	Oswestry LBPDQ	Sensory symptoms and signs	Motor symptoms and signs	X-rays	CT-scan MRI
1	23	m	190	1	No	1 (2%)	No	No	n.p.	deg L4–L5, deg L5–S1
2	22	m	175	2	No	12 (24%)	No	No	n.p.	n.p.
3	31	m	175	2	No	5 (10%)	No	No	n.p.	Normal
4	40	f	159	10	No	19 (38%)	No	No	deg L5–S1	deg L1–L2, deg L4–L5, deg L5–S1
5	32	f	167	5	No	1 (2%)	No	No	deg L5–S1	deg L4–L5, deg L5–S1
6	29	m	184	8	No	2 (4%)	No	No	n.p.	deg L2–L3, deg L5–S1
7	25	m	188	9	No	4 (8%)	No	No	Dorsal scoliosis listhesisL5	deg L5–S1
8	57	m	178	10	No	6 (12%)	No	No	Spondylosis multiple deg	Multiple disc protrusions
9	36	f	165	5	No	24 (48%)	No	No	n.p.	deg L4–L5, deg L5–S1
10	31	f	170	1.5	No	7 (14%)	No	No	n.p.	n.p.
11	38	f	172	5	No	6 (12%)	No	No	n.p.	n.p.
12	61	m	176	4	No	7 (14%)	No	No	deg L1–L2	n.p.

LBFDQ: Low Back Pain Disability Questionnaire; deg: degenerative; L: lumbar; S: sacral; n.p.: not performed.