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Original article

Impaired trunk muscle function in sub-acute neck pain: etiologic in the subsequent development of low back pain?

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Abstract

Low back pain (LBP) and neck pain are associated with dysfunction of the trunk and neck muscles, respectively, and may involve common or similar mechanisms. In both cases, dysfunction may compromise spinal control. Anecdotally, neck pain patients commonly develop LBP. This study investigated the possibility that trunk muscle function is compromised in neck pain patients and that compromised trunk muscle function is associated with increased risk of LBP. Fifty-four neck pain patients and 52 controls were assessed on an abdominal drawing-in task (ADIT) and on self-report tests. Performance on the ADIT was able to detect neck pain patients with 85% sensitivity and 73% specificity. Catastrophizing and McGill pain questionnaire (affective) scores were higher in patients with an abnormal task response than in patients with an uncertain or normal response, although the self-report data did not predict task performance. Fifty subjects from each group were contactable by telephone at 2 years. They were asked whether they had experienced persistent or recurrent LBP since the assessment. Subjects (patients and controls) who obtained an abnormal response on the ADIT were 3 to 6 times more likely to develop persistent or recurrent LBP than those who obtained an uncertain or normal response. ADIT performance was the main predictor of development of LBP in patients. The results suggest that reduced voluntary trunk muscle control in neck pain patients is associated with an increased risk of developing LBP.

1. Introduction

Postural activation of the deepest abdominal muscle, transverses abdominis (TrA), is altered in people with chronic recurrent low back pain (LBP) (Hodges and Richardson, 1996) and in healthy subjects given experimentally induced acute LBP (Hodges, 1999; Moseley et al., 2003). Those findings suggest that LBP may cause TrA dysfunction. The converse, that TrA dysfunction causes LBP, may also be true; extensive data now exist that show that TrA makes an important contribution to stiffness between vertebral segments (Hodges et al., 2001) and that postural activity of this muscle is consistent with such a contribution (Hodges and Richardson, 1999). Furthermore, normal control of TrA is lost in people with chronic recurrent LBP, even when they are pain free at the time (Hodges, 2001).

TrA activity is decreased and/or delayed during acute experimentally induced LBP and this is accompanied by an increase and/or augmentation of at least one superficial abdominal muscle (Hodges et al., 2003; Moseley et al., 2003). This pattern of activity suggests that, during spinal pain, the central nervous system (CNS) effectively splints the trunk to limit amplitude and velocity of movement. This would be consistent with the pain-adaptation model (Lund et al., 1991), which proposes that the motor response to pain depends on the task at hand, such that the agonist and antagonist are inhibited and facilitated, respectively. Although general spinal splinting may be beneficial in the short term, there may be an associated cost. For instance, splinting reduces spinal flexibility, which is probably important for normal function and dampening of reactive forces (Hodges et al., 1999). Splinting may also result in increased loading of the spine and stimulation of nociceptors in spinal structures (Gardner-Morse and Stokes, 1998) and these mechanisms may be etiologic in recurrence and/or chronicity.

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One clinical area to which these findings are relevant is that of chronic neck pain. Recent findings that suggest similar mechanisms may underlie neck muscle dysfunction in neck pain and trunk muscle dysfunction in LBP (Jull et al., 1999; Sterling et al., 2001) raise the possibility that there may be a common effect. Perhaps spinal pain imparts similar effects regardless of the level of the spine at which the pain is experienced. If so, trunk muscle dysfunction associated with neck pain may be etiologic in the development of LBP. Alternatively and in contrast, perhaps a generic problem in the motor system leads to symptoms in the neck and low back. Anecdotally, insidious onset of LBP is common in people with chronic neck pain.

Assessment of postural control of TrA requires intramuscular electromyography (EMG), which is costly and invasive. An alternative method, albeit less accurate, is assessment of voluntary drawing-in of the lower abdominal wall. This method, the so-called abdominal drawing-in task (ADIT), is an established clinical assessment tool. Performance on the task has been linked to postural activity during limb movements using intramuscular EMG and is able to effectively discriminate people according to whether or not they have LBP (Hodges et al., 1996).

The current work aimed to test two hypotheses. First, that people with sub-acute neck pain perform badly on the ADIT and second, that reduced performance on the ADIT is associated with increased risk of LBP in the following 2 years.

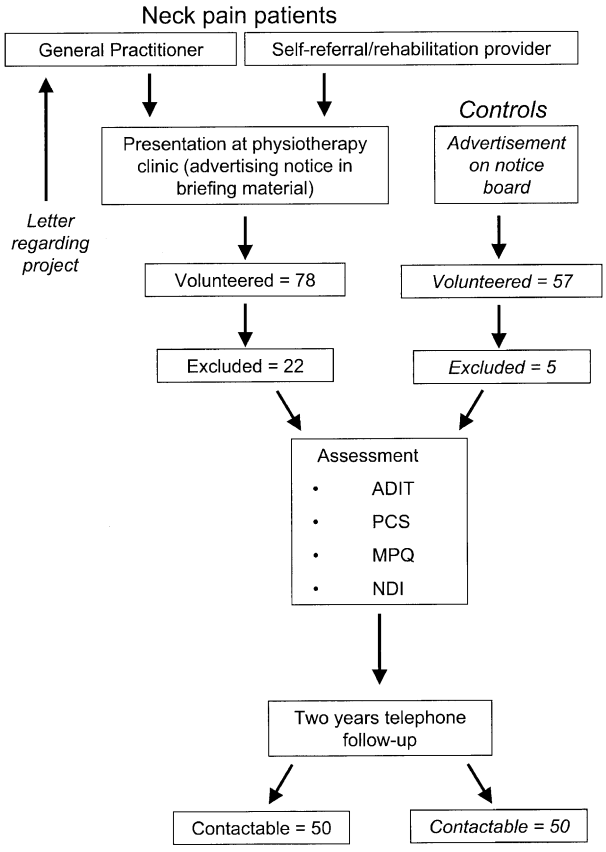

Fig. 1. Subject recruitment and experimental plan.

2. Methods

2.1. Subjects

Seventy-eight patients and 57 age-matched controls, mean ± SD age, height and weight 30 ± 6 years, 173 ± 14 cm and 74 ± 7 kg, respectively, participated in the study. Subject recruitment and experimental plan is shown in Fig. 1. Volunteer patients were drawn from those who presented for physiotherapy for neck pain of more than 4 months and not more than 1 year duration, with or without radiating shoulder and/or arm pain. Subjects were excluded if they had experienced LBP within the last 2 years, had participated in a trunk muscle training program other than their normal gymnasium or fitness work, had undergone abdominal or spinal surgery in the last 2 years, or were referred for medical opinion and/or pain management. Twenty-two subjects were excluded. Volunteer control participants were obtained via advertisements on notice boards and direct approach. Control participants were excluded from the study if they reported chronic pain, current neck pain, a history of neck pain in the last 4 years, or met any of the other exclusion criteria listed above. Five subjects were excluded. Informed consent was obtained and all procedures were approved by the institutional ethics committee and conformed with the Declaration of Helskinki.

2.2. Experimental procedure

ADIT: prior to testing, each subject was instructed in the performance of the ADIT in four-point kneeling, according to the guidelines recommended by Richardson et al. (1999). Any substitution strategies that were identified were corrected. Seven subjects were unable to adopt this position, so an adjustable support was placed under their forehead. It is not known whether this may have affected their performance.

The testing procedure was the same as that described by Hodges et al. (1996). The subject lay prone, with an air filled non-elastic pressure bag placed under the lower abdomen. The pressure bag was connected to a sphygmamanometer gauge (Stabilizer, Chattanooga®) and the test score was assessed as the change in pressure when the subject attempted the ADIT. The investigator was blind to the category of the subject and strictly followed the testing process. Prior to the subject entering the room, the pressure bag was placed deflated in the middle of a treatment plinth, and a small pillow was

placed at the foot of the plinth. The subject was given the following instruction; ''Lie face down on the bed, with the blue bag underneath the lower portion of your abdomen, your legs straight and your shins on the pillow, and your head resting comfortably in the hole. Please ensure that your head and neck are comfortable''. The position of the bag was adjusted if necessary, and then the bag was pumped to a pressure of 70 mmHg. The subject was then given the following instruction ''Slowly draw your abdomen off the pressure bag, hold it for 5 s and then slowly relax''. The subject was given 4 practice trials, and then allowed to rest for 30 s. The pressure was then readjusted to 70 mmHg, and the task was performed again, this time for 10 s. The maximum pressure change sustained for more than 2 s was converted to a rating based on Hodges et al. (1996) in which $\geqslant$4 mmHg was considered a normal response, 2–4 mmHg uncertain, and $<$4 mmHg an abnormal response.

This testing strategy is reliable (intratester ICC (95% CI) = 0.91 (0.71–0.99)) (Moseley, 2001).

2.3. Self-report data

The neck disability index (NDI) (Vernon and Mior, 1991) is a 10-item self-report questionnaire, which assesses to what extent the patient feels they are affected in daily activities by their neck pain. NDI has satisfactory internal validity and reliability (Vernon and Mior, 1991).

The McGill pain questionnaire (MPQ) (Melzack, 1975) is a widely used clinical tool, has high validity and is sufficiently sensitive to change in the majority of pain populations (Melzack, 1975). The subfactors of the MPQ provide information about the sensory, affective, evaluative and miscellaneous aspects of pain, and a rating of present pain intensity.

The pain catastrophizing scale (PCS) (Sullivan et al., 1995) is a self-report questionnaire that assesses catastrophic thinking about pain and injury. Thirteen statements such as ''I feel I can't go on'' are rated according to the degree to which the subject has that particular thought or feeling when they are experiencing pain. The PCS has strong construct validity and is a reliable and stable measurement tool (Sullivan et al., 1995).

2.4. Treatment delivered

Neck pain patients were treated at the discretion of the treating physiotherapist. No limits were placed on the type, frequency or duration of treatment. Informal assessment indicated that treatment would typically involve manual therapy and electrophysical modalities.

2.5. Two-year telephone follow-up

Participants were contacted via telephone $\sim$2 years after the initial assessment. Patients were asked ''Do you still have neck pain?'' Pilot studies showed that this item was likely to detect those patients for whom their neck pain had not completely resolved, even if they were pain-free at the time of testing (GL Moseley, Unpublished data). Patients and controls were asked ''Have you experienced recurrent (more than 4 episodes) or persistent LBP in the last 2 years?''

2.6. Statistical analysis

The following statistical analyses were performed in Statistica 5.1 (Statsoft® Tulsa, USA): (i) A one-way MANOVA was used to compare the unconverted pressure change score on the ADIT and the self-report data (MPQ, sensory, affective, evaluative, miscellaneous, present pain intensity, subfactors of the MPQ, PCS; dependent variables, DV) between patients and controls (independent variable, IV). (ii) A one-way MANOVA was used to compare the MPQ, the subfactors of the MPQ and the PCS (DV) according to categorized ADIT score (IV). (iii) A logistic regression analysis was used to investigate the relationship between ADIT category (DV) and the self-report data (IV). (iv) A separate logistic regression analysis was used to determine, the relationship between the presence of chronic or recurrent LBP at 2 years (DV) and the ADIT category and self-report data (IV). (v) Fisher's exact tests were used to determine if there was an increased likelihood of developing LBP if subjects had an abnormal ADIT score.

Thus, five separate statistical tests were conducted, which elevated the probability of a type I error. A Bonferroni correction would elevate the probability of a type II error and set $\alpha = \sim 0.01$, which was considered to be too conservative. Because the current work was exploratory in nature, and in light of criticism in the literature of Bonferroni and other corrections, e.g. Perneger (1998), it was considered appropriate to maintain $\alpha = 0.05$.

3. Results

Subject characteristics are presented in Table 1. There were no differences between groups in demographic details.

The mean score on the ADIT was lower for patients than for controls (1.8 mmHg $\pm$ 1.5 mmHg and 5.3 mmHg $\pm$ 1.8 mmHg for the patients and controls, respectively, $P < 0.01$). Based on categorized ADIT data, 75% of patients (40/54) and 15% of controls (8/52) obtained an abnormal response and 15% of patients

(8/54) and 53% of controls (28/52) obtained a normal response (Fig. 2A). Thus, performance on the ADIT was able to detect the patients with 83% specificity and 75% sensitivity. PCS and MPQa were higher in the abnormal ADIT group than the other two groups ($P<0.04$ for both), but there were no other differences in self-report data between groups (Fig. 2B). The logistic regression with the ADIT category as the dependent variable and the self-report data as independent variables revealed no relationship ($P=0.83$).

Fifty subjects in each group (93% and 96% for patients and controls, respectively) were contactable at 2 years. The sub-factors of the MPQ were removed from the logistic regression as they demonstrated multicolinearity with the MPQ total score (tolerance <0.05 for all). ADIT category and self-report data were predictive of LBP at 2 years ($P=0.02$) (Table 2).

Eighty per cent (40/50) of patients indicated that they still had neck pain at 2 years. Seventy-four per cent (23/31) of patients who obtained an abnormal response on the ADIT reported persistent or recurrent LBP at 2 years, which was ~3 times that for the uncertain (25% 3/12) and normal (14% 1/7) response groups, respectively ($P<0.03$ for both). Of the control subjects who had an abnormal response on the ADIT, (75% 6/8) went on to develop persistent or recurrent LBP in the following 2 years, which was ~6 times that for the

Table 1
Demographic and self-report data for patients and control subjects

	Neck pain patients ($n=54$)	Control subjects ($n=52$)
Age (yr)	33±7	30±7
Height	172±10 cm	170±8 cm
Weight	75±5 kg	70±6 cm
Female	59%	64%
Working full-time	46%	100%
Working part-time	34%	0%
Neck disability index	35±7	—
McGill pain questionnaire		
Total	32.6±8.0	—
Sensory	12.1±6.3	—
Affective	8.0±4.0	—
Evaluative	4.1±1.3	—
Miscellaneous	8.3±4.1	—
Present pain intensity	3.5±1.0	—
Duration of neck pain (months)	7±3	—
Currently receiving compensation	47%	0%
Non-English speaking background	9%	18%

Table 2
Summary of the logistic regression for patients, with low back pain at 2 years as the dependent variable and pain catastrophizing scale (PCS), McGill pain questionnaire total score (MPQT), neck disability index (NDI) and ADIT category as independent variables

Final loss: 31.608; $\chi^2(4)=11.569$; $P=0.020$

	Const.B0	PCS	MPQT	NDI	ADIT
Estimate	−1.090	0.126	−0.077	0.006	0.562
Odds ratio (unit ch)	0.336	1.1342	0.926	1.006	1.755
Odds ratio (range)		119.761	0.0228	1.128	29.262

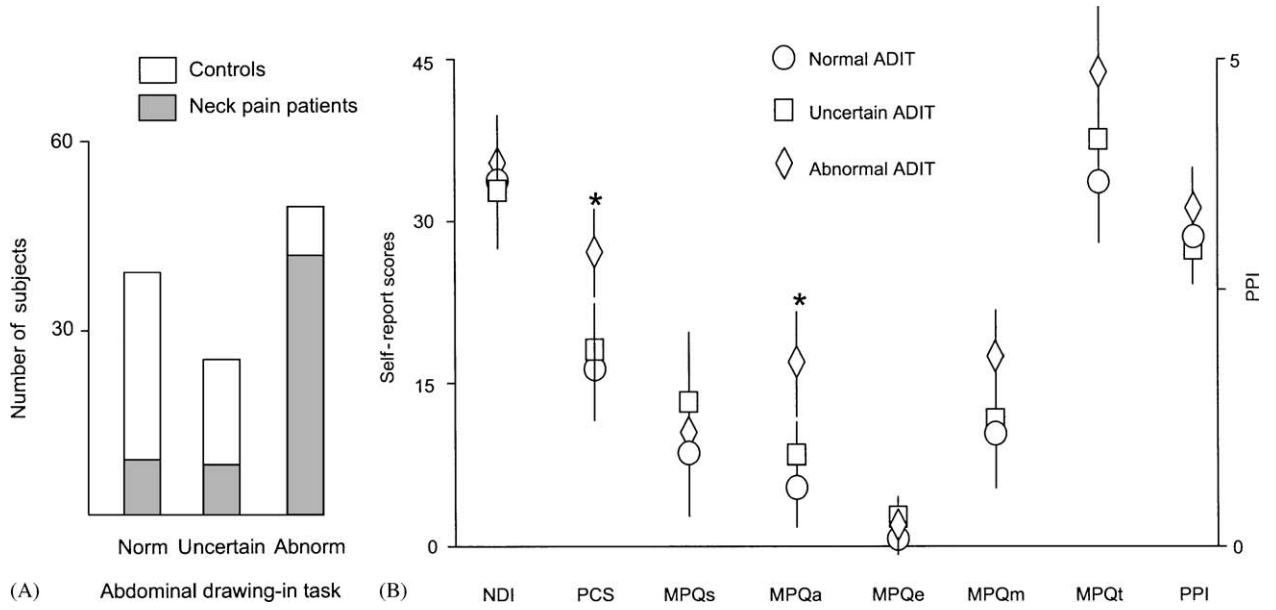

Fig. 2. (A) Distribution of normal, uncertain and abnormal scores on the ADIT. Shaded column denotes patients and open column denotes control subjects. (B) Self report data according to ADIT performance for neck disability index (NDI), Pain catastrophizing scale (PCS), and the subscales of the McGill pain questionnaire (MPQ); sensory, affective, evaluative, miscellaneous and total (left y-axis), and the present pain intensity (PPI) (right y-axis). Mean and SD are shown. Note higher PCS and MPQa scores in the abnormal response groups (asterix denotes Scheffe $P<0.04$).

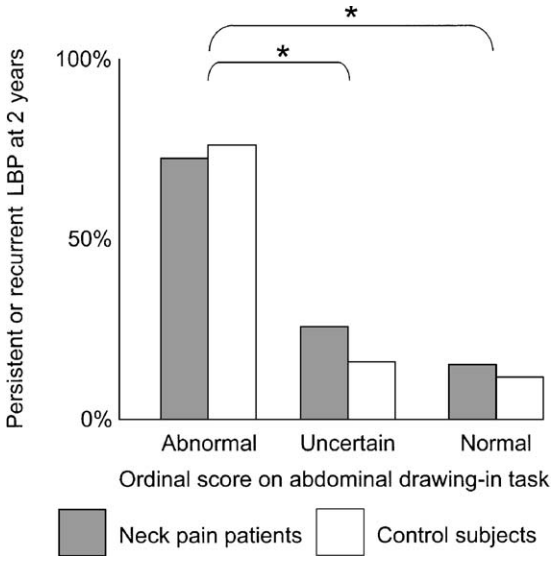

Fig. 3. Percentage of sample with persistent or recurrent (more than 4 episodes) LBP over the following 2 years grouped according to ADIT performance for patients (filled columns) and controls (open columns). Note greater likelihood of LBP in those with an abnormal response for patients and controls (asterix denotes Fisher's exact $P<0.04$).

uncertain (14% 2/14) and normal response 11% (3/28) groups ($P<0.04$ for both) (Fig. 3).

4. Discussion

The first main finding of the current work is that people with sub-acute neck pain have a reduced capacity to perform the ADIT. This is supported by a lower mean score for the ADIT in patients than the controls and a higher proportion of abnormal responses in the patient group than in the control group. The second main finding is that reduced performance is associated with increased risk of LBP over the following 2 years, for both patients and controls. This is supported by a significant relationship between ADIT category and LBP at 2 years and a greater likelihood of LBP in participants (both patients and controls) who obtained an abnormal response on the ADIT than those who obtained an uncertain or normal response.

The pressure change values for the ADIT in the present work, for patients and controls, are similar to results from previous studies in subjects with and without chronic recurrent LBP (Hodges et al., 1996). The current work supports the finding from that study that converting the pressure change score to a rating improves the utility of the test: in the current study the rating had 83% specificity and 75% sensitivity in detecting neck pain patients. These results suggest that performance at this task may be reduced in a similar fashion in both neck pain patients and LBP patients. This is an intriguing finding. It suggests that there may be effects on trunk muscle function that are consistent between neck pain and LBP and therefore raises the possibility that neck muscle function may be compromised in LBP patients. These issues could be clarified with further research.

The current work suggests that the anatomical specificity of pain is not critical in determining its impact on muscle control. This is not consistent with recent work by our group that showed a clear effect of impending LBP but not impending elbow pain on trunk muscle control (Moseley et al., 2002b). However, this discrepancy may be explained by the biomechanical demands of the response to the stimuli involved. That is, because the changes in trunk muscle function associated with pain probably reflect an alternative postural strategy (Moseley and Hodges, 2004), a similar effect may be observed during neck pain because the biomechanical demands associated with neck pain are sufficiently similar to those associated with LBP, whereas those associated with elbow pain are not.

The present work casts new light on the mechanisms that underlie development of LBP in patients with neck pain. However, several questions remain unanswered. For example, is trunk muscle dysfunction present prior to neck pain and, in which case, is trunk muscle dysfunction a risk factor for neck pain? Also, is trunk muscle dysfunction an etiological mechanism, for instance via reduced intersegmental control during functional movements as has been proposed (Hodges, 1999), or is it a benign epiphenomenon of some other mechanism? These questions are currently being investigated by our group. The results also have implications for understanding the development of LBP in the otherwise asymptomatic population: 75% of control subjects who had an abnormal ADIT score went on to experience LBP. Although a link between altered function of the deep trunk muscles and the development of back trouble is clinically well accepted, the present findings provide the first prospective evidence for such a link, at least between performance of this voluntary task, thought to target the deepest abdominal muscle, TrA, and development of LBP. Notably, the current study did not evaluate pain-related beliefs and attitudes in the control subjects, which may also have been predictive. Further research is required to explore these issues in otherwise asymptomatic subjects.

It is not known why some chronic neck pain patients demonstrate trunk muscle dysfunction and others do not. This question has been asked previously within the context of neck muscle dysfunction in chronic neck pain by evaluating trapezius muscle activity at rest and during isometric and dynamic tasks (Nederhand et al., 2001, 2002, 2003). Those studies revealed increased activity in the upper trapezius on the contralateral side and decreased activity on the ipsilateral side to the task and changes were independent of the nature of onset but

dependent on the degree of disability. While the current study found no difference in disability between those who could and couldn't perform the ADIT, it did find higher PCS and MPQa scores in those with an abnormal response on initial assessment. That finding raises the possibility that catastrophic thought processes about pain, or an elevated affective/emotional impact of pain, has a particular impact on performance of the task. In light of those data, it is notable that the psychosocial measures used here did not contribute to the development of LBP at 2 years ($P > 0.07$ for all). Importantly, psychosocial factors have been implicated in chronic pain (Vlaeyen et al., 1995; Sullivan et al., 1998; Turner et al., 2000) and fear of pain and (re)injury has been shown to impact on trunk muscle function during trunk movements (Watson et al., 1997; Vlaeyen et al., 1999) and on postural trunk muscle activity during limb movements (Moseley et al., 2002a). It is possible that the current work was underpowered to detect an effect of psychosocial factors on the development of LBP in this group. Alternatively, the current results may reflect a distinct mechanism underlying recurrence or the development of pain at a secondary anatomical site. Further investigation using a wider battery of tests may be indicated.

The present results should be interpreted in light of several issues. First, although subjects were excluded if they had experienced LBP in the previous 2 years, episodes of LBP prior to that were not evaluated. This criterion was based on findings from Hides et al. (2001), which showed 84% recurrence inside 1 year. However, the exclusion criteria did not remove the possible confounder that performance at the ADIT is associated with the frequency and/or severity of LBP more than 2 years previously. Second, the ADIT is effectively a behavioural test, which requires voluntary effort. People who experience chronic pain are often limited by poor motivation during the performance of voluntary tasks (Luoto et al., 1996). The possibility that this may have undermined the results cannot be excluded, however it is probably unlikely to fully explain them. Importantly reduced motivation on the part of patients would not corrupt the second finding that performance on the ADIT was related to development of LBP, it would just imply that a different task would yield the same result. Third, the items used for telephone follow-up at 2 years may have been limited in their sensitivity to delineate between those with and without LBP. That is, participants may have difficulty in recalling experiences over such a long period. That said, the effect of this limitation would be to falsely categorize those with LBP in the no-LBP group, which would weaken the sensitivity of the protocol. Thus, this limitation does not threaten the main finding of the study. The follow-up question concerning neck pain may also be misleading. Although pilot work suggested that the question used was sensitive to detect those for whom neck pain had not completely resolved, it provides little information about the resolution of the particular episode for which the patient sought attention. This limitation may have underpinned the large proportion of patients (80%) who answered that question to the affirmative. Finally, the ADIT does not assess muscle activity during functional tasks. Although a link has been demonstrated between ADIT rating and postural activity of TrA during limb movements (Hodges et al., 1996), it is possible that people with chronic neck pain demonstrate normal motor control on functional tasks even though they perform badly on the ADIT.

Although several questions remain unanswered, it seems reasonable to suggest that on the basis of the current results, maintenance of normal trunk muscle control should be a goal of therapy in patients with sub-acute neck pain. This preventative strategy may limit the incidence of persistent or recurrent LBP in neck pain patients. A clinical trial would verify this possibility.

5. Conclusion

The current study showed that people with sub-acute neck pain have a reduced capacity to perform the ADIT, and that reduced ADIT performance is associated with increased risk of LBP over the following 2 years. These findings suggest (i) that spinal pain impacts on trunk muscle control regardless of the spinal level at which pain is experienced, (ii) that altered trunk muscle function associated with neck pain may be etiologic in the subsequent development of LBP, and (iii) that altered trunk muscle function in otherwise asymptomatic people may also be etiologic in the subsequent development of LBP. The results imply that maintenance of normal voluntary trunk muscle function may be important in preventing LBP in patients with sub-acute neck pain. Further research is required to evaluate the mechanism and meaning of altered trunk muscle function in neck pain and LBP and to optimize prevention and management strategies.

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