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Ultrasound tissue Doppler imaging reveals no delay in abdominal muscle feed-forward activity during rapid arm movements in patients with chronic low back pain.

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Structured Abstract

Study Design. Cross-sectional study.

Objective. Comparison of the timing of onset of lateral abdominal muscle activity during rapid arm movements in patients with chronic non-specific low back pain (cLBP) and back-pain-free controls.

Summary of Background Data. Rapid movements of the arm are normally associated with prior activation of trunk stabilizing muscles in readiness for the impending postural perturbation. Using invasive intramuscular electromyography techniques, studies have shown that this feed-forward function is delayed in some patients with LBP. Ultrasound tissue Doppler imaging (TDI) provides an ultrasound method for quantifying muscle activation in a non-invasive manner, allowing investigation of larger groups of patients and controls.

Methods. 96 individuals participated (48 patients with cLBP and 48 matched LBP-free controls). During rapid shoulder flexion, abduction and extension, surface electromyographic signals from the deltoid and motion-mode TDI images from the contralateral lateral abdominal muscles were recorded simultaneously. The onset of muscle activity was given by changes in the tissue velocity of the abdominal muscles, as measured with TDI. Pain and disability in the patients were assessed using standardized questionnaires. Data were analyzed using repeated measures ANOVA.

Results. In both groups, feed-forward activity of the lateral abdominal muscles was recorded during arm movements in all directions. The main effect of “group membership” revealed no significant difference between the groups for the earliest onset of abdominal muscle activity (p=0.398). However, a significant “group x body side” interaction (p=0.015) was observed, and this was the result of earlier onsets
in the cLBP group than controls for the abdominal muscles on the right (but not left) body side. No relationship was found between the time of onset of the earliest abdominal muscle activity and pain intensity, pain frequency, pain medication usage or Roland Morris disability scores.

**Conclusion.** Patients with cLBP did not show a delayed onset of feedforward activation of the lateral abdominal muscles during rapid arm movements. Earlier activation was observed for one body side compared with the controls. However, the clinical relevance of this finding remains obscure, especially since there was no relationship between the onset of activation and any clinical parameters.
**Key Points**

- This study sought to compare the timing of activation of the lateral abdominal muscles during rapid arm movements in patients with cLBP and pain-free controls.

- *Both groups showed feed-forward activity* of the lateral abdominal muscles (i.e., onset of activity < 50 ms after activation of the deltoid muscle, the prime mover for the movement).

- The ANOVA revealed a statistically significant (*p*=0.015) “group x body side” interaction, which was the result of earlier onsets in the cLBP group than controls for the abdominal muscles on the right (but not left) body side.

- No relationship was found between the onset of the earliest abdominal muscle activity and pain intensity, pain frequency, pain medication usage or Roland Morris disability scores.

- The clinical relevance of the time of onset of lateral abdominal muscle activity remains obscure.
Mini Abstract

Using non-invasive tissue Doppler imaging, the timing of activation of the lateral abdominal muscles during rapid arm movements was compared in patients with chronic low back pain and controls. Both groups showed feed-forward activation. For left-arm movements, the patients showed earlier activation than the controls. No relationship was found between the onset of activation and pain or disability, questioning the clinical relevance of the findings.
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Introduction

Studies have shown that, in individuals without low back pain (LBP), rapid movements of the arm are associated with activation of M. transversus abdominis (TrA) and obliquus internus (OI) before the arm movement begins and even before the prime mover for the movement (the deltoid muscle (MD)) is activated [1-3]. Further studies indicate that TrA is the first muscle to be activated during the expected and unexpected presentation of loads to the trunk [4]. The early activation of these abdominal muscles is understood to be an attempt to stabilize the spine in readiness for the impending postural perturbation. These responses cannot be reflex mediated, because they occur either before or <50 ms after the activation of the MD; instead, they represent feed-forward activity, pre-programmed by the central nervous system [1, 5, 6]. Using fine-wire intramuscular electromyography (EMG), it has been shown that this mechanism of feed-forward activation is impaired in some patients with periodic LBP [7-10]. This is believed to pose a threat to spinal stability and predispose to continuing/recurring episodes of pain [1, 8-10]. However, in recent years work by other investigators has questioned this interpretation. The fine-wire EMG studies of Mannion et al [11] could not confirm the previous findings that the onset of TrA activity was independent of arm-movement direction (this being the finding that had previously led to a "spine-stabilising role" being conferred upon TrA) [1]; instead, TrA was activated significantly earlier during shoulder flexion than during extension or abduction, in a manner that was entirely consistent with its involvement in (direction-dependent) anticipatory postural adjustments (APA). The authors suggested that their results challenged the concept of a unique role for TrA in stabilisation of the spine. Allison et al. [12] measured TrA activation bilaterally during unilateral arm movements and
showed an asymmetrical response of the muscle of interest: TrA on the contralateral side to the arm movement was activated before TrA on the ipsilateral side. Unilateral arm flexion showed different responses in TrA depending on which arm was used [12]. Since stabilization of the spine by means of TrA activation is dependent on bilateral contraction of the muscle [13, 14], with unilateral TrA activation failing to influence segmental stiffness [14], this further questioned the unique role for TrA in stabilization of the spine.

The seemingly discrepant findings in the aforementioned studies may, in part, be attributable to the fact that the phenomenon has only been investigated in small groups of selected individuals, because fine-wire EMG is invasive and time consuming. The availability of a non-invasive alternative would allow the phenomenon to be investigated in greater depth, and in larger groups of patients.

In the mid-90s, the use of non-invasive methods for assessing skeletal muscle activity, based on Tissue Doppler imaging (TDI), were investigated [15]. TDI is an ultrasound (US) technique that uses modified color Doppler processing to quantify the velocity of tissue motion relative to the transducer [16]. This information provides a sensitive indication of muscle activation, even at very low levels of contraction (e.g. associated with low-level electrical stimulation of muscle, or reflex responses) [15, 17, 18]. The increased sampling rates afforded by more modern US machines has prompted the development of TDI-based techniques that are able to indicate the precise onset of skeletal muscle activity, in a manner previously only possible with electromyography. A recent study comparing intramuscular fine-wire EMG to TDI tissue velocity changes showed that the latter provides a valid and reliable measure of the earliest onset of activity of the lateral abdominal muscle group (comprising TrA, OI and obliquus externus (OE)) during rapid arm
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movements [11], and its use was recommended in larger research studies in the
field of LBP.

Using this new method, the present study sought to compare the timing of the
earliest activation of the lateral abdominal muscles during rapid arm movements in
patients with non-specific chronic LBP (cLBP) and pain-free controls. Additionally,
the correlation between the timing of abdominal muscle activation and various
clinical variables (pain, disability, etc.) was examined.

Materials and Methods

Study participants and protocol

48 patients (17 men) with chronic non-specific LBP and 48 controls, matched in
terms of age, gender and body weight, participated in the study. Their physical
characteristics are shown in Table 1.

The healthy control participants were recruited from the local universities/hospitals;
patients were recruited from the authors’ clinical departments and via
advertisement in the local media. Prior to inclusion, all the patients underwent
clinical assessment and medical history-taking by the study doctor. They also
completed a questionnaire enquiring about the following: average and worst LBP
intensity in the last week (on a 0-10 graphic rating scale), LBP duration (in years),
LBP frequency in the last 6 months (4 categories: never, occasional, frequent,
constant), frequency of pain medication intake in the last 6 months (4 categories:
never, occasional, frequent, constant), and disability in activities of everyday living
(Roland Morris disability questionnaire (RM)) [19, 20]. The inclusion and exclusion
criteria are shown in Table 2 [21].
The study was approved by the cantonal medical ethics committee of Zurich (Kantonale Ethikkomission Zurich). Eligible participants were informed verbally and in writing about the test procedure and gave their signed informed consent to participate.

The test set-up was similar to that described by Hodges et al. [8] (Figure 1) and detailed in Mannion et al [11]. In brief, the participant stood barefoot on a thin rubber mat, with feet approximately shoulder-width apart, upright but relaxed. In response to a computerized visual stimulus (customized software, Schulthess Clinic, Zurich, Switzerland), the participant performed rapid shoulder flexion (up to 60°), abduction (up to 60°) or extension (up to 40°) in randomized order, moving the extended arm as quickly as possible in the direction displayed on the computer screen. 10 arm movements (with a one minute break between each) were performed in each of the three directions, on both right and left body sides. A customized contact switch (Biomechanics Laboratory, Swiss Federal Institute of Technology, Zurich, Switzerland), with one part attached to the wrist and its counter-piece attached to the outer thigh, was used to indicate the start of the arm movement and to time-synchronize the EMG/TDI signals.

Electromyography recordings

Surface EMG (sEMG) signals were recorded from the medial deltoid (MD) (Dantec, Medtronic Functional Diagnostics A/S, Skovlunde, Denmark). After skin preparation (abrasion and cleaning with alcohol; shaving if necessary), pairs of disposable Ag/AgCl bipolar sEMG electrodes (Electrodes ECG Universelles; Contrôle-Graphique S.A., Brie Compte Robert Cedex, France) were placed over the muscle
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with an inter-electrode distance of 2 cm [22]. A reference electrode was placed over
the C7 spinous process. The raw sEMG signals were band-pass filtered (50-500
Hz), amplified, analogue-to-digital converted at a sampling rate of 5000 Hz and
stored on the hard disc of the computer.

Ultrasound recordings

For the US data collection (Philips HDI-5000, Philips Medical Systems, Bothell, WA,
USA) a linear array transducer (L5-12 MHz, 38 mm, SN 01NPTV, Philips Medical
Systems, Bothell, WA, USA) was fixed in a high-density foam supporting block [11].
Under US guidance in B (brightness)-mode the transducer was positioned on the
contralateral side to the arm to be moved during the test, at a point 2.5 cm
anteromedial to the mid-point between the iliac crest and the costal margin on the
mid-axillary line, where the fascial boundaries between TrA, OI and OE and the
inferior edge of the TrA fascia lie parallel (Figure 2A) [23]. A sonar-aid
(130x120x10mm; Alloga AG, Burgdorf, Switzerland) and transmission gel were
placed between the transducer head and the skin to permit good signal
transmission. To minimize relative movement between the transducer and the
abdomen the foam block was fixed to the abdomen with Velcro straps [11].
The data were sampled in M(motion)-mode at the machine’s maximum possible
sampling rate of 333 Hz using TDI. The quality of the recordings was optimized by
adjusting depth, focus and gain for each participant; the remaining scanner settings
were standardized for all subjects alike. On the US machine screen, tissue
velocities were visualized by a TDI color layer and coded as yellow and red for
movement towards the transducer, and blue and green for movement away (Figure
2B). The US-cineloop files containing the gray scale, TDI-velocity, and movement
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switch data were stored on a computer to which the US machine was interfaced using the ResearchLink function of the HDI-5000 system.

Data processing

Full details of the data processing methods are reported in Mannion et al [11]. The data were exported from the US software program HDI-Lab [24] into a customized program written in MATLAB [25]. The area of interest of each abdominal muscle was marked in the gray-scale image and the corresponding muscle velocity versus time data for that region were exported as a text file. The tissue velocity data and the corresponding raw EMG data were then imported into a second customized MATLAB program for manual identification of the muscle activity onsets. Signals were displayed individually. For both TDI-velocity and surface EMG data, the onsets were given by the earliest rise above baseline levels [8]. Each sEMG signal trace was displayed both raw and rectified: the onset was marked in the raw trace, with the rectified signal providing further guidance.

Only trials for which the onset was physiologically tenable, i.e., within −200 and +200 ms of the EMG onset of MD [1, 6, 26] were analysed further.

Blinding

For logistic reasons, the investigators carrying out the tests could not be blinded to the subject’s group membership (patient or control). However, the tests were carried out following a standardized protocol that was strictly adhered to, and the ultrasound data were recorded under automated conditions, with little potential for any bias to be introduced by the investigator.
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During the onset determination procedure (Figure 3) the investigators were blinded to the subject (and therefore his/her group membership), the specific muscle being examined, and the start and direction of the movement being analyzed. The data of the patients were randomly mixed in with those of the control group and encoded without user interaction, as previously described.

Statistical analysis

Since there is no information in the literature as to what constitutes a “clinically relevant difference” for the onset of abdominal muscle activation during rapid arm movements, sample size calculations were carried out [27] on the basis of expecting to record a medium effect size (of approx 0.6) for the difference between the two groups, assuming a type I error probability of 5% and a type II error probability of 20% (i.e. power of 80%). An additional 3 patients per group over and above the required sample size of approx. 45 per group were recruited, to ensure that the study would still be sufficiently powered if any subjects failed to complete the test-trials or yielded unusable data, or if technical problems resulting in data-loss should occur (all these have been reported as an issue in previous EMG studies [26, 28, 29]). Descriptive statistics are given as means ± standard deviation (SD). For each individual arm movement trial, the onset time for the earliest muscle activity (in either TrA, OI or OE muscles) was expressed in relation to the onset of MD; mean values for the “earliest onset of activity” were then calculated for each body side and movement direction. Per person, up to ten trials were available for each body side and direction, although some files had to be excluded later, e.g. due to unacceptable US or EMG quality, or onsets out of the physiologically tenable range (see earlier). The difference between the two groups for the earliest onset of
lateral abdominal muscle activity for each body side and movement direction was analyzed using a repeated-measures ANOVA with one between-group factor (group membership; patient or control) and two within-group factors (body side and movement direction). Prior matching of the two groups in terms of gender distribution, age and anthropometry served to minimize differences in potential confounders between the groups. Relationships between the onset of the earliest abdominal muscle activity and the various clinical variables (pain, disability, etc.) were examined with Pearson Product-Moment correlation coefficients. Significance was accepted throughout at the p <0.05 level.

Results

Clinical characteristics of the cLBP patients

The pain and disability characteristics of the cLBP patients are shown in Table 3.

Data quality

In the patient group, the data from 1090/4046 (27%) test trials had to be disregarded due to poor US or EMG quality, technical problems with the switch, difficulties in accurately determining the onset of activity, or “out of range” onsets (see earlier); the corresponding figure for the control group was 986/3941 (25%) data sets. This did not result in the loss of any whole datasets for a given individual; instead, it meant that mean values were calculated for <10 trials for some individuals for some test movements.

Mean time difference between the onset of the earliest abdominal muscle activity and the onset of the MD activity
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The mean onsets of abdominal muscle activity for each movement direction, body side, and group are shown in Figure 4. With the exception of right shoulder flexion movements, there was a consistent tendency for earlier lateral abdominal muscle onsets in the cLBP group than in the control group although the main effect of group membership did not achieve statistical significance (p=0.398; Table 4). However, a significant interaction between body side and group membership was observed (p=0.015; Figure 5), which was the result of earlier onsets in the cLBP group than in the control group (by on average 0.007 s; Table 5) for the right-side abdominals (i.e. left arm movements).

There was a significant main effect of movement direction (p<0.0001), with the onset of the first muscle active being significantly earlier in shoulder flexion than in either extension or abduction movements (Table 4).

There was also a significant main effect of body side (p=0.0002), with the onset of the earliest abdominal muscle activity being approximately 6ms earlier for the left than the right side abdominals (Table 5).

Relationship between the onset of abdominal muscle activity and clinical variables

There was no significant relationship between the onset of the earliest abdominal muscle activity (mean over all directions and sides) and duration of cLBP (r=0.11, p=0.46), average pain in the last week (r=0.11, p=0.46), worst pain in the last week (r=0.13, p=0.38), pain frequency (r=0.12, p=0.42), pain medication usage (r=0.15, p=0.32) or RM disability score (r=0.02, p=0.89).

Comparable results to those reported above were found when only the data from right-handed individuals (in each group) were examined.
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Discussion

The main aim of the present study was to compare the onset of activity of the lateral abdominal muscles during rapid movements of the arm in patients with non-specific cLBP and healthy controls. Both groups showed feed-forward activity of the lateral abdominal muscles. There was no suggestion of a delayed onset of activation in the patient group. Indeed, the patient group showed an overall tendency for earlier onsets of activity of the abdominal muscles, especially for the muscles on the right side i.e., with left arm movements. For logistic reasons, data collection procedures were not blinded, but they followed a standardized test protocol, were highly automated, and were hence unlikely to have introduced major bias; data analysis (onset determination) was done completely blind to group membership.

Rapid arm movements represent a challenge to postural equilibrium, and the relationship between the muscles initiating the movement and the body’s reaction is complex [6]. The execution of focal voluntary movements causes reactive forces that result in a shift of the center of gravity of the body [30, 31]. To counteract the perturbation, a carefully orchestrated interaction between the resistance offered by the active (muscular) and passive (osteoigamentous) components is required [32]. The trunk stabilizing muscles must be activated with appropriate timing and magnitude in order to prepare adequately for the impending postural perturbation.

The slightly earlier onset of activation in the cLBP patients, seen in the present study, may reflect an innate characteristic of the patients or may be the consequence of their chronic pain [26, 33, 34]. Different models have been proffered to explain the interaction between muscle activity and pain, albeit based
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predominantly on the amplitude rather than the timing of activation. The “pain-adaptation” model proposed by Lund et al. appears to be characteristic of several types of chronic pain [35]. This describes a decreased activation of the muscles during movements in which they act as agonists and an increased activation during movements in which they serve as antagonists. The process is considered to be a normal protective adaptation to avoid further pain and possible damage. The clinical circumstances (chronic pain) and findings (earlier antagonistic muscle activity) of the present study would be compatible with the chronic-pain adaptation model. Accordingly, the somewhat earlier activation of the lateral abdominal muscles in the cLBP group might be interpreted as an attempt by the central nervous system to initiate a protective “pre-stabilization” of the spine to prevent an exacerbation of pain.

Differences between cLBP patients and controls in their force-generating capacity (strength) or power/speed of contraction of the trunk muscles [36, 37] is another mechanism that might explain the slightly earlier muscle activity in patients. Weaker or slower muscles may need to initiate their feed-forward activity earlier, in order to be able to generate the necessary force, in a sufficiently timely manner, to provide stabilization. Interestingly, studies on healthy volunteers have shown that when the trunk-stabilizing muscles are fatigued — and fatigued muscles are effectively weakened, slower muscles — their anticipatory postural adjustments (APAs) occur earlier, as if in an attempt to counteract their compromised force-generating capacity and slower speed of contraction [28, 38].

It has been proposed that APAs depend on the “postural set”, i.e. the individual’s perception of their steady-state postural equilibrium and quality of external support [39], and that modifications to APAs in certain circumstances (e.g., trunk fatigue)
are centrally mediated [40]. Hence, it is conceivable that factors such as pain, fear
of pain, fear of falling over, weakness, etc. — factors that might lead patients with
cLBP to suspect that they will be less able to withstand challenges to postural
stability — could precipitate the earlier APAs.

As mentioned earlier, the greatest group difference in abdominal muscle onsets
was found for the muscles on the right-hand side, i.e., for movements made with
the left arm. If the hypothesized scenario were true, i.e., that the earlier APAs in the
patient group resulted from their greater perceived threat to balance, then this
might be accentuated during less familiar movements such as those made with the
non-dominant arm (the majority were right handed; subgroup analyses of just the
right-handed individuals showed identical findings to those presented for the whole
group). To the authors’ knowledge, no studies have examined the influence of
handedness on APAs or, in particular, handedness coupled with other (potential)
central modifiers of APAs.

The sidedness to the low back pain of the patients was not investigated in the
present study. However, previous investigations (on 23 patients with pain on the
right side, 17 on the left and 136 patients with no predominant side problem)
revealed that the location of pain played little role in determining the sidedness of
the deep trunk muscle function: in abdominal hollowing exercises there was no
association between side of symptoms and side of apparent muscular dysfunction
[41].

The results of our study do not concur with those of previous EMG studies of feed-
forward activity of the individual lateral abdominal muscles, which have instead
shown that, during rapid arm movements, the trunk stabilizing muscles are
activated significantly later in patients with LBP [1, 6, 8, 11, 42]. Whilst part of the
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answer may reside in the different measurement techniques employed (mean
intramuscular EMG onsets of individual muscles versus mean TDI tissue-velocity
measures of the earliest lateral abdominal muscle activity (present study)), we
consider it unlikely that this would result in such diametrically opposed findings. The
onset of the earliest lateral abdominal muscle activity during rapid arm movements
was considerably earlier in shoulder flexion than in extension or abduction, for both
patients and controls. This is entirely consistent with the findings reported for the
onsets measured with both TDI-velocity and intramuscular-EMG in the original TDI
validation study [11] giving credence to the validity of the data reported here. In the
original study the simultaneous use of wire-EMG and TDI recordings allowed
examination of the individual abdominal muscles’ contribution to the “earliest
activation”: the earlier mean onset in flexion was the result of the significantly
earlier TrA activation; in extension and abduction, the three abdominal muscles
(TrA, OI and OE) displayed almost equivalent mean onset times. One criticism that
can be leveled at the TDI-based assessment of the earliest muscle active is that it
does not allow differentiation between the respective activities of the three muscles.
However, it at least addresses the potential for individual variation, which, as
verified in a number of recent studies [43, 44] appears to be greater and more
important than previously appreciated. In this sense, examination of the earliest
muscle activity may better reflect the individual activation strategies that are
otherwise obscured by averaging group data for each given muscle. In the present
study, the use of the US transducer support-block and its fixation around the pelvis
may have provided extrinsic trunk stabilization and led to altered motor control
strategies; however, this would be expected to affect the cLBP and control groups
to a similar extent.
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Other potential differences compared with previous studies concern the type of LBP sufferers examined: the patients in the present study had long-term chronic LBP, and most were consulting for treatment; in contrast, in previous studies the LBP of the volunteers was more “episodic/periodic” in nature, and “less debilitating”.

Hence, our patients may well have differed from those in previous studies. Nonetheless, in terms of external validity, we would argue that our patients represented more closely the “typical” LBP patients seen in clinical practice.

Interestingly, our pilot studies on patients fitting the inclusion criteria of the former studies (patients with episodic pain) showed that these patients, too, displayed a similarly early activation pattern compared with controls (unpublished data). Clearly, the differences will only be reconciled upon further investigation in larger groups of patients, perhaps employing simultaneous EMG and TDI recordings.

It must be highlighted that the magnitude of the group difference in lateral abdominal muscle onset times was small (on average, just 7 ms earlier in the patient group than the controls) and, regardless of statistical significance, unlikely to be of clinical relevance. This is further substantiated by the lack of any relationship between the time of onset of abdominal muscle activity and either pain intensity, pain frequency, Roland Morris disability, or pain medication usage.

Our results are not the only ones to challenge the current understanding of the role of feed-forward activity of the deep abdominal muscles in spinal stability and its relevance to back pain: the work of Allison and Morris also questioned the notion of the correct anticipatory activation of TrA being important for core stability [45].

Recent data in healthy subjects showed that during one-armed movements there is feedforward-activation of the contralateral side to the arm movement, but not the ipsilateral side [15]. This questions the notion of TrA contracting bilaterally in a
corset-like manner to provide stability to the spine. Other researchers have also questioned whether unilateral TrA activation can be effective in stabilizing the spine [13, 46]. Overall, it appears that the role of lateral abdominal muscle feedforward activity in spine stabilization and in low back pain may have been overestimated.

In summary, the patients with chronic LBP examined in the present study did not show a delayed onset of feedforward activation of the lateral abdominal muscles during rapid arm movements. Earlier activation was observed for one side only compared with the controls, but within the group of patients there was no significant correlation between the time of onset of muscle activity and any of the clinical variables (pain, disability, etc.). Possibly, it is not the severity of the symptoms per se, but simply the existence of a long-term pain problem that results in the adoption of a slightly different motor control pattern. Nonetheless, the clinical significance of the phenomenon is still unclear, and should be subject to further investigation.

Although spine stabilization exercises (as a type of physiotherapy for cLBP) show clinical effectiveness, it is probably not for the reasons currently proposed.
Figure Legends

Figure 1
With the participant standing upright on a mat in a relaxed posture the ultrasound transducer was fastened around the lower trunk. Surface EMG electrodes were placed on the deltoid muscle and over the C7 spinous process. The contact switch device was attached to the wrist on the contralateral side to the ultrasound transducer, with its contact surface being attached to the lateral side of the thigh.

Figure 2
A motion-mode recording of the trunk stabilizing muscles during a rapid arm movement. **A**: A gray-scale image of the approximately parallel lying fascial borders of transversus abdominis (TrA), internal (OI) and external oblique (OE). The switch (the line at the bottom of the image) signal, which was fed in to the ECG channel of the ultrasound (US) machine, and indicated the start of the arm movement (arrow). **B**: The same trial superimposed with the tissue Doppler color (TDI) information. Tissue velocity is displayed in yellow to red for tissue movements towards the transducer, and in green to blue for movements away from the transducer.

Figure 3
Example of tissue velocity data for each muscle, acquired from the color Doppler information collected in motion-mode during one trial of rapid arm movements. The abscissa shows time in seconds [s]; the ordinate, tissue velocity [mm/s-1]. For determination of the onset of contraction one file at a time was presented on the screen, i.e. each muscle separately.
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Figure 4
Mean abdominal muscle onsets in relation to MD onset [s], for each movement direction, body side and group (see text and Table 4 for further details).

Figure 5
Mean abdominal muscle onsets [s] for each body side (mean of all movement directions) for cLBP and control groups.
References


27. *MedCalc* [Software]. Mariakerke, Belgium.


1 **Table 1.** Physical characteristics of the participants

<table>
<thead>
<tr>
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<th>cLBP(^a) participants</th>
<th>Control participants</th>
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<tr>
<td>Age (years)</td>
<td>46.5 ± 12.2</td>
<td>45.1 ± 10.9</td>
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<tr>
<td>Height (m)</td>
<td>1.69 ± 0.09</td>
<td>1.70 ± 0.10</td>
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<tr>
<td>Weight (kg)</td>
<td>73.3 ± 12.4</td>
<td>70.1 ± 13.2</td>
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<tr>
<td>BMI (kg.m(^{-2}))</td>
<td>25.8 ± 4.6</td>
<td>24.0 ± 4.1</td>
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\(^a\) Chronic low back pain (cLBP). Values given as mean ± SD. The difference between the groups was not significant for any of these parameters (p>0.05).
Table 2. Inclusion and exclusion criteria for the participants

<table>
<thead>
<tr>
<th><strong>Inclusion criteria control group</strong></th>
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<tbody>
<tr>
<td>- LBP (^a)-free for the last year</td>
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<tr>
<td>- no history of LBP requiring medical attention or time off work</td>
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<th><strong>Inclusion criteria patient group</strong></th>
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<tr>
<td>- persistent LBP with or without referred pain of a non-radical nature for at least 3 months, serious enough to cause absence from work or solicitation of medical attention</td>
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<tr>
<td>- average pain intensity over the last week and at the time of testing ≥ 3 and ≤ 8 on a 0-10 graphic rating scale and having pain in the stated range at the time of testing</td>
</tr>
<tr>
<td>- fluency in spoken and written German (spoken and reading comprehension)</td>
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<td>- willingness to comply with the study protocol</td>
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<tr>
<th><strong>Exclusion criteria patient group</strong></th>
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<tr>
<td>- constant or persistent severe pain</td>
</tr>
<tr>
<td>- non-mechanical LBP (pain unrelated to movement)</td>
</tr>
<tr>
<td>- neurological symptoms</td>
</tr>
<tr>
<td>- severe spinal instability (spondylolisthesis Grade 3 or higher [18]) osteoporosis (height loss of ≥ 4 cm since the age of 20)</td>
</tr>
<tr>
<td>- structural deformity (rigid scoliosis in clinical examination, flexion movements)</td>
</tr>
<tr>
<td>- systemic inflammatory diseases</td>
</tr>
<tr>
<td>- decompensated metabolic diseases or any other corresponding disorders preventing physical activity</td>
</tr>
</tbody>
</table>
| - participation in a structured exercise/medical training therapy program within }
Diagnostic ultrasound in low back pain

- previous spinal fusion; severe cardiovascular diseases (NYHA III and IV)
- acute infection
- recent (in the last 3 months) major abdominal surgery
- lack of co-operation
- uncontrolled alcohol or drug abuse and decompensated psychopathological diseases

**Exclusion criteria for both groups**

- visual impairment that would preclude the response to a visual stimulus
- pregnancy (or pregnancy within the last two years)

---

1a Low back pain (LBP).
Table 3 shows the pain and disability characteristics of the chronic low back pain patients (cLBP) patients.

<table>
<thead>
<tr>
<th>cLBP participants</th>
<th>Numbers (percentage)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LBP duration in years</strong>&lt;sup&gt;a&lt;/sup&gt;</td>
<td>10.1 ± 12.0 (range 8 months – 12 years).</td>
</tr>
<tr>
<td><strong>LBP intensities (0-10 graphic rating scale) in the last week</strong>&lt;sup&gt;a&lt;/sup&gt;:</td>
<td></td>
</tr>
<tr>
<td>- average</td>
<td>5.0 ± 1.7</td>
</tr>
<tr>
<td>- worst</td>
<td>6.5 ± 1.8</td>
</tr>
<tr>
<td><strong>LBP frequency</strong>:</td>
<td></td>
</tr>
<tr>
<td>- occasional</td>
<td>1/48 (2.1%)</td>
</tr>
<tr>
<td>- frequent</td>
<td>23/48 (47.9%)</td>
</tr>
<tr>
<td>- constant</td>
<td>24/48 (50.0%)</td>
</tr>
<tr>
<td><strong>Pain medication taken</strong>:</td>
<td></td>
</tr>
<tr>
<td>- not at all</td>
<td>12/48 (25.0%)</td>
</tr>
<tr>
<td>- occasionally (few times a month)</td>
<td>13/48 (27.1%)</td>
</tr>
<tr>
<td>- frequently (few times a week)</td>
<td>16/48 (33.3%)</td>
</tr>
<tr>
<td>- constantly (daily)</td>
<td>7/48 (14.6%)</td>
</tr>
<tr>
<td><strong>Mean Roland Morris score</strong>&lt;sup&gt;a&lt;/sup&gt;</td>
<td>8.8 ± 4.7 (equates to moderate disability)</td>
</tr>
</tbody>
</table>

<sup>a</sup>Values given as mean ± SD.
Table 4. Mean (SD) values for the onset [s] of the earliest abdominal muscle activity in relation to the onset of medial deltoid (MD) EMG activity for each body side and movement direction in patients with cLBP and controls.

<table>
<thead>
<tr>
<th>Direction</th>
<th>Abdominal muscle side</th>
<th>Abbreviation</th>
<th>Mean values (Controls)</th>
<th>Mean values (cLBP)</th>
<th>Mean values main effect of movement direction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Abbreviation</td>
<td>Group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extension</td>
<td>right REXT</td>
<td>0.029 ± 0.024</td>
<td>0.022 ± 0.020</td>
<td>0.022 ± 0.025</td>
<td></td>
</tr>
<tr>
<td></td>
<td>left LEXT</td>
<td>0.020 ± 0.029</td>
<td>0.018 ± 0.024</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abduction</td>
<td>right RABD</td>
<td>0.024 ± 0.023</td>
<td>0.019 ± 0.019</td>
<td></td>
<td>0.019 ± 0.022 a,b</td>
</tr>
<tr>
<td></td>
<td>left LABD</td>
<td>0.016 ± 0.023</td>
<td>0.015 ± 0.022</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexion</td>
<td>right RFLE</td>
<td>0.001 ± 0.029</td>
<td>-0.010 ± 0.028</td>
<td>-0.008 ± 0.030</td>
<td></td>
</tr>
<tr>
<td></td>
<td>left LFLE</td>
<td>-0.014 ± 0.033</td>
<td>-0.008 ± 0.027</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean values</td>
<td></td>
<td></td>
<td>0.013 ± 0.031</td>
<td>0.009 ± 0.027</td>
<td></td>
</tr>
<tr>
<td>main effect of group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P value, main effect of group</td>
<td></td>
<td></td>
<td></td>
<td>0.398</td>
<td></td>
</tr>
</tbody>
</table>

a p < 0.0001, compared with flexion. b p < 0.07, compared with extension.
Table 5. Mean (SD) values for the onset [s] of the earliest abdominal muscle activity in relation to the onset of medial deltoid (MD) EMG activity for each body side (all movement directions) in patients with cLBP and controls.

<table>
<thead>
<tr>
<th>Abdominal muscle side</th>
<th>Group</th>
<th>Both groups</th>
<th>P value, main effect of body side</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Controls</td>
<td>cLBP</td>
<td></td>
</tr>
<tr>
<td>right</td>
<td>0.018 ± 0.028</td>
<td>0.011 ± 0.027</td>
<td><strong>0.014 ± 0.028</strong></td>
</tr>
<tr>
<td>left</td>
<td>0.008 ± 0.032</td>
<td>0.008 ± 0.027</td>
<td><strong>0.008 ± 0.030</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.0002</td>
</tr>
</tbody>
</table>
Test set-up. Modified after Hodges and Richardson [8].
Figure 3.

TrA

A  TDI trace
B  Switch trace

Time [s]

Tissue Velocity [mm/s -]
Figure 4.

Mean abdominal muscle onset in relation to MD onset (s)

Controls

-0.06 -0.04 -0.02 0 0.02 0.04 0.06

LFLE
RFLE
LABD
RABD
LEXT
REXT

R/LEXT = right/left abdominal muscles, extension
R/LABD = right/left abdominal muscles, abduction
R/LFLE = right/left abdominal muscles, flexion
* p = 0.015, between the cLBP and control groups for the side difference in muscle onsets