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RESEARCH ARTICLE

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Perturbed upper limb movements cause short-latency postural responses in trunk muscles

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Abstract Addition of a load to a moving upper limb produces a perturbation of the trunk due to transmission of mechanical forces. This experiment investigated the postural response of the trunk muscles in relation to unexpected limb loading. Subjects performed rapid, bilateral shoulder flexion in response to a stimulus. In one third of trials, an unexpected load was added bilaterally to the upper limbs in the first third of the movement. Trunk muscle electromyography, intra-abdominal pressure and upper limb and trunk motion were measured. A short-latency response of the erector spinae and transversus abdominis muscles occurred ~50 ms after the onset of the limb perturbation that resulted from addition of the load early in the movement and was coincident with the onset of the observed perturbation at the trunk. The results provide evidence of initiation of a complex postural response of the trunk muscles that is consistent with mediation by afferent input from a site distant to the lumbar spine, which may include afferents of the upper limb.

Keywords Postural control · Trunk stability · Abdominal muscles · Human

Introduction

Specific trunk muscles are activated in a feedforward manner prior to rapid movements of the upper and lower limbs (Aruin and Latash 1995; Belenkii et al. 1967; Bouisset and Zattara 1981; Hodges and Richardson 1997a, 1997b) and prior to self-initiated trunk loading (Cresswell et al. 1994). This muscle activity contributes to the control of trunk orientation and centre of mass against the perturbation caused by the limb movement or loading. The perturbation to the trunk that results from voluntarily initiated tasks such as these is predictable and allows the central nervous system to initiate a specific pattern of muscle activation and trunk movements in advance of the movement (Belenkii et al. 1967; Bouisset and Zattara 1981).

The addition of an unexpected load to the upper limbs during voluntary movement introduces a further non-anticipated challenge to the trunk which cannot be dealt with in a feedforward manner. However, a delay exists between the addition of the load to the upper limb and the trunk perturbation (disturbance to the predicted movement of the trunk) owing to the relatively remote location of the lumbar spine from the upper limb. Thus, the fastest mechanism by which the central nervous system could deal with this challenge would be the initiation of trunk muscle activity in response to the afferent input from muscle and/or joint receptors of the perturbed upper limb or other structures in close proximity. Alternatively, trunk muscle activity may be initiated with a longer total latency (owing to the delay between limb and trunk perturbations) by segmental reflexes at the lumbar spine associated with afferent input from the trunk. Previous investigations of perturbed upper limb movements have identified postural changes in the contra-lateral upper limb and the lower limbs that were consistent with mediation by afferent input directly from the perturbed upper limb (Cordo and Nashner 1982; Marsden et al. 1977; Traub et al. 1980). However, investigations of postural responses in the trunk have failed to elucidate a response to excitation of upper limb afferents by vibration (Zedka and Prochazka 1997).

The first aim of this investigation was to determine whether activation of the trunk muscles could be modified by the unexpected addition of a load to the upper limbs during rapid shoulder movement. If such responses occurred, the second aim was to measure the latency of the response in individual trunk muscles, to ascertain whether this latency is consistent with mediation by afferent input from regions distant to the lumbar spine, in-

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cluding those from the upper limb, or local proprioceptive reflexes from trunk receptors. Preliminary results have been presented as an abstract (Hodges et al. 1998).

Materials and methods

Subjects

Five healthy male volunteers of mean (±SD) age 25 (±4) years, height 1.74 (±0.07) m and body mass 76 (±5) kg participated in the study. The study was approved by the Institutional Medical Research Ethics Committee and informed written consent was obtained from all subjects. Studies were performed in accordance with the Declaration of Helsinki.

Electromyography

Electromyographic (EMG) recordings were made using surface and indwelling fine-wire electrodes. Fine-wire electrodes were fabricated from multi-strand silver wire (7×75 µm-diameter wires; Leico, USA) threaded into a hypodermic needle (0.8×80 mm) and inserted into the right ventro-lateral abdominal wall muscles: transversus abdominis (TrA), obliquus internus abdominis (OI), and obliquus externus abdominis (OE) under the guidance of ultrasound imaging. The electrode insertion sites and the procedure have been described in detail elsewhere (Cresswell et al. 1992; Hodges and Richardson 1997b). Surface electrodes were placed with an interelectrode distance of 2 cm over the muscle bellies of the right rectus abdominis (RA; at the level of the anterior superior iliac spines) and erector spinae (ES) muscles (adjacent to L4) and over the right anterior deltoid in parallel with the muscle fibres. EMG was recorded from one side as movement was performed bilaterally and symmetrically. EMG was sampled at 2 kHz, amplified ×1,000, band-pass filtered between 10 and 1,000 Hz (Noraxon, Finland), and then analogue-to-digital converted at a sampling rate of 2 kHz.

EMG recordings were inspected visually to identify the onset of each burst. EMG onset was defined as the earliest increase above baseline. Traces were enlarged to a resolution of 0.5 ms and displayed individually without reference points in order to exclude observer bias. Computer-based methods of EMG onset determination are sensitive to changes in background EMG (Hodges and Bui 1996) and were deemed unsuitable for the present study, as it was expected that bursts of EMG activity may occur with activity already present in the muscle. EMG onset was expressed relative to the onset of limb movement and the onset of perturbation to the limb.

Intra-abdominal pressure

Intra-abdominal pressure (IAP) was recorded using a pressure transducer (Gaeltec, UK) inserted via the naso-pharynx into the gastric ventricle. The pressure signal was amplified 100 times using a custom-made amplifier. Changes in IAP magnitude were expressed relative to the mean baseline magnitude recorded for 75 ms prior to the stimulus to move. The onset of the increase in IAP and the time of IAP peaks were identified visually and expressed relative to the onset of limb movement and the onset of the perturbation to the limb.

Trunk and upper limb movement

Movements of the trunk and upper limb were measured using a Selspot II (Selcom, Sweden) optoelectronic system, with two cameras situated 1.5–2 m behind and to the left of the subject and angled at approximately 90° to each other. Six infra-red markers were attached to the skin (see Fig. 1 for marker locations). Position data were sampled at 350 Hz, automatically converted into three-dimensional co-ordinates and 20-Hz digitally low-pass filtered using a Butterworth-type filter (4th-order, zero lag). Calibration was performed using a pyramidal frame with four fixed markers supplied with the Selspot II. A measurement error of 0.1° was identified for the current experimental set-up (Hodges et al. 1999).

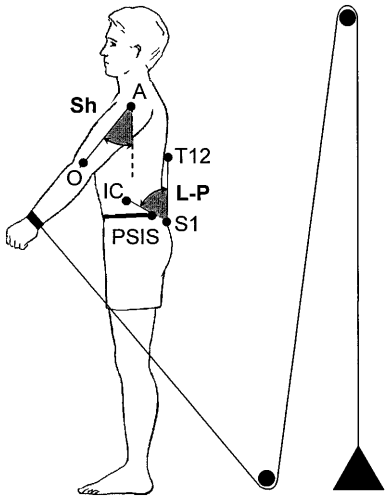

Fig. 1 Location of infra-red markers for measurement of shoulder (*Sh*) and lumbo-pelvic (*L-P*) angular motion and horizontal linear acceleration of the elbow and trunk (*S1*). The 2-kg load was attached at a specific point on the rope so that it was lifted from the ground (producing the perturbation to limb movement) when the shoulder was flexed to either 20° or 40° in the loaded trials. The pulleys were aligned so that the rope was perpendicular to the upper limb at 30° of shoulder flexion (*A* acromion, *IC* iliac crest half-way between the anterior and posterior superior iliac spines, *O* olecranon, *PSIS* posterior superior iliac spine)

Motion of the trunk and limbs was calculated as a change in angle between adjacent segments relative to a baseline angle (i.e. mean angle for 75 ms prior to the stimulus to move). The segment angles evaluated were the shoulder angle (Sh) between the upper limb (acromion process to olecranon process) and the vertical, and the lumbo-pelvic angle (L-P) between the lumbar spine (T12-S1) and pelvis (posterior superior iliac spine to iliac crest: PSIS-IC; Fig. 1). From preliminary analysis of the data, we determined that the perturbations to limbs and trunk could be most consistently identified from the trajectory data in the antero-posterior (horizontal) direction for the markers on the upper limb (olecranon) and S1 spinous process, respectively. The initiation of the perturbation was identified from the linear acceleration (the second derivative of the linear displacement data) and defined as the point where the linear acceleration of the S1 and olecranon markers deviated from the acceleration profile of the non-perturbed trials (Fig. 2A, B). The results were the same if the limb perturbation was identified from the angular acceleration of the shoulder (compared with the linear acceleration of the olecranon). In addition, the onset of perturbation to the trunk identified from the linear acceleration of S1 was the same as that identified from the angular acceleration of the lumbar spine relative to the pelvis. To assist with identification of the point of initiation of the change in acceleration, all of the perturbed and non-perturbed movement trials for each subject were plotted together for visual inspection of the data (cf. Fig. 2). By comparison of the acceleration data in this manner, it was possible to confirm that the deviation was the true onset of the perturbation and not a random irregularity in the acceleration curve. The onsets of initial upper limb and trunk movements were also identified from the linear acceleration data of the markers on the olecranon and S1 as the point of change in acceleration from the baseline. The range of angular displacement at which the perturbations to the shoulder and trunk occurred and the effect that the perturba-

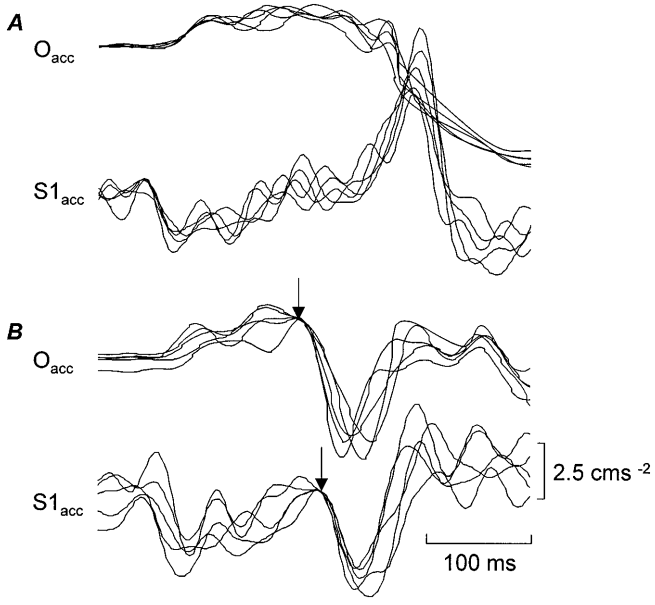

Fig. 2A, B Upper limb and trunk linear acceleration. Superimposed data (five trials) from a representative subject (same subject as in Fig. 3) of upper limb movement without (**A**) and with (**B**) addition of load to the limb. Horizontal linear acceleration of the elbow (O_{acc}) and S1 marker ($S1_{acc}$) are shown. The time of perturbation to the upper limb and trunk that were identified from the onset of deceleration of the olecranon and S1 markers, respectively, are indicated with *arrows* in **B**

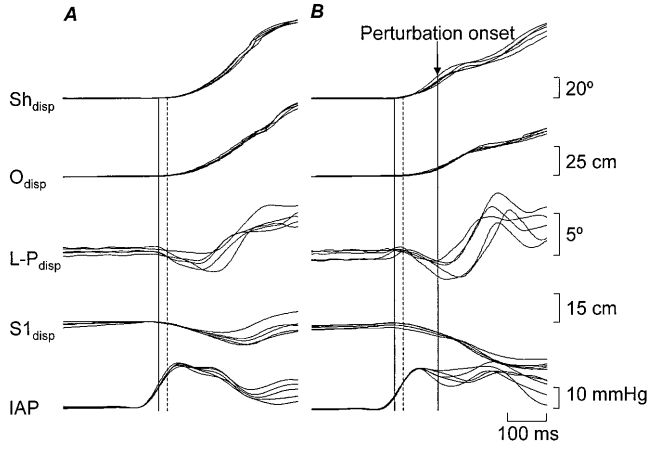

Fig. 3A, B Effect of addition of load to the upper limbs. Superimposed data (five trials) from a representative subject of upper limb movement without (**A**) and with (**B**) addition of load to the limb. Shoulder angular displacement (Sh_{disp}), elbow linear horizontal displacement (O_{disp}), lumbo-pelvic angular displacement ($L\text{-}P_{disp}$), S1 linear horizontal displacement ($S1_{disp}$) and intra-abdominal pressure (*IAP*) are shown. Flexion between segments and forward displacement is defined in the upward direction. The *vertical dotted line* indicates the onset of shoulder movement and the *solid line* indicates the onset of trunk motion identified from the linear horizontal acceleration of the olecranon and sacrum (see Fig. 2). The movements and perturbation were reproducible between trials. The onset of perturbation to the upper limb (identified from the onset of decreased acceleration of the olecranon marker (see Fig. 2) is indicated in **B**

tion had on the angular displacement of the upper limb and trunk were identified from the shoulder (Sh) and lumbo-pelvic (L-P) angular displacements (Fig. 3).

Procedure

In standing, subjects performed 30 repetitions of bilateral shoulder flexion from the start position, with the upper limbs beside the body, to 60° forwards from the vertical position in response to a light stimulus. Subjects were shown the approximate distance to move and instructed to move as fast as possible. An auditory warning was presented at a random period (0.5–4 s) prior to the light stimulus to increase the consistency of the reaction time (Schmidt 1988). Subjects were requested to remain relaxed when they received this warning and to focus their attention on the forthcoming light stimulus. Each trial was separated by approximately 30 s and subjects were allowed to rest sitting between each set of ten trials. Ropes were attached to straps around both wrists and were drawn through pulleys that were positioned such that the rope was perpendicular to the upper limb when the upper limb was at 30° flexion from the vertical (Fig. 1). In the majority of trials (20/30), no load was attached to the rope and the motion of the upper limbs was only minimally resisted by the weight of the rope. In ten trials a 2-kg load was added to the end of a rope connected at the junction of the two ropes from the subject's wrists. Care was taken to ensure that the unexpected loading of the limbs occurred symmetrically and simultaneously. The attachment of the load to the rope was adjusted such that it was lifted from the support surface at either 20° (five trials) or 40° (five trials) of limb movement. The addition of the load at 40° of shoulder flexion was included to reduce the predictability of the loading, and data from these trials were not included in the analysis. The order of presentation of trials (no load, 20 trials; loading at the 20° position, 5 trials; loading at the 40° position, 5 trials) was randomised and unknown to the subject to insure that the loading was unexpected. In all trials the subject was required to move his upper limbs as fast as possible and to continue to approximately 60°, even when a load was applied. After reaching the end position, the limbs were returned to the side of the body.

Statistical analysis

Analyses were undertaken to compare, between conditions, the EMG, IAP and trunk motion parameters for the initial response to confirm that the initial phase of the movement was similar between trials with and without the addition of the load to the limbs. Five trials from both conditions were stored for analysis. This involved each of the perturbed trials and 5 trials that were selected randomly (using shuffled index cards) from the pool of 20 available trials in which no perturbation was applied. The presence of artefact (due to movement or obstruction of markers, for example) in the EMG or movement recordings resulted in rejection of those data for that trial in less than 10% of trials. Analysis of the initial response involved comparison of the timing of onset of the increases in EMG, IAP and L-P angular displacement (relative to the onset of limb movement) between conditions using a one-way repeated-measures, multi-variate analysis of variance (MANOVA) with post-hoc testing using Duncan's multiple-range test.

For the trials in which load was added to the upper limb, the latencies between the deceleration of the olecranon marker (i.e. onset of upper limb perturbation) and the onsets of EMG, IAP and deceleration of the S1 marker (i.e. onset of perturbation to the trunk) were compared with a one-way analysis of variance (ANOVA) with post-hoc testing, using Duncan's multiple-range test. The amplitude of the peak L-P angular displacement was compared between conditions using Student's *t*-test.

A second response of ES and TrA was recorded in many of the trials with no perturbation. Thus, it was necessary to confirm that the response identified after the addition of the load in the perturbation trials was not simply this normally occurring second re-

sponse. To confirm this, an additional analysis (one-way repeated-measures MANOVA) was undertaken to compare, between trials with and without loading, the latency of the second EMG burst from the onset of upper limb movement. The significance level was set at 0.05. Data are presented as means (±SEM) throughout the text.

Results

Non-perturbed trials

Rapid shoulder flexion performed by subjects without the addition of an unexpected load resulted in an overall flexion motion between the lumbar spine (T12-S1) and pelvis (PSIS-iliac crest) to a mean maximum of 5.4° (±0.9°; L-P_{disp} in Fig. 3A). The direction of angular motion between these segments reversed (i.e. time of peak flexion) at 336 (±25) ms after the onset of shoulder motion. A small but consistent extension motion (downward motion of L-P_{disp} in Fig. 3A) between these segments of 1.9° (±0.1°) was initiated a mean of 26 (±6) ms prior to the onset of limb movement. This preparatory motion is demonstrated in the first panel in Fig. 3 as the onset of downward deviation of the L-P_{disp}. This anticipatory trunk motion has been described previously (Hodges et al. 1999). The duration of limb movement from the start position to 60° from vertical was 450 (±39) ms.

Activation of TrA, ES and deltoid preceded the onset of limb movement by 35 (±6) ms, 36 (±5) ms and 29 (±4) ms, respectively (Fig. 4A, C). Although there was some variability in the initial responses of the other abdominal muscles, the EMG onset of OI, OE and RA followed the onset of limb movement, on average, by 4 (±20), 47 (±12) and 169 (±17) ms, respectively. A second burst of ES and deltoid EMG occurred 338 (±23) ms and 337 (±21) ms, respectively, after the limb movement onset. However, the onset of these second responses showed considerable variation, even between trials in a single subject (Fig. 4A, C), and was not clearly identified in some subjects (e.g. Fig. 4A). In 44% of trials, a second burst of TrA EMG was identified 278 (±14) ms after the onset of limb movement (Fig. 4A). In the remaining trials, the initial burst of TrA EMG was followed by activation of TrA at a lower magnitude. An increase in IAP was initiated 31 (±6) ms prior to limb movement and reached its peak of 26 (±2) mmHg at 127 (±7) ms after the onset of limb movement (Fig. 3A).

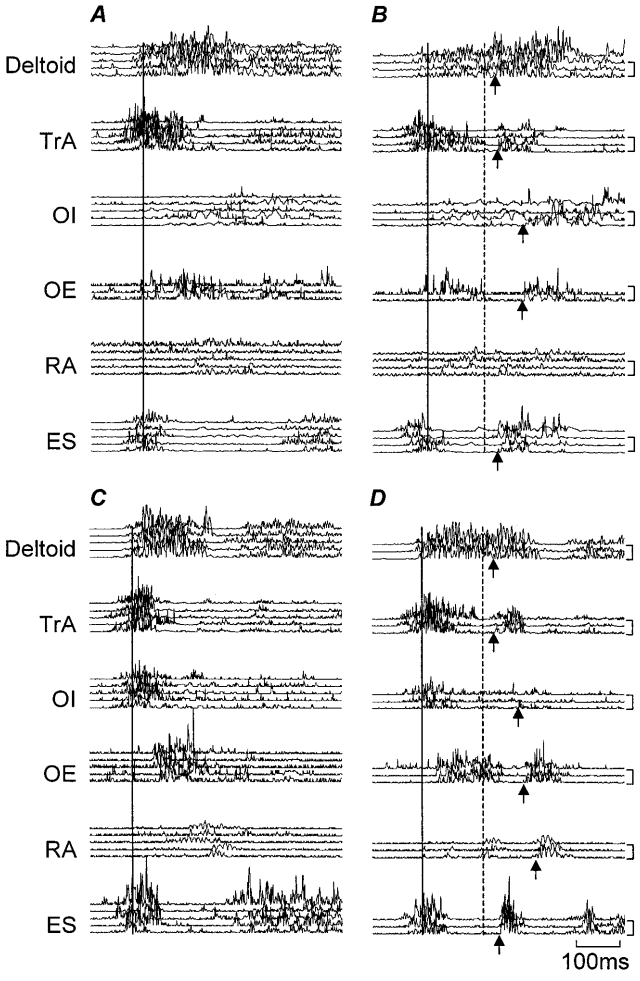

Fig. 4A–D Response of the trunk muscles to addition of a load to the upper limbs. Rectified EMG data for five individual trials are presented for two representative subjects for trials without (**A, C**) and with (**B, D**) addition of a load to the upper limbs. Trials with movement artefact have been removed from **A**, **B** and **D**. The onset of limb movement is identified by the *solid line* and the *dashed line* indicates the mean onset of deceleration of the upper limb in the trials with addition of the load. The *arrows* indicate the approximate mean onsets of EMG for each muscle (taking into account the trial-to-trial variation) following the perturbation. Despite some variation between subjects (e.g. absence of a definitive right rectus abdominis, *RA*, response in the *upper panels*), the general features, including the short-latency response of transversus abdominis (*TrA*) and erector spinae muscles (*ES*) after the perturbation to the upper limb remained consistent between trials and between subjects. The obliquus internus abdominis (*OI*) traces in **A** and **B** contain some movement artefact. Vertical calibration is 0.2 mV

Perturbation at 20° of shoulder flexion

There was no statistical difference in temporal and spatial parameters of EMG, IAP and trunk motion in the initial phase of movement prior to the perturbation between the control trials and the trials where load was suddenly added to the upper limbs at approximately 20° (range 13–27°), 141 (±8) ms after the onset of limb movement (cf. Figs. 3, 4). However, changes in trunk motion and an additional response of the trunk muscles occurred shortly after the perturbation to the upper limbs. A change in the trunk movement (deceleration of S1 marker; Fig. 2) occurred 40 (±4) ms after the onset of the perturbation to the upper limbs (Fig. 2) and resulted in an increased relative flexion motion between the lumbar spine and pelvis (L-P in Fig. 3B) that was initiated when the L-P angle was at 1.5 (±0.6)° of extension relative to the initial position. A peak L-P flexion of 5.4 (±0.4)° was recorded a mean of 417 (±21) ms after the onset of limb movement (but prior to the end of range of shoulder flexion). This

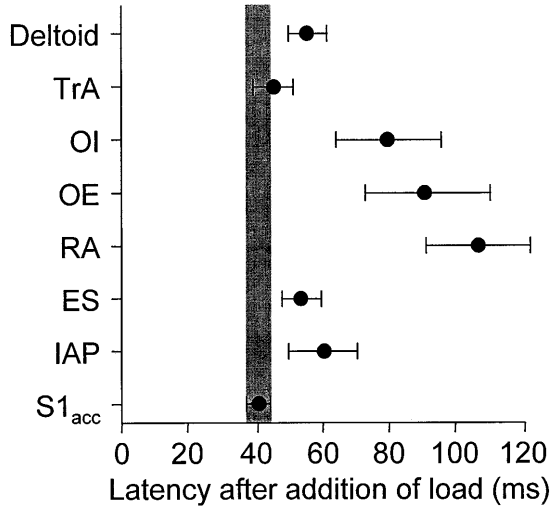

Fig. 5 Short- and long-latency responses of the trunk muscles following addition of a load to the upper limbs. Mean latencies (±1 SD) of the EMG responses of the trunk and limb muscles and IAP from the onset of deceleration of the upper limb resulting from addition of a load to the upper limb (time zero). The *grey box* indicates the mean of the onset of perturbation to the trunk (defined as the onset of the change in acceleration of the S1 marker). The onsets of TrA and ES EMG occurred at the same time as the onset of the perturbation to the trunk

angle was not different in magnitude from the peak angle of trunk flexion identified in the control trials. The duration of shoulder flexion movement was increased to 557 (±43) ms.

In 64% of trials, it was difficult to identify the onset of the response of the deltoid to the upper limb perturbation, since this muscle was already active for movement production (Fig. 4D). However, in trials where the onset was obvious, it occurred 56 (±6) ms after the onset of the perturbation (Fig. 4B).

The earliest responses of the trunk muscles after the perturbation to the upper limbs were those of ES and TrA, which were not significantly different from each other (Figs. 4B, D, 5). These responses occurred either before or with short latency after the perturbation to the trunk, with mean latencies of 53 (±6) ms and 45 (±8) ms after perturbation to the upper limbs for ES and TrA, respectively, and were not statistically different from the onset of the perturbation to the trunk (i.e. 40±4 ms after addition of load to the arms; P=0.94). This short-latency response of TrA and ES was consistent for all subjects. The latency between the onset of shoulder movement and the onsets of these EMG responses of TrA and ES (181±5 ms and 194±5 ms, respectively) was significantly less than those for the second responses identified in the non-perturbed trials. This finding provides evidence that the second response in the perturbed trials was associated with the perturbation to limb movement. The onsets of EMG of the other trunk muscles occurred with a significantly longer latency after the onset of perturbation than for ES and TrA ($P<0.01$; Fig. 5) and were not significantly different from each other ($P=0.52$). Unlike the short-latency responses of TrA and ES, there was greater variability in the EMG onsets for OI, OE and RA.

In addition to the early IAP peak of 20 (±3) mmHg that occurred 119 (±9) ms after the onset of movement, a second IAP peak was initiated 60 (±11) ms after the perturbation to shoulder motion and reached its maximum 142 (±9) ms after the perturbation (Fig. 3B). The magnitude of the second IAP increase was 30 (±2) mmHg.

Discussion

The results of this study demonstrate a short-latency response of specific trunk muscles to an unexpected perturbation to upper limb movement that was too fast to be mediated by segmental spinal reflexes in response to reactive trunk movement. The latency of this response is consistent with a response mediated by afferent input from receptors distant to the lumbar spine and potentially may involve afferents of the upper limb.

Short-latency response of the trunk muscles

The latency from the arm perturbation to the responses of ES (45–53 ms) is similar to that reported in the previous studies for different tasks. ES activity has been identified with a latency of: ~65 ms after a load is applied to a harness over the shoulders (Carlson et al. 1981) or when an upper limb is pulled unexpectedly (Marsden et al. 1981); ~60 ms after the surface on which a subject stands is rotated forwards (Carpenter et al. 1999; Keshner et al. 1988); ~55 ms after posterior translation of the support surface (Keshner et al. 1988); and ~80 ms after a load is caught in a box held in the hands (Wilder et al. 1996). A response of ES has also been identified with a latency of ~88 ms after electrical stimulation of the brachial plexus, which resulted in abduction of the upper limb (Ertekin and Ertekin 1981). However, due to electromechanical delay between electrical stimulus and arm movement (up to ~40 ms; Norman and Komi 1979), the true latency of this response from onset of arm movement would be somewhat shorter. While some of the variability in ES latency between tasks may be due to differences in determination of onset of EMG activity as a result of factors such as data processing (see Hodges and Bui 1996), differences in the task characteristics and perturbation method are also likely to influence the response. Only one previous study has investigated the response of TrA to an unexpected load. A response of TrA was identified ~24 ms after a perturbation directly to the trunk as a result of addition of a load to a harness over the shoulders (Cresswell et al. 1994).

As the latency between perturbation and mechanical changes at the spine is likely to vary between tasks (owing to factors such as the stiffness of segments between the perturbation and trunk), and as few studies have directly measured these mechanical events, it is dif-

ficult to determine the mechanism responsible for their initiation. An exception is the study of support-surface rotation, in which trunk motion was measured 20 ms after the commencement of the rotation (Carpenter et al. 1999). This would allow ~40 ms between trunk perturbation and ES response for that task if the latency to ES activity is 60 ms. In the present study of perturbed arm movement, the change in acceleration of the trunk – the onset of perturbation to the trunk – was initiated 40 ms after the addition of the load to the arm. The onset of EMG of ES and TrA occurred at a latency that was not different to the latency to the trunk perturbation.

The response of the ES and superficial abdominal muscles to a direct tap to the muscle (i.e. stretch reflex) has been shown to occur with a latency of 12–16 ms (Dimitrijevic et al. 1980; Tani et al. 1997; Zedka and Prochazka 1997) and ~19 ms (Kondo and Bishop 1987), respectively. These potentials represent the fastest responses that can be expected to occur in these muscles as a result of mechanical stimuli. For TrA, the mean difference in latency between the EMG onset and the trunk perturbation was ~5 ms (although there was no statistical difference between the latency of these two events), which is not sufficient for even the fastest reflex response to be responsible for its generation. In contrast the mean latency between the trunk perturbation and the onset of ES activity was ~15 ms (although again there was no statistical difference between the latency of trunk perturbation and onset of EMG activity) and may be consistent with mediation by a segmental reflex to homonymous muscle stretch. However, in ~40% of the trials, the response of ES either preceded the perturbation or followed it by less than 2 ms. Thus, the present findings cannot be explained by simple segmental reflex pathways as a result of mechanical disturbance at the spine and, therefore, other mechanisms must explain the origin of these short-latency responses.

Previous authors have identified short-latency responses (55–88 ms) of muscles of the legs and contralateral upper limb following perturbation of an upper limb produced by a rapid change in limb loading (Marsden et al. 1977, 1981; Traub et al. 1980) or by lever pulls (Cordo and Nashner 1982). Similar to the present data, the responses in leg muscles to perturbation to the upper limb have been shown to occur prior to the motion of the leg that results from forces transmitted from the perturbation to the upper limb (Marsden et al. 1977, 1981; Traub et al. 1980). Furthermore, these responses have been shown to occur even after anaesthetisation of the leg to exclude mediation by peripheral afferent information from the leg (Traub et al. 1980). Thus, the responses were considered to be mediated directly by afferent input from the perturbed upper limb and not in response to segmental stretch reflexes caused by motion transmitted to the distant segment from the perturbation (Marsden et al. 1977). In the present study, responses of TrA and ES that were initiated with short-latency after the perturbation to upper limb movement are consistent with the responses identified in leg muscles. As the responses occurred at the same time as the onset of the change in trunk acceleration, reflexes from lumbar spine, motion could not have mediated them. In addition, since TrA has a horizontal fibre orientation, there would be little change in muscle length with trunk motion, thus ruling out the possibility of mediation of its response by type Ia afferents from homonymous muscle spindles.

The latency from the perturbation to the responses of TrA and ES (45–53 ms) is similar to that identified in the previous studies of biceps and triceps brachii of the opposite upper limb (55 ms; Marsden et al. 1977) or triceps surae (80 ms, which includes increased latency due to conduction in long peripheral nerves; Traub et al. 1980) with perturbation to the upper limb. Although this short latency is insufficient to involve transcortical mechanisms (more than 55 ms; Marsden et al. 1977), this does not exclude the possibility of modulation of the response by excitatory or inhibitory influences from higher centres. It is unlikely that the response is mediated as a simple stereotypical stimulus-response and, in all likelihood, there must be some integration of inputs. For instance, simple excitation of upper limb afferents by provision of a vibration stimulus or electrical stimulation of the flexor and extensor muscles of the wrist does not reproduce the response of ES that is normally observed during voluntary wrist motion (Zedka and Prochazka 1997). Thus, it appears that some judgement is made of the functional significance of the afferent information in order for the trunk muscle response to be formulated. However, the short latency (45–53 ms) allows for limited integratory networks to be involved.

Consideration of the origin of the reflex changes in trunk muscle activity requires extreme precision in the determination of the mechanical events of the arm and spine, and the onsets of EMG activity. The identification of EMG onset following the perturbation was facilitated by the fact that this response of the trunk muscles was initiated during a period of relative EMG silence after the initial bursts of activity of the trunk muscles that were initiated prior to the onset of arm movement. Thus the onset of the majority of the EMG responses of the trunk muscles could be clearly defined. This was not the case for deltoid muscle, which was frequently active at the initiation of the response to unexpected loading. Furthermore, EMG recordings of the abdominal muscles (and also for ES in pilot trials) were made with fine-wire electrodes that recorded action potentials from a limited number of motor units. In this type of recording, the determination of EMG onset is more precise than multiunit recordings, as it is possible to identify the instant of recruitment of the first motor unit within the recording volume. With the sampling frequency used, this was accurate to within 0.5 ms. The accuracy of determination of the instant of perturbation onset was optimised by evaluation of the linear acceleration rather than the displacement, and the time of perturbation was identified as the point where the acceleration profile deviated from the curve in the non-perturbed trials. Movement trials from all repetitions by each subject were displayed together to

optimise the selection of the perturbation point. The resolution of the movement analysis allowed an accuracy of ~3 ms. While this would provide adequate resolution to make judgements of the latency between spinal movement and EMG onset, developments in in vivo measurement of tension in muscle fibres and/or spinal ligaments or microneurographic recordings of spinal afferent volleys would clarify this issue.

Long-latency response of the trunk muscles

No responses of RA, OE or OI were identified in response to the perturbation to the upper limb with a similar latency to that of TrA and ES. However, a longer-latency EMG response of these muscles was often observed 79–145 ms after the load was added to the moving limb. This latency after the perturbation to the trunk (38–108 ms) is sufficient to include segmental inputs from the lumbar spine due to movement transmitted from the perturbation of the upper limb. Alternatively, the response of the antagonist "flexor" abdominal muscles may act to modulate or "tune" the output of the early response of ES. A similar longer latency response of the antagonist trunk muscles has been identified in association with rapid movement of the upper limb (Aruin and Latash 1995; Friedli et al. 1988; Hodges et al. 1999) and with support-surface translation (Keshner et al. 1988).

Significance of the trunk muscle response

The response of the trunk muscles associated with voluntary limb movement is thought to contribute to control of the trunk against the reactive moments from the movement (Belenkii et al. 1967; Bouisset and Zattara 1981; Friedli et al. 1988; Hodges and Richardson 1997b). Recent evidence has confirmed a relationship between ES, RA, OE and OI activity and control of trunk orientation with bilateral shoulder flexion and extension (Hodges et al. 1999). With voluntary upper limb flexion, which produces a perturbation to the trunk causing trunk flexion (Hodges et al. 2000), activity of ES is initiated prior to the movement and is consistent with the requirement to control the trunk flexion moment (Friedli et al. 1988; Hodges et al. 1999, 2000). If the arm is considered as a stiff segment, with a fixed centre of rotation about the shoulder joint, the force applied by the addition of the unexpected load at the distal end of the arm (and perpendicularly to its longitudinal axis) will generate an additional force at the shoulder joint that acts on the trunk in the opposite direction to the applied force. The first effect (before any change in muscle moment) of the backward force on the hand will thus be to push the shoulder forward, thereby acting to flex the trunk. After this initial effect, the addition of an increased shoulder torque will further emphasise the flexion moment resulting from the reactive moments from shoulder flexion. Consistent with this prediction, the deceleration of the upper limb was followed by a rapid deceleration, or decreased acceleration, of forward motion of the pelvic (S1) marker, then an acceleration of trunk flexion. The flexion ($L\text{-}P_{disp}$) that was produced by the addition of the load to the limbs is similar to the perturbation to the trunk evoked by the initiation of shoulder flexion, although the central nervous system cannot predict its onset. Accordingly, the early response of ES following the unexpected perturbation to the upper limbs is consistent with the requirement to control the acceleration of trunk flexion. Other studies in which an unexpected trunk flexion perturbation has been generated by forward translation of the support surface have also identified a short-latency response of ES following the trunk acceleration (Keshner et al. 1988).

In contrast to the other trunk muscles, anticipatory activity of TrA has been found irrespective of the direction of perturbation to the trunk from upper limb movement and is, thus, inconsistent with the control of trunk orientation (Hodges and Richardson 1997b; Hodges et al. 1997, 1999). Anatomically TrA has a limited ability to generate trunk flexion or extension torque due to its mainly horizontal fibre orientation (Williams et al. 1989). Yet TrA has been found to be the first trunk muscle active with voluntary upper (Hodges and Richardson 1997b) and lower (Hodges and Richardson 1997a) limb movement in each direction and with expected and unexpected loading of the trunk producing trunk flexion (Cresswell et al. 1994). It is hypothesised that this muscle may contribute to trunk control through the maintenance of spinal inter-segmental stiffness (Cresswell et al. 1994; Hodges and Richardson 1997b). The short-latency co-activation of TrA and ES in the present study is consistent with the control of both segmental stiffness and trunk orientation. In addition, it has been proposed that TrA may contribute to trunk extension via its role in the production of IAP (Daggfeldt and Thorstensson 1997; Grillner et al. 1978). In the present study, increases in IAP were recorded shortly after the activation of TrA that occurred prior to movement onset and after the perturbation produced by upper limb loading. However, it is uncertain whether the small pressures recorded are sufficient to significantly assist ES in the control of trunk flexion.

Conclusion

Functionally, internal (self-initiated) and external forces acting on the body rarely result in discrete perturbations to the trunk. In contrast, a complicated and varying combination of forces acts on the trunk to produce a mixture of feedforward and feedback-mediated responses of the trunk muscles in order to maintain stability and perhaps limit the risk of injury. The present study provides evidence that postural responses of the trunk muscles may be initiated in response to afferent feedback from distant segments in addition to proprioceptive information resulting from trunk motion.

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