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ORIGINAL ARTICLE

Paraspinal muscle control in people with osteoporotic vertebral fracture

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Abstract The high risk of sustaining subsequent vertebral fractures after an initial fracture cannot be explained solely by low bone mass. Extra-osseous factors, such as neuromuscular characteristics may help to explain this clinical dilemma. Elderly women with ($n = 11$) and without ($n = 14$) osteoporotic vertebral fractures performed rapid shoulder flexion to perturb the trunk while standing on a flat and short base. Neuromuscular postural responses of the paraspinal muscles at T6 and T12, and deep lumbar multifidus at L4 were recorded using intramuscular electromyography (EMG). Both groups demonstrated bursts of EMG that were initiated either before or shortly after the onset of shoulder flexion ($P < 0.05$). Paraspinal and multifidus onset occurred earlier in the non-fracture group (50–0 ms before deltoid onset) compared to the fracture group (25 ms before and 25 ms after deltoid onset) in the flat base condition. In the short base condition, EMG amplitude increased significantly above baseline earlier in the non-fracture group (75–25 ms before deltoid onset) compared to the fracture group (25–0 ms before deltoid onset) at T6 and T12; yet multifidus EMG increased above baseline earlier in the fracture group (50–25 ms before deltoid) compared to the non-fracture group (25–0 ms before deltoid). Time to reach maximum amplitude was shorter in the fracture group. Hypothetically, the longer time to initiate a postural response and shorter time to reach maximum amplitude in the fracture group may indicate a neuromuscular contribution towards subsequent fracture aetiology. This response could also be an adaptive characteristic of the central nervous system to minimise vertebral loading time.

Keywords Osteoporosis · Vertebral fracture · Paraspinal muscle · Electromyography · Neuromuscular control

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Introduction

Vertebral fractures are associated with a number of physical impairments and psychosocial morbidities and pose a significant burden on the public health system [12, 32]. Furthermore, these sequelae become more pronounced with each subsequent vertebral fracture sustained [26]. Once an individual sustains a vertebral fracture, the risk of subsequent vertebral fracture increases by up to four to sevenfold and then exponentially for each fracture sustained thereafter [37, 38]—known as the 'vertebral fracture cascade'. However, despite the large volume of research dedicated to examining fracture risk and the efficacy of pharmacologic agents, little is understood surrounding the aetiology of vertebral fractures, particularly mechanisms underlying subsequent fractures.

Although bone mineral density (BMD) accounts for a large proportion of the variance observed in vertebral bone strength in the thoracic and lumbar spine [43], areal BMD still remains an inadequate predictor of vertebral fracture risk. The strong relationship between BMD and bone strength underlines the rationale for the use of densitometry in assessing fracture risk and skeletal status. However, evidence from epidemiologic studies suggests a lack of discriminant power of standard BMD measures [28] and a poor sensitivity of areal BMD T-scores in identifying fracture cases [10]. This suggests that factors other than BMD may influence the aetiology of first-time and subsequent vertebral fracture [4]. Local factors such as cortical bone structure [33], bone quality [1] and bone geometry [13] appear to have an influential role. However, extra-osseous factors may also influence vertebral fracture aetiology. Previous research has found the presence of an osteoporotic vertebral fracture to be associated with reduced back extensor strength [41], decreased spinal mobility [30], altered balance characteristics and changes in trunk muscle control [14] compared to individuals with osteoporosis and no history of vertebral fracture. However, muscle control characteristics specifically around common fracture sites and in muscles that provide intersegmental stability remain unexplored

Osteoporotic vertebral fractures occur most frequently in the mid-thoracic spine and thoracolumbar junction [7, 11]. The paraspinal muscles attach to the vertebrae or in close proximity, and are responsible for generating large compressive forces. Therefore quantification of muscle force at the mid-thoracic spine and thoracolumbar junction would be advantageous. Notably, vertebral fractures have been associated with increased segmental spinal loads and trunk muscle forces predicted through biomechanical models using optimisation routines [6]. A limitation of optimisation models is that differences in neuromuscular strategies between individuals cannot be explored. Neuromuscular strategies are best explored using electromyography (EMG). However, deriving muscle force from muscle activity data collected through EMG usually requires normalisation of EMG data to a maximum voluntary contraction. This procedure would not be appropriate in a population with compromised vertebral strength. An alternative approach is to examine temporal characteristics of muscle activity during a postural response. Rapid, voluntary arm movement is a suitable paradigm in which to measure a muscular postural response and has been used for this purpose in previous research [18–20, 23, 31]. Rapid flexion of the upper limb moves the centre of mass of the body anteriorly and causes resultant flexion motion between trunk segments of up to 8°, and to maintain equilibrium, the central nervous system initiates appropriate responses in the paraspinal muscles [18]. The nature of these responses may be different between individuals with and without fractures and therefore help to explain a potential mechanism underlying the aetiology of subsequent vertebral fracture.

The anatomy of the lumbar multifidus muscle suggests that it has an important role in maintaining stability of the lumbar spine [45]. Previous research has identified that individuals with low back pain have atrophy of the multifidus muscle [16] and changes in recruitment [27]. Recent studies suggest that impaired activity of multifidus in low back pain may be associated with a greater response of more superficial paraspinal muscles [17]. Maladaptive multifidus recruitment patterns may compromise intersegmental stability of the lumbar spine and therefore reduce the ability of the spine to resist shear loading. In a population with underlying vertebral fragility, either of these changes may be sufficient to increase fracture risk.

To specifically measure paraspinal muscle activity at the mid-thoracic and thoracolumbar levels and deep lumbar multifidus, intra-muscular EMG is required [31]. This study examined the association between vertebral fracture and paraspinal muscle recruitment characteristics in a population with osteoporosis using intra-muscular EMG.

Materials and methods

Participants

Twenty-five elderly, community dwelling females with osteoporosis were recruited and divided into two groups—those with an osteoporotic vertebral fracture in the thoracic spine ($n = 11$) and those without fracture ($n = 14$). Osteoporosis was confirmed on the basis of bone densitometry tests according to the classification system proposed by the World Health Organization. Vertebral fractures were identified from standing, lateral radiographs of the thoracic and lumbar spine based on a conservative morphometric deformity criteria. Vertebrae were classified as wedge-fractured when anterior vertebral height was reduced ≥30% compared with posterior height in that and the adjacent superior or inferior vertebra, measured using digital image processing software [29]. Qualitative review by a radiologist ensured that compression fractures were not overlooked. Fifteen thoracic wedge

fractures were identified in the fracture group at vertebrae T4 (20%), T5 (13.3%), T6 (26.6%), T7 (6.7%), T8 (20%), T9 (6.7%), T12 (6.7%). Radiographs were also used to measure thoracic curvature using the regional vertebral centroid angle [5, 15]. Physical activity was assessed using the Physical Activity Scale for the Elderly (PASE), which has been validated previously [44]. Pain prior to and during testing was measured using a visual analogue scale (VAS). There were no significant differences in the physical characteristics between the groups (all: $P > 0.05$, Table 1). Pain prior to, and during testing ranged from 0 to 2/10 on the VAS and was not different between the groups ($P > 0.05$, Table 1).

All participants provided written, informed consent, and approval to conduct the study was granted by institutional Human Research Ethics Committees. Participants described in this study have also been involved in other projects conducted by our group.

Electromyography

Electromyographic activity of the longissimus thoracis at T6 and T12, and deep lumbar multifidus at L4 were recorded using intra-muscular electrodes. We did not record EMG from participant-specific fracture sites, but rather vertebral levels that commonly fracture in osteoporosis, namely T6 and T12. Bipolar fine-wire electrodes were made from two equal lengths of Teflon coated stainless steel wire (140 μm diameter, A-M systems Inc., Carlsborg, WA, USA) and were inserted into a hypodermic needle (longissimus: 0.65 mm × 32 mm; multifidus: 0.65 mm × 70 mm). Teflon coating (1–2 mm) was removed from the end of the wires and the tips were bent back for form a hook that would embed in the muscle tissue. Electrodes were inserted with ultrasound guidance into the left longissimus thoracis muscle at the T6 and T12 levels ~1 cm lateral to the spinous process and into the deep multifidus muscle at L4, ~4 cm lateral to the L4 spinous process in an antero-medial direction [30]. A pair of Ag/AgCl adhesive EMG electrodes with a 10 mm diameter and 20 mm inter-electrode distance were placed over the right anterior deltoid muscle. A ground electrode was placed over the iliac crest. EMG data were amplified with a gain of 1,000, band pass filtered between 20 and 1,000 Hz using a second order Butterworth 12 dB/octave filter, including a notch filter at 50 Hz, and sampled at 2,000 Hz. Data were recorded and stored using Spike 2, version 4.10 software (Cambridge Electronic Design Limited, UK), and exported for analysis with Matlab 7.5 (The Mathworks Inc., Natick, MA, USA).

Task protocol

Participants stood in a frame surrounded on three sides by rails, which they were encouraged to only use for balance when required. Two standing surfaces were used in the frame, presented in a random order—a flat base and a short base. The short base consisted of a beam of wood (40 mm × 120 mm × 1,000 mm) on which participants stood with feet shoulder width apart and equal weight through both feet. When standing on the short base, the participants' toes and heels were placed over the edges of the beam but were unable to touch the floor. The short base was used to challenge balance by reducing the contribution of the ankle muscles to postural control, thus increasing the demand for the hip strategy involving hip and trunk movement [24]. Participants performed rapid right arm movements (shoulder flexion to ~60°) in response to a light positioned at eye level, during which EMG data were collected. Between each arm movement trial, participants were instructed to relax their back muscles, and this was confirmed from the real-time EMG recording. An accelerometer was attached to the right hand to provide information regarding the onset of arm movement. EMG data for the back muscles were collected over ten trials of rapid arm movements.

Data analysis

For each participant and each base, EMG data for the deltoid were displayed and time of muscle onset was identified visually as the point where EMG activity increased above baseline (Fig. 1, top). EMG data were

Table 1 Physical characteristics and subjective reporting of pain using the VAS in each group, presented as the mean (SD)

Group	*n*	Age (years)	Mass (kg)	Height (m)	BMI	Kyphosis (°)	PASE	VAS score prior to test	VAS score during test
Fracture	11	68.4 (6.7)	67.6 (10.7)	1.61 (0.06)	26.1 (4.0)	33.5 (8.6)	161.1 (48.8)	0.6 (0.7)	1.1 (0.8)
No fracture	14	64.0 (8.9)	59.8 (9.2)	1.58 (0.04)	24.0 (3.3)	32.5 (7.6)	156.7 (54.5)	0.7 (0.8)	1.1 (0.6)

BMI body mass index, *PASE* physical activity scale for the elderly, *VAS* visual analogue scale for pain (scored between 0 and 10)

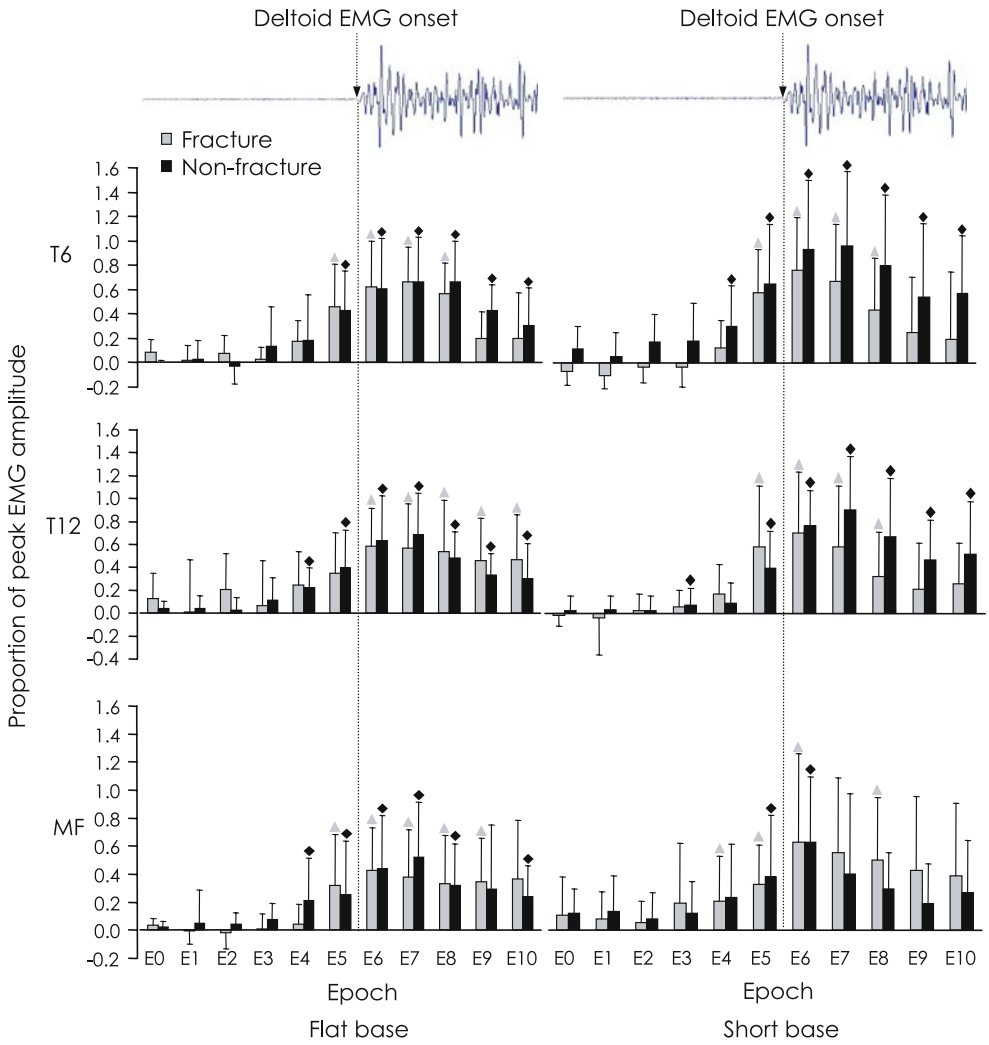

Fig. 1 Normalised mean EMG (as a proportion of peak amplitude in the flat surface condition) for *epochs* before (125–0 ms) and after (0–125 ms) onset of deltoid for fracture (*grey bars*) and non-fracture (*black bars*) groups in the flat base (*left panel*) and short base (*right panel*) conditions for longissimus thoracis at T6 and T12, and deep multifidus (*MF*) at L4. *Epochs* in which EMG amplitude rose significantly above baseline EMG (*epoch* 0) are indicated with symbols (fracture group = *triangle*, non-fracture group = *diamond*, $P < 0.05$). The EMG normalisation approach precludes statistical comparisons of EMG amplitude within *epochs* between subject groups and between muscles; however qualitative differences between groups in response times are identified in this figure. *Error bars* represent 1 SD

displayed without reference to activity of other muscles, other biomechanical data and without knowledge of the participant group from which the data were derived. EMG amplitude was then calculated for the back muscles during 25 ms *epochs* before and after the time of onset of deltoid EMG (10 *epochs* in total). As performance of a maximum voluntary contraction was not appropriate in this population due to vertebral fragility it was not possible to normalise EMG data in this manner. Instead, EMG data were normalised to the peak activity recorded for each muscle across *epochs* in the flat base condition to allow comparison between *epochs* and bases. This type of normalisation increases the sensitivity to detect differences between *epochs* and bases but does not permit comparison between muscles or subject groups. Where EMG traces were overtly affected by motion artefact, these data were removed from statistical analysis. This occurred for 1 participant in the flat base and three participants in the short base condition.

Statistical analysis

Descriptive characteristics were compared between subject groups with independent *t* tests and Mann–Whitney *U* tests. For each group, a one-way repeated measures ANOVA was used to compare normalised EMG amplitude between *epochs* for a given base. Separate ANOVA analyses were conducted for each base. Paired *t* tests were used to compare normalised EMG between bases at three *epochs* selected a priori for each muscle: baseline, maximum, and earliest response (defined as the first *epoch* in which amplitude rose significantly above baseline). Given the exploratory nature of the study, we considered Bonferroni adjustments for post hoc comparisons to be too conservative; therefore Sharpened Bonferroni adjustments were used for multiple post hoc comparisons. The level of statistical significance was set at $\alpha = 0.05$. All statistical analyses were conducted using SPSS for Windows, version 11.0 (SPSS Inc, Chicago, IL, USA).

Results

Both groups demonstrated bursts of EMG activity before and after rapid shoulder flexion. For each muscle, normalised EMG amplitude increased above the baseline amplitude recorded during *epoch* 0 (125 ms before deltoid onset) ($P < 0.05$, Fig. 1) at some point during the trial as determined by the ANOVA. The time to initiate a postural response, defined as the *epoch* in which EMG amplitude increased significantly above baseline ($P < 0.05$), identified with post hoc comparisons, differed between the groups.

Flat base condition

At T6, EMG amplitude increased significantly above baseline during *epoch* 5 (25–0 ms before deltoid onset) in both groups. At T12, EMG amplitude in the non-fracture group increased in *epoch* 4 (50–25 ms before deltoid onset), whereas EMG amplitude in the fracture group did not increase until *epoch* 6 (0–25 ms after deltoid onset). Similarly for multifidus, EMG amplitude in the non-fracture group increased in *epoch* 4 (50–25 ms before deltoid onset), whereas EMG amplitude in the fracture group did not increase until *epoch* 5 (25–0 ms before deltoid onset).

Short base condition

At T6, EMG amplitude in the non-fracture group increased in *epoch* 4 (50–25 ms before deltoid onset), whereas EMG amplitude in the fracture group did not increase until *epoch* 5 (25–0 ms before deltoid onset). At T12, EMG amplitude in the non-fracture group increased in *epoch* 3 (75–50 ms before deltoid onset), whereas amplitude in the fracture group did not increase until *epoch* 5 (25–0 ms before deltoid onset). The EMG amplitude increase above baseline in multifidus showed an opposite pattern; EMG activity in the fracture group increased earlier (*epoch* 4: 50–25 ms before deltoid onset) than in the non-fracture group (*epoch* 5: 25–0 ms before deltoid onset).

Time to reach maximum EMG amplitude

The time to reach maximum amplitude differed between groups in the short base condition for each muscle, and only at T12 for the flat base condition (Table 2). In the short base condition, participants with fractures reached maximum amplitude in *epoch* 6, compared to the non-fracture group who reached maximum in *epoch* 7. In the flat base condition both groups reached maximum amplitude at in *epoch* 7 for T6 and *epoch* 6 for multifidus. At the T12 level, the fracture group reached maximum amplitude during *epoch* 6, while the non-fracture group reached maximum in *epoch* 7 during the flat base condition. See Table 2 for a summary of these results.

Comparison between bases

For both groups there was no difference in normalised maximum EMG amplitude or earliest response EMG amplitude between bases for any muscle. Baseline EMG was significantly lower in the short base condition for the fracture group at T6 ($P = 0.029$). No other differences were apparent for baseline EMG between bases at T12 or multifidus for both groups.

Discussion

This study provides evidence that there is a differential pattern of paraspinal muscle recruitment between individuals with and without osteoporotic vertebral fractures and these changes are present at commonly fractured levels in the mid-thoracic spine and thoracolumbar junction. This finding may partly help to explain the complex and worrying clinical problem of the ‘vertebral fracture cascade’. Most notably, individuals who had sustained a vertebral fracture

Table 2 Summary of epoch data for both groups at longissimus thoracis (*LT*) at T6 and T12, and deep lumbar multifidus (*MF*) at L4

	Time to increase above baseline				Time to reach max amplitude			
	Flat base		Short base		Flat base		Short base	
	Fracture	Non-fracture	Fracture	Non-fracture	Fracture	Non-fracture	Fracture	Non-fracture
LT-T6	5	5	5	4	7	7	6	7
LT-T12	6	4	5	3	6	7	6	7
MF-L4	5	4	4	5	6	6	6	7

demonstrated delayed activation and a shorter time to reach maximum amplitude of the paraspinal muscles compared to individuals with no history of vertebral fracture.

The rapid arm movement paradigm used in this study provides an opportunity to investigate the strategy implemented by the central nervous system (CNS) to manage a sudden change in trunk equilibrium [3, 22]. The differential neuromuscular responses observed may be associated with greater vertebral loading in the fracture group given that muscle force is delivered over a shorter time, and thus point to a mechanism underlying the vertebral fracture cascade. However, the decision by the CNS to adopt this neuromuscular response may also be an adaptive/protective strategy. The longer time to initiate a response and shorter time to reach maximum amplitude may highlight a mechanism aimed at minimising the duration of vertebral loading. Further studies using a detailed anatomic model driven by EMG would be required to clarify the nature of these loading strategies.

A consistent pattern of activity was observed in longissimus thoracis during the arm movement task; onsets of T6 and T12 EMG activity occurred 25–50 ms after those of the non-fracture group, except in the flat base condition for T6 in which both groups demonstrated a significant rise in EMG activity above baseline at *epoch* 5. Results of this study are consistent with those reported previously using surface EMG in individuals with osteoporotic vertebral fractures [14]. That study demonstrated a delay in activation of the erector spinae muscle at T7 in individuals with vertebral fractures by 50 ms, and a reliance on trunk muscle co-contraction to maintain equilibrium. Co-contraction contrasts to the alternating trunk muscle activity patterns reported in younger populations during similar tasks [2].

Trunk muscle activity may be regarded as 'feed-forward' if onsets occur between 100 ms before to 50 ms after the onset of deltoid [21]. Thus, results of the present study agree with previous reports that suggest a feed-forward pattern for erector spinae and multifidus activation relative to deltoid onset [3, 17, 21, 23, 47]. A major element of the present study is that EMG recordings were made at commonly fractured sites, and from the paraspinal muscles, which are known to contribute significantly to compressive vertebral loading due to their short moment arm, particularly in individuals with vertebral fractures [13]. Although EMG was not collected from participant-specific fracture levels, the majority of fractures sustained by participants in this study occurred at T6, in agreement with previous reports [7, 11].

For multifidus, the onset of EMG activity in the non-fracture group preceded that of the fracture group in the flat base condition; however the opposite pattern was noticed in the short base condition. Consistent with previous research, the deep multifidus was active prior to deltoid [31]. The reason for earlier activation of the multifidus in the fracture group of 25 ms during the short base condition is uncertain; however we propose three possible explanations. First, it may be that individuals with osteoporotic vertebral fractures experience greater spinal instability therefore requiring a more rapid activation of the multifidus muscle compared to those without fractures. Second, the earlier response of multifidus may be necessary to accommodate for the delayed response of the more superficial long erector spinae muscles in the thoracic spine. Third, EMG of the lumbar multifidus was collected at L4 and vertebral fractures rarely occur at this level. Findings presented in this study may indicate that neuromuscular changes in the trunk extensors occur specifically at commonly fractured levels or that a CNS adaptation has occurred in the fracture group to increase lumbar intersegmental stability by recruiting multifidus relatively earlier.

As expected, muscle responses varied according to the task. In general, paraspinal muscles were recruited earlier in the short base condition (75–50 ms) compared to the flat base condition (50–25 ms), although little difference was observed in EMG amplitude between bases. This may reflect a greater demand placed on the CNS in the short base condition that required more rapid activation of the paraspinal muscles. On a flat base, the body rotates as a rigid mass about the ankle joints to maintain equilibrium in response to sagittal plane perturbations [24]. The short base decreases the ability for individuals to use an ankle strategy (ankle torques) to maintain postural control, and equilibrium is maintained by generation of horizontal shear forces from hip and trunk movement [24]. Muscular responses in the trunk therefore become more pronounced. Indeed, difficulty in executing postural tasks, particularly involving balance, has been reported previously in the elderly population [46].

The delay in recruitment of the paraspinal muscles and its likely consequence, a shorter time to reach maximum amplitude, may have several implications. A previous study showed greater segmental loading in upright stance in individuals who had sustained a vertebral fracture [6]. Combining higher static vertebral loads with a higher loading rate may be sufficient to cause vertebral failure by increasing trabecular strains [25]. Alternatively, cyclic repetitions of this neuromuscular response may fatigue trabecular bone

and accelerate disc degeneration, thereby increasing subsequent fracture risk [8, 36]. However, the neuromuscular contribution to these degenerative mechanisms may only be viewed as speculative at this time given the current knowledge in the literature. Importantly, the generally shorter time to reach maximum amplitude displayed by the fracture group may represent a compensatory strategy employed by the CNS to overcome the delay in activation and maintain trunk equilibrium, or minimise the duration of muscle loading.

The mechanisms explaining the delayed paraspinal muscle activity in the fracture group are uncertain. Inhibition of muscle function due to pain may be attributable to symptomatic fractures, while subtle changes in thoracic kyphosis may have altered the mechanical properties of the muscles [39]. Previous research has confirmed changes in muscle recruitment as a consequence of pain [19, 21, 23, 48]. Other factors related to vertebral fractures such as decreased mobility and fear of falling could also influence muscle activation characteristics [35]. Furthermore, individuals with vertebral fractures demonstrate lower back-extensor and systemic strength compared to individuals without fractures [9, 42]. In the presence of weakened musculature a more rapid response to reach maximum amplitude may be required to in order to satisfy the equilibrium requirements. Indeed, this hypothesis may help to explain the reduced risk of subsequent vertebral fracture seen after a programme of back-extensor strengthening [40].

Previous studies have established that back-extensor strengthening, orthoses and proprioceptive re-education are beneficial in reducing the risk of osteoporotic vertebral fractures [39]. However, care should be taken when prescribing paraspinal-strengthening exercises in order to minimise compression forces through already weakened vertebrae, and orthoses should not replace the role of active muscles in the long-term to avoid muscle deconditioning. The findings presented in this study have clinical significance and may help to optimise musculoskeletal rehabilitation for this population. This study provides evidence of the existence of altered neuromuscular patterns in individuals who have sustained vertebral fractures compared to those who have no history of vertebral fracture and this may be interpreted as one of the sequelae of vertebral fractures. Future research examining the efficacy of interventions directed towards modifying this neuromuscular response and the longitudinal efficacy in reducing fracture risk is therefore warranted. Neuromuscular retraining in individuals with low back pain has proved to be effective in reducing pain and improving function [34], thus benefits, particularly a reduction in the vertebral fracture cascade, may be seen in the population of individuals with osteoporotic vertebral fractures. However, we cannot be sure whether changing the response will decrease fracture risk as it not yet known whether the altered neuromuscular responses are an adaptive strategy employed by the CNS. The cross-sectional design of the study precludes a cause-effect inference between an altered neuromuscular strategy and vertebral fracture, thus future research should adopt a longitudinal design to overcome this limitation. Future research should also utilise biomechanical trunk models driven by EMG to elucidate the influence of neuromuscular strategies on vertebral loading in this population. Temporal activation in this study was limited to specific *epochs*, thus more specific information might be obtained from identifying accurate onset/offset times.

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