

Effect of trunk flexion speed on flexion relaxation of erector spinae

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Flexion relaxation of erector spinae (ES) has mainly been studied during slow trunk movements. Due to the viscoelastic properties of spinal ligaments, the ES activity may change at different movement speeds. Twenty-one normal females (20-25 years) were examined during slow, intermediate and natural speeds of trunk flexion. The movements and simultaneous ES surface EMG recordings were recorded by two synchronised video cameras. The ES relaxed at approximately 80 per cent of vertebral flexion at each speed, and no difference was found among the three speeds. This implies that either ES activity is independent of speed in the slow to natural functional speed range, or the flexion relaxation phenomenon is not related to changes in spinal ligamentous tension.

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The erector spinae (ES) muscle group is not active throughout the entire trunk flexion range; it suddenly relaxes towards the end of flexion and then remains quiescent throughout the rest of the flexion range (Floyd and Silver 1951 and 1955, Kippers and Parker 1983 and 1984, Okada 1970, Pauly 1966, Portnoy and Morin 1956, Schultz et al 1985, Sihvonen et al 1988 and 1991, Tani and Masuda 1985, Wolf et al 1991). The position at which the ES relaxes has been referred to as the position of flexion relaxation, or the critical position (Floyd and Silver 1951 and 1955, Kippers and Parker 1984).

Most authors have explained the critical position by assuming that the ES and passive posterior spinal structures work in tandem to support the spine during flexion. Tension in the spinal ligaments and connective tissue component of the ES increases with trunk flexion. Towards the end of flexion range, passive tension in these structures may be sufficient to counter-balance the upper body load, and therefore allow the muscles to relax (Floyd and Silver 1951 and 1955, Kippers and Parker 1984, Portnoy and Morin 1956).

Previous studies have tested subjects moving at slow, non-functional speeds. In the studies of Floyd and Silver (1951 and 1955) and Tani and Masuda (1985), subjects performed full trunk flexion over a six second period. The subjects in the study of Portnoy and Morin (1956) performed trunk flexion

over five seconds, whilst Kippers and Parker (1983 and 1984) tested the trunk flexion and extension cycle over a 10 second period. The validity of these findings is questionable when applied to the situations in which the movement occurs at a natural functional speed.

Animal studies have shown that structural stiffness of ligaments increased with the strain rate (Crowinshield and Pope 1976, Haut and Little 1969, Noyes et al 1974, Peterson and Woo 1986). Most ligaments in the spine are structurally similar to the ligaments of the limbs, except the ligamentum flavum, which has a high percentage of elastin, and these spinal ligaments are believed to exhibit the same viscoelastic behaviour as ligaments of the limbs.

An increase in posterior spinal ligament stiffness associated with faster elongation during natural speed trunk flexion will cause tension to develop at a higher rate. The faster development of tension could allow the ES to relax earlier in the flexion range. This may be to the detriment of the spinal ligaments, as these structures will be subjected to a higher level of tension for longer periods.

To date, no study has been performed to examine the behaviour of ES during trunk flexion at a normal functional speed. Therefore the present study aimed to test the effect of trunk flexion speed on the flexion relaxation of ES.

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Method

Twenty-one normal female volunteers from the student population of La Trobe University, Melbourne, with a mean age of 21.2 years (range 20-25 years) were tested. People with known medical problems, histories of spinal or hip surgery, or episodes of back or hip pain requiring treatment within two months prior to the study were excluded. All subjects gave written consent before being tested and the study was approved by the Human Research Ethics Committee at La Trobe University.

The methodology of this study was based on that developed by Ng and Walter (1995). After suitable skin preparations (Gilmore and Meyers 1983), surface electrodes (Qantec 800, 810 and 820) were applied at the level of L3 on each subject, 3cm from the midline on both sides over the bulk of the ES musculature. The amplifier pass band was set at 10-500Hz and the raw EMG signals were displayed on a dual channel oscilloscope (Kenwood 50MHz, CS-5155).

The effect of trunk flexion speed on the critical position was examined by comparing the trunk and vertebral flexion angles across three speeds. Subjects were instructed to take five seconds to complete the slow speed trunk flexion movement and three seconds for the intermediate speed movement. The third speed was a natural self-selected speed which took approximately one second for most subjects. A metronome set at one beat per second was used to help subjects control their movement during the slow and intermediate speed trunk flexion.

The trunk angle (TA) was calculated as the angle between a line joining the L1 spinous process and the posterior superior iliac spine (PSIS) and a line joining the PSIS to the lateral epicondyle of the knee. The vertebral angle (VA) was calculated as the angle between lines joining L1 to PSIS and PSIS to the anterior superior iliac spine (ASIS) (Figure 1). These bony

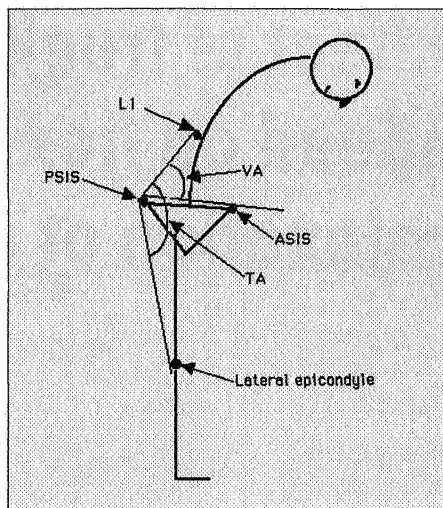


Figure 1. The calculation of trunk angle (TA) and vertebral angle (VA) during trunk flexion. The symbols L1, ASIS and PSIS stand for the first lumbar spinous process, anterior and posterior superior iliac spine respectively.

landmarks were identified by careful palpation and small spherical reflective markers were attached prior to testing.

Subjects stood with feet shoulder width apart against a video camera (National F10 CCD) positioned 2.5 metres to the right of the subject and centred at the ASIS marker to record the movement. The EMG signals displayed on the oscilloscope were also recorded by a second video camera (National A2). These cameras were linked to a video signal mixer which produced a split screen image. The EMG signal was displayed on one half of the screen and the simultaneous position of the subject was displayed on the other half. The split screen image was recorded on HS-VHS tapes using a National J11 video recorder, and the video tapes were replayed on a professional editing system for still picture analysis. A computer program was used to measure the body positions by calculating the angle between three selected points on the screen. The video image and computer screen were superimposed and displayed on a video monitor (Sony Kx21 "profeel") for analysis.

Each subject performed three complete trunk flexion movements at each speed with one to two minutes rest between each movement. The order of tests was randomised. The VA and TA were measured at erect standing, at the onset of the critical position, and at full flexion. The critical position was determined by replaying the video in slow motion so that the sudden decrease in the amplitude of the EMG signal could be detected. This was very sharp and reproducible in all subjects. When the critical position was identified, the image was frozen and the two angles were measured. Measurements were made on all the recordings for each subject and the mean value for TA and VA at each speed was calculated.

Results

The VA and TA at the critical position were expressed as percentages of the respective angles at full trunk flexion, to eliminate the effect of difference in flexibility among subjects. Since the percentage data of VA and TA were not normally distributed, the non-parametric Friedman's test was used to compare between the different speeds. The α level was set at 0.05.

The Friedman's tests did not reveal significant difference among the three speeds for both the VA and TA (Table 1). The critical position occurred between 74.0 and 74.2 degrees of vertebral flexion and between 109.3 and 112.7 degrees of trunk flexion.

Discussion

The present study has confirmed previous findings that ES activity decreases towards the end of trunk flexion (Floyd and Silver 1951 and 1955, Kippers and Parker 1983 and 1984, Pauly 1966, Portnoy and Morin 1956, Schultz et al 1985, Tani and Masuda 1985, Wolf et al 1991). Furthermore, there was no significant difference in vertebral or trunk flexion angle at the critical position among the three test speeds.

The finding that movement speed did not affect the critical position suggests two possibilities. Studies reporting

Table 1.
The means and standard deviations (in brackets) of the vertebral angle (VA) and trunk angle (TA) and the percentages of these angles at the critical position with respect to the full vertebral and trunk movements for each speed during trunk flexion.

	Slow	Intermediate	Fast	p values
VA at critical position (degree)	74.0 (5.3)	74.2 (4.6)	74.0 (5.5)	0.98
% full VA at critical position	78.3 (20.2)	82.9 (7.4)	78.1 (13.6)	0.47
TA at critical position (degree)	111.7 (15.7)	112.7 (14.0)	109.3 (13.8)	0.56
% full TA at critical position	68.9 (19.2)	67.0 (15.3)	68.2 (15.2)	0.72

changes in ligament stiffness with rate of elongation have used strain rates in the vicinity of 66 to 500 per cent per second (Crowninshield and Pope 1976, Noyes et al 1974, Peterson and Woo 1986), with the changes being most profound at higher strain rates. It is possible that in the present study, even the fastest testing speed might not have been high enough to alter the stiffness of the spinal ligaments, despite this speed being approximately 500 per cent faster than the slowest speed. However, this does not negate the possibility that high speed movement may affect the critical position, since there are situations in which people flex their trunk at very high speeds. Investigation of ES activity at these high speeds may yet reveal speed related changes.

Another possible reason is that the proposed explanation for the critical position, based on the assumption that spinal ligamentous structures and ES work in tandem during flexion, is incorrect. On the basis of the present data, it cannot be determined which explanation is true. The finding that ES activity is similar between natural and slower speeds of trunk flexion suggests that the neuromotor control for this movement is independent of the speed in the present tested range. It is suggested that future studies of the critical position should investigate the

more natural movement speed. This will eliminate the inconsistent spinal kinematics sometimes associated with slow speed movements.

Conclusion

The present findings indicate that the positions of the vertebral column and trunk of normal healthy subjects at the critical position were not affected by the speed of movement in the range tested. This suggests that either the natural speed of trunk flexion is not fast enough to alter the stiffness of the spinal ligaments, or the current explanation for the critical position is incorrect. This needs to be investigated with further studies.

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