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Abstract: Background context
The kinematics of the lumbar region and the activation patterns of the erector spinae muscle have been associated to the genesis of low back pain, which is one of the most common complications associated to pregnancy. Despite the high prevalence of pregnancy-related low back pain, the biomechanical adaptations of the lumbar region during pregnancy remain unknown.
Purpose
This study analyses lumbar spine motion and the activation pattern of the lumbar erector spinae muscle in healthy pregnant women.
Study design
Case-control study.
Patient sample
The study involved 34 nulliparous women (control group) and 34 pregnant women in the third trimester (week 36 ± 1).
Outcome measures
We recorded the parameters of angular displacement of the lumbar spine in the sagittal plane during trunk flexion-extension, and the EMG activity of the erector spinae muscles during flexion, extension, eccentric and concentric contractions and the myolectrical silence.
Methods
The participants performed several series of trunk flexion-extension movements, which were repeated two months postpartum. The position of the lumbar spine was recorded using an electromagnetic motion capture system. EMG activity was recorded by a surface EMG system, and expressed as a percentage of a submaximal reference contraction.
Results
Antepartum measurements showed a decrease (relative to control and postpartum measurements) in lumbar maximum flexion (52.5± 10.5° vs 57.3±7.7° and 58.7±8.6°; $p<0.01$), the percentage of lumbar flexion during forward bending (56.4± 5.6% vs 59.4± 6.8% and 59.7±5.6%; $p<0.01$) and the time keeping maximum levels of lumbar flexion (35.7±6.7% vs 43.8±5.3% and

50.1±3.7%; $p<0.01$). Higher levels of erector spinae activation were observed in pregnant women during forward bending (10.1±4.8% vs 6.3±2.4% and 6.6±2.7%; $p<0.01$) and eccentric contraction (12.1±5.2% vs 9.4±3.1% and 9.1±2.9%; $p<0.01$), as well as a shortened erector spinae myoelectric silence during flexion.

Conclusions

Pregnant women show adaptations in their patterns of lumbar motion and erector spinae activity during trunk flexion-extension. These changes could be associated to the genesis of pregnancy-related low back pain, by means of biomechanical protection mechanisms against the increase on abdominal mass and ligamentous laxity.

Effects of pregnancy on lumbar motion patterns and muscle responses

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Antepartum measurements showed a decrease (relative to control and postpartum measurements) in lumbar maximum flexion (52.5± 10.5º vs 57.3±7.7º and 58.7±8.6º; $p<0.01$), the percentage of lumbar flexion during forward bending (56.4± 5.6% vs 59.4± 6.8% and 59.7±5.6%; $p<0.01$) and the time keeping maximum levels of lumbar flexion (35.7±6.7% vs 43.8±5.3% and 50.1±3.7%; $p<0.01$). Higher levels of erector spinae activation were observed in pregnant women during forward bending (10.1±4.8% vs 6.3±2.4% and 6.6±2.7%; $p<0.01$) and eccentric contraction (12.1±5.2% vs 9.4±3.1% and 9.1±2.9%; $p<0.01$), as well as a shortened erector spinae myoelectric silence during flexion.

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genesis of pregnancy-related low back pain, by means of biomechanical protection mechanisms against the increase on abdominal mass and ligamentous laxity.

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Introduction

Pregnancy is a physiological period of reproduction, which involves a series of changes in the various systems of the mother's body, preparing it for embryonic development, fetal growth and childbirth. Throughout the process of gestation the lumbar spine has to adapt gradually to increasing burdens which in the end virtually always cause some form of musculoskeletal discomfort [1]. One of the most common complications of pregnancy is low back pain (LBP), with a prevalence between 40 and 80% [2-5]. Despite its high prevalence, the etiology of LBP associated with pregnancy remains unknown. It is supposed to be caused by a combination of biomechanical and hormonal factors [6]. The increased levels of relaxin during pregnancy, for instance, significantly increase ligamentous laxity [7-9]. Regarding biomechanical factors, some movements, such as trunk flexion-extension, have been related to the appearance of LBP. There are numerous studies on lumbar biomechanics during trunk flexion-extension in both healthy and LBP subjects, but there is little evidence about its behavior in pregnant women. A few studies have analyzed the relationship between the progressive enlargement of the abdomen during pregnancy and the mobility of the lumbopelvic region [10-12], and there has been some speculation about how the gradual increase in loading of the abdominal region during pregnancy could have an adaptive effect on the strength and endurance of the lumbar muscles [13]. As for the dynamic function of the lumbar region, there are hardly any studies analyzing the behavior of the trunk musculature in this population. So far, only Sivhonen et al. [14] have used the pattern of activation of the erector spinae (ES) during trunk flexion and extension in pregnant women, but only to investigate possible correlations between average muscle activity during flexion-extension and measures of pain intensity. The authors neither included a control group of nulliparous women in their study, nor recorded muscle activity after childbirth, so any changes caused by pregnancy are unknown.

Such changes in lumbar muscle responses could be behind LBP production. In fact, they could also be a target for LBP treatment, as they already are in non-pregnant subjects [15]. However, the alterations caused by pregnancy on the patterns of lumbar motion and muscle activation are unknown, even in healthy pregnant women. In order to shed more light on this subject, the aim of this study was to analyze the movement of the lumbar spine and the pattern of activation of the lumbar ES during trunk flexion-extension in healthy pregnant women. Our hypothesis was that the changes that occur during pregnancy alter the kinematics of the lumbar region and the patterns of activation of its muscles, in ways that could be related to LBP production.

Methods

Ethical approval

This research was approved by the local Corporate Ethics Committee for Clinical Research in Primary Care, and the University Ethics Committee on Human Research. All the procedures were conducted in accordance with the principles of the World

Medical Association's Declaration of Helsinki and all participants provided their written informed consent.

Participants

68 women participated in the study. The pregnancy group was composed of 34 pregnant women, either in their first or second pregnancy, all in their third trimester, when biomechanical changes are more evident (age: 34.7 ± 3.1; weeks of gestation: 36 ± 1). 34 nulliparous women formed the control group (age: 32.9 ± 4.9). Three groups were assessed, even though the same women were in two of them. Groups were the control (not pregnant) participants; the pregnant participants; and two months later, the formerly pregnant participants assessed postpartum.

Exclusion criteria for both the control and pregnancy groups were: (1) past or present LBP, intense enough to cause work absence for illness, (2) spinal pathology (fractures, scoliosis, spondylolisthesis, spondylolysis, neoplastic processes, infections, vascular, metabolic or endocrine), (3) significant lower limb-length discrepancy and / or (4) past low back surgery. Additional exclusion criteria for the pregnancy group were: (5) LBP associated with the current pregnancy and (6) any risk of abortion or any complications related to pregnancy.

Instrumentation

The angular displacement of the lumbar spine in the sagittal plane was recorded using a Liberty 240/16 electromagnetic motion capture system (Polhemus Inc., Colchester, USA). This apparatus uses a low frequency magnetic field generated by an electromagnetic source, which is placed in a plastic platform adjusted at hip level for each participant. Two sensors detect the magnetic pulses, with a sampling frequency of

240 Hz. The first sensor (L1) is attached to the skin overlying the spinous process of the first lumbar vertebra, and provides data on the displacement of the trunk as a whole (lumbar and pelvis). The second sensor (S1) is placed at the level of the first sacral vertebra, and provides data on the inclination of the sacrum at the coxofemoral joint [16]. Subtracting the S1 data from the L1 data gives the true lumbar spine motion [12, 17].

The EMG activity was recorded by an EMG100C Biopac module (Biopac Systems, Inc., Goleta, CA), using pre-gelled disposable silver-silver chloride (Ag/AgCl) surface disk electrodes (2 cm diameter). Prior to EMG electrode placement, the registration points of the activity of the ES were located following the recommendations of the Surface Electromyography for Non-Invasive Assessment of Muscles (SENIAM) project [18], at the third lumbar vertebra, with electrodes placed 3 cm to the right of the spinous process. A reference electrode was placed at the level of the sternal body. After carefully cleaning and lightly abrading the skin with an alcohol pad, 2 recording electrodes were attached parallel to the underlying muscle fibers, with a center-to-center distance of 2 cm. The raw EMG signal was band-pass filtered (cutoff frequencies: 10 Hz high pass, 500 Hz low pass) and amplified (input impedance greater than 100 MΩ, common mode rejection ratio of 110 dB at 60 Hz, overall gain of 1000). EMG signals were A/D converted at a sampling frequency of 1000 Hz with a 16-bit data acquisition system (model MP150; Biopac Systems Inc.). A synchronic signal generated at the beginning of each measurement simultaneously sent a "start recording" order to both the electromagnetic motion capture system and the EMG recorder.

Measurements and data processing

Age, height, weight and body mass index of all participants were recorded, as well as waist circumference before and after delivery in the pregnancy group.

Women stood barefoot on 2 markers on the floor with their feet pelvis-width apart, knees straight, arms by their sides, and with their hands facing inwards. At the start and end of each cycle, they stared at a mark set at eye level on the opposite wall. Individuals were told to move as far in the sagittal plane as possible following these instructions: bend neck, place chin on chest, then bend forward and try to flex as far as possible, rest for a second at maximum flexion and then return to the starting position. The subjects slipped their hands down and up the front of their lower limbs to prevent their upper limbs from swinging, and a metronome set at one beat per second marked the pace of the movements. Trunk flexion took four seconds, followed by a one-second pause at maximum flexion and four seconds of trunk extension. Five repetitions of the trunk flexion-extension cycle were performed. Trial runs allowed subjects to practice the movements, and two experienced observers monitored the subjects' actions during testing. When the instructions had not been correctly followed (exertion, rhythm, range of movement), the cycles were repeated. All tests were carried out at least two hours after the subjects had risen from bed in order to minimize diurnal variations in their spinal mechanics [19].

EMG data were rectified and smoothed by calculating their root mean square, with a time window of 0.02 seconds. They were then normalized as a percentage of that muscle's peak activity during a submaximal voluntary contraction, which has been found to be more reliable than maximum voluntary contractions for trunk muscle EMG normalization [20]. Prior to the standing position recordings, all the subjects performed a Biering-Sørensen maneuver (an isometric trunk extension with the upper body suspended in the air in front of the edge of a Roman chair) to normalize the ES signals [21].

The degrees of spine flexion were normalized as percentages of the maximum spine flexion during each flexion-extension cycle to make comparisons on the same scale as the EMG data, and to allow motion measurements to be compared between subjects. Lumbar flexion and EMG percentages were plotted, which resulted in relative motion and EMG activity curves representing the process of the movements (Fig. 1).

In order to compare between curves, several summary measurements were calculated for each of the three central flexion-extension cycles [22], and these were subsequently averaged. To describe lumbar motion during trunk flexion-extension, we analyzed the maximum range of lumbar flexion, the average percentage of lumbar flexion during both flexion and extension, and the percentage of flexion-extension time during which the lumbar spine was flexed over 90% of its maximum flexion [23]. To describe the activation pattern of the ES we analyzed the average percentage of ES activation during flexion, extension, eccentric and concentric contractions of EMG activity and the myolectrical silence. The start and end of flexion, extension, and EMG activation contraction were identified by visual inspection of the EMG curves [24, 25], using a threshold method [23, 26]. Other summary measurements were the lumbar postures at the start and end of ES relaxation, and a flexion-extension ratio between the average ES activity at trunk flexion and its average activity at extension, to quantify the ES relaxation level [27].

MATLAB 2010a (MathWorks Inc., Massachusetts USA) was used for all data analysis.

Statistical Analysis

An a priori analysis of effect and sample size was made for a desired power of 90%. Effect size was estimated by means of Cohen's d, calculated from the results of published work which calculated similar dependent variables (EMG activity of the ES, lumbar position in the sagittal plane) in pregnant and non-pregnant women [11,14]. Sample size was calculated using the G*Power 3 software [28]. The result was an estimated minimum sample size of 32 subjects per group.

Compliance with the assumption of normality was checked for each dependent variable and each study group by means of the Kolmogorov-Smirnov test. In order to check the homogeneity in age and height between the control and pregnancy groups, a Student t-test for independent samples was used, with an alpha level of 0.05. Differences between the control and pregnancy groups, both ante and postpartum, were also analyzed by means of the Student t-test for independent samples. Differences between measurements in pregnant women before and after childbirth were analyzed by means of the Student t-test for related samples. To avoid the inflation of type I error due to repetition of pairwise comparisons, a Bonferroni adjustment was applied to the level of significance of these three comparisons (control vs postpartum, control vs antepartum and antepartum vs postpartum). Thus the alpha level for these three comparisons was 0.05/3, that is, 0.016.

Ante-postpartum differences in abdominal circumference and percentages of muscle activation and lumbar flexion were calculated. To search for possible associations between changes in waist circumference and changes in lumbar flexion and muscle activation, Pearson correlation coefficients were calculated, with a significance level set at $p<0.05$.

SPSS version 18.0 for Windows (SPSS Inc, Chicago, IL, USA) was used for all analysis.

Results

Table 1 shows age, weight, height, body mass index and waist circumference of all the study groups. No significant differences in age and height were found between women in the control and pregnancy groups. Weight and body mass index were significantly lower in the control and postpartum groups respect to the pregnancy group ($p < 0.01$), and abdominal circumference was higher in the antepartum respect to the postpartum measurements ($p < 0.01$), as expected due to pregnancy-related changes in mass and body composition.

In the pregnant women group, there was a significant decrease in the maximum range of flexion, average lumbar flexion during the flexion phase and the percentage of flexion-extension time during which the lumbar spine was flexed by 90% or more of maximum flexion, when compared to nulliparous women and postpartum ($p<0.01$) (table 2). The results showed that, after childbirth, pregnant women reached values similar to those presented by nulliparous women in these 3 variables. There were no significant correlations between the change in waist circumference and changes in the kinematic parameters.

Pregnant women showed a significant increase in muscle activation during the flexion phase and the eccentric contraction of muscle activity, and also a higher flexion-extension ratio than control and postpartum groups ($p < 0.01$). All nulliparous women showed the FRP. Two antepartum and two postpartum recordings showed an absence of FRP, but they came from four different women. Pregnant women reached the beginning of the FRP with a percentage of lumbar flexion greater than that of the control and postpartum groups ($p<0.01$) (table 3). A significant but low positive correlation ($r = 0.44$; $p <0.05$) was observed between ante-postpartum waist circumference variation and the ante-postpartum variation of the average activity during the eccentric activation of the ES.

Discussion

This study is the first to describe the changes caused by pregnancy in the kinematics and neuromuscular responses of the spine. We have found alterations both in lumbar motion and muscular activation of the trunk extensor muscles.

Pregnant women showed a lower maximum range of lumbar flexion compared to nulliparous and postpartum women. To our knowledge, only a few studies have analyzed trunk ranges of motion in pregnant women [10, 11], and they agree with our results. The reduction of the maximum range of lumbar flexion could be explained by the restriction to movement caused by the expanded abdominal volume. However, no correlation between waist circumference and maximum lumbar flexion was observed.

This lack of correlation suggests that abdominal volume may not be the only factor associated to the restricted ranges of flexion. In fact, our study showed that pregnant women not only reach lower maximum ranges of lumbar motion than the other two groups, but also flex less their lumbar spine during the course of trunk flexion, and hold maximum lumbar flexion values for a shorter time. It would seem that pregnant women are exhibiting a slow, guarded pattern of lumbar motion during flexion. These same results have been observed in patients with LBP [23, 29-34]. The increased abdominal mass of pregnant women causes an increase of bending moment. It is known that the greater the degree of trunk flexion, the greater the risk of injury, given that bending moment is higher at the end ranges of trunk flexion and is one of the main causes of injury to spinal connective tissues [35]. One possible explanation for our findings could then be that the altered pattern of lumbar motion in pregnancy is part of a spinal protective strategy against the increased abdominal mass, in the same way that patients with LBP develop protective strategies for their damaged spinal structures.

This explanation agrees with the changes we observed in the contraction patterns of the ES. Our results showed a greater percentage of ES activity during the flexion phase and the eccentric activation peak in the group of pregnant women. There is very scarce scientific evidence about the patterns of activation of trunk muscles during pregnancy: most studies about the alterations of the neuromuscular responses of lumbar muscles have focused on LBP patients. Patients with LBP show an increased ES activation during trunk flexion, when compared to healthy people [27, 29, 36], even during pain-free periods [23]. In pregnant women, the increased abdominal mass during pregnancy may play a direct role on the increased activity of the ES during trunk flexion and eccentric activation. In fact, we observed a positive correlation between the ante-postpartum variation in waist circumference and the ante-postpartum variation of EMG activity during the eccentric activation of the ES.

In our results, we observed changes in the parameters of the myoelectric silence of the ES, which began later in pregnant women than in nulliparous and postpartum measurements. In non-LBP subjects, when the trunk reaches 75-85% of its maximum flexion, EMG activity of the trunk extensors ceases abruptly, entering a phase of myoelectric silence. This response is called the flexion-relaxation phenomenon (FRP) [15, 27, 30, 37, 38], and is associated to high loading levels of vertebral ligaments and intervertebral discs [39] without an active protection from the ES muscles. There are no previous studies dealing with the characteristics of the ES FRP in pregnant women. Coinciding with our results, some studies have shown a delay in the onset of myoelectric silence when trunk flexion-extension is performed while holding external loads, either dumbbells or loaded vests [24, 25, 40]. The addition of weight forces the ES to remain active for a greater range of flexion, until passive structures are stretched further, acquire enough tension and passively generate enough extensor moment to compensate the increase in bending moment. Only then the tensed passive structures can relieve the extensor muscles in their role as spinal stabilizers, thus allowing the delayed relaxation of the ES [41].

The effect of relaxin on spinal ligaments may also contribute to such delay. This hormone causes an alteration in the viscoelastic properties of ligaments and an increase of their laxity, by means of an increase in collagenolytic activity and alterations in the remodeling of collagen fibers and extracellular matrix in mammalian animals [7-9]. Some authors have observed a relationship between blood levels of relaxin and higher scores in the active straight leg raising test in pregnant women [42].

Also maintained or repeated forward flexion result in an increased deformation of viscoelastic tissues called creep, which causes ligamentous laxity [43-45]. Changes produced by creep in the stress-strain relationship of passive tissues force ligaments to achieve a greater degree of elongation so their tension is enough to compensate forward bending moment. Animal studies have shown, by inducing creep in the supraspinous ligament, that an increase in laxity alters the afferent feedback provided by ligamentous mechanoreceptors, causing a deterioration of reflex ligamentomuscular responses [43]. This creep-induced desensitization of mechanoreceptors has also been shown in humans: the increased laxity caused by creep leads to a delayed reflex activation of the ES, when subjects face an unexpected destabilization [46, 47]. In line with our results, some authors have observed an increase in the angle at which the FRP occurs when creep is induced in the passive spinal structures by means of a repetitive flexion movement of the lumbar spine [48] or a maintained static flexion [49]. Solomonow et al. [49] also observed that this increase was more pronounced in women than men. The delay in the beginning of the FRP may thus be reflecting a desensitization of mechanoreceptors as a result of increased laxity in the ligaments, caused either by creep or by hormonal factors.

The desensitization of mechanoreceptors exposes the lumbar region to further instability, making it more vulnerable to destabilizing injuries [43]. In fact patients with chronic LBP show a delay in the start of ES myoelectrical silence [50], even during pain-free periods [23]. The reduction of spinal stiffness as a result of increased ligamentous compliance seems to be related to spinal injury, maybe through the alteration of protective reflex neuromuscular responses. The shortened ES myoelectrical silence observed in our research implies that the ES remains active for longer. This increase in muscle activity could also have a protective effect, providing active support to a posterior ligamentous system with altered viscoelastic properties due to hormonal effects, and also subjected to higher loadings by the augmented abdominal mass. The coexistence of these two factors during pregnancy and their effects on the trunk motion patterns and the responses of the trunk extensor muscles, as shown in the present study, could have a direct relationship with the origin of pregnancy-related LBP. Such mechanisms of LBP production could be especially important in women who perform activities implying full trunk flexion.

Multiple interventions have been used to try to relieve LBP associated with pregnancy [51]. Exercise programs focused on the back extensor muscles are among the few that offer some evidence on their effect. A Cochrane review [52] showed that there is evidence that this type of programs reduces pain and disability in cases of LBP during pregnancy. However, the same review recognized that such evidence is of low quality and more studies are needed. The effect of this kind of exercise programs could be mediated by an alteration of the patterns of muscle activation described in the present study.

This study had some limitations. In order to achieve the calculated sample size, and given the difficulties in recruiting this kind of participants, our sample was heterogeneous in terms of the number of previous pregnancies. This limitation, however, is shared by several previous studies with lower sample sizes [11, 53, 54]. Future studies should investigate whether the number of previous pregnancies influence the position and muscle activity of the lumbopelvic region.

This study focuses on pain-free pregnant women. We have tried to argue how the alterations described on the patterns of spinal motion and muscle responses could be related to the genesis of LBP in this population. Another line of future studies should test these assumptions in groups of LBP pregnant women. It would be extremely interesting to also check if exercise programs focused on the back extensor muscles cause any effects on these patterns of motion and muscle responses.

In conclusion, pregnancy causes alterations in the patterns of lumbar motion, with a lower range of flexion and reductions both in the percentages of lumbar flexion during spine flexion and in the time pregnant women keep maximum values of lumbar flexion. These kinematic changes are coupled with changes in the neuromuscular responses of the lumbar extensor muscles, which show an increased activation during spine flexion and a shortening of their myolectrical silence. All these could represent biomechanical adaptations to the increased anterior loading and the augmented ligamentous laxity associated to pregnancy, and could be associated to the genesis of pregnancy-related LBP.

Competing interests

The authors report no competing interests for this work.

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Table 1. Descriptive characteristics of the participants

	Control group	Antepartum measurements	Postpartum measurements
n	34	34	34
Age (years)	32.9 ± 4.9	34.7 ± 3.1	35.0 ± 3.1
Weight (kg)	59.4 ± 8.6†	70.0 ± 8.7*	62.1 ± 8.7
Height (cm)	163.8 ± 5.5	163.2 ± 6.7	163.2 ± 6.7
Body mass index (kg / m^2)	22.1 ± 2.9†	26.2 ± 2.7*	24.8 ± 9.8
Abdominal circumference (cm)	-	100.5 ± 8.6*	82.0 ± 6.5

Values presented as mean ± SD. P<0.01: *antepartum-postpartum, †antepartum-control.

Table 2. Kinematic variables during trunk flexion-extension for the three study groups

	Control group	Antepartum measurements	Postpartum measurements
Maximum range of lumbar flexion *(º)*	57.3 ± 7.7†	52.5 ± 10.5*	58.7 ± 8.6
Average lumbar flexion during flexion *(% maximum range)*	59.4 ± 6.8†	56.4 ± 5.6*	59.7 ± 5.6
Average lumbar flexion during extension *(% maximum range)*	48.3 ± 5.3	51.2 ± 4.8	50.1 ± 3.7
Time of lumbar flexion over 90% of maximum *(% of flexion-extension time)*	43.8 ± 6.8†	35.7 ± 6.7*	41.4 ± 4.7

Values presented as mean ± SD. $P<0.01$: *antepartum-postpartum, †antepartum-control.

Table 3. Erector spinae variables during trunk flexion-extension for the three study groups

	Control group	Antepartum measurements	Postpartum measurements
Average activation during flexion *(% submaximal contraction)*	6.3 ± 2.4†	10.1 ± 4.8*	6.6 ± 2.7
Average activation during extension *(% submaximal contraction)*	22.8 ± 5.8	26.4 ± 6.8	25.2 ± 6.2
Flexion-extension ratio	0.3 ± 0.1†	0.4 ± 0.2*	0.3 ± 0.1
Average activation during eccentric contraction *(% submaximal contraction)*	9.4 ± 3.1†	12.1 ± 5.2*	9.1 ± 2.9
Average activation during concentric contraction *(% submaximal contraction)*	23.8 ± 5.6	26.7 ± 6.5	26.3 ± 5.4
Average activation during myolectrical silence *(% submaximal contraction)*	2.0 ± 1.2	2.2 ± 1.5	1.6 ± 0.9
% of lumbar flexion at the onset of myoelectrical silence *(% maximum range)*	83.1 ± 5.5†	87.9 ± 6.7*	84.8 ± 5.8
% of lumbar flexion at the end of myoelectrical silence *(% maximum range)*	97.7 ± 1.7	98.4 ± 2.8	98.2 ± 1.7

Values presented as mean ± SD. P<0.01: *antepartum-postpartum, †antepartum-control.

Figure(s) in .jpg, .tif, or .eps

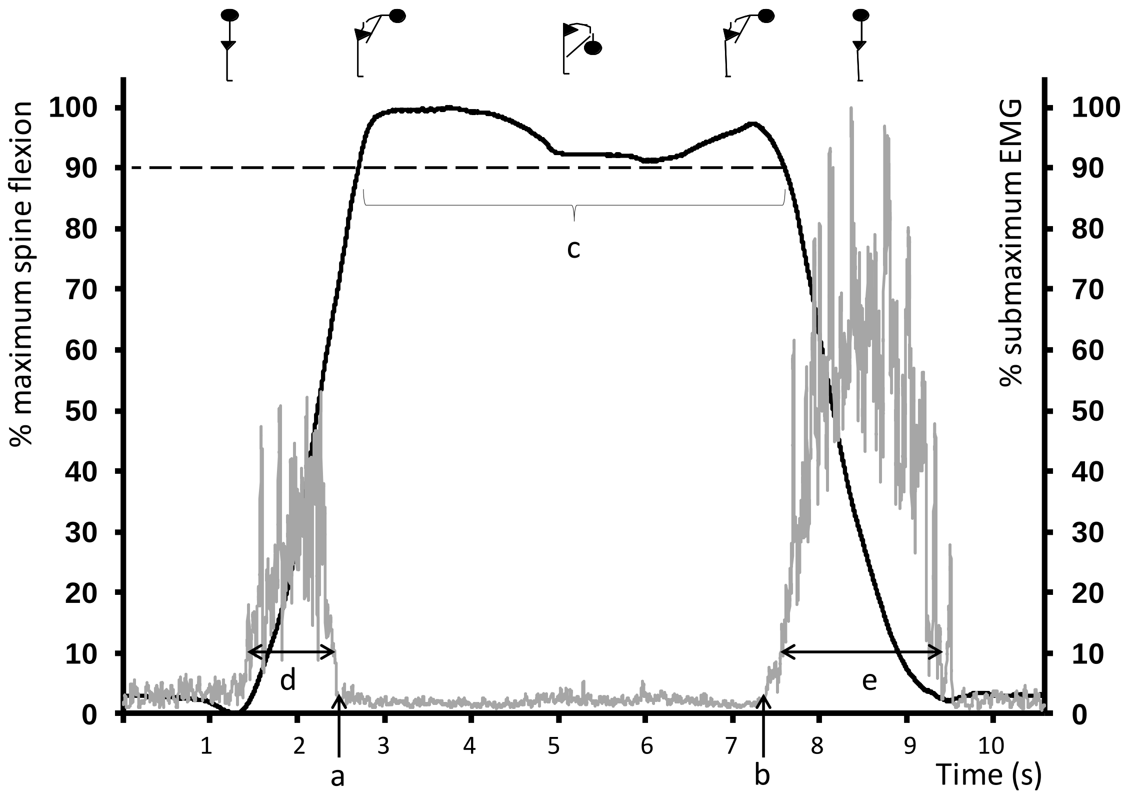

Fig 1. Representative curve of lumbar spine motion (black line) and ES activity pattern (grey line) during trunk flexion extension. (a) start of FRP, (b) end of FRP, (c) time of lumbar flexion over 90%, (d) eccentric contraction, (e) concentric contraction. EMG, electromyography