■ The Effects of Abdominal Muscle Coactivation on Lumbar Spine Stability

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Study Design. A biomechanical model of the lumbar spine was used to calculate the effects of abdominal muscle coactivation on spinal stability.

Objectives. To estimate the effects of abdominal muscle coactivation on lumbar spine stability, muscle fatigue rate, and lumbar spine compression forces.

Summary of Background Data. The activation of human trunk muscles has been found to involve coactivation of antagonistic muscles, which has not been adequately predicted by biomechanical models. Antagonistic activation of abdominal muscles might produce flexion moments resulting from abdominal pressurization. Qualitatively, antagonistic activity also has been attributed to the need to stabilize the spine.

Methods. Spinal loads and spinal stability were calculated for maximum and submaximum (40%, 60% and 80%) efforts in extension and lateral bending using a previously published, anatomically realistic biomechanical model of the lumbar spine and its musculature. Three different antagonistic abdominal muscle coactivation patterns were imposed, and results were compared with those found in a model with no imposed coactivation.

Results. Results were quantified in terms of the sum of cubed muscle stresses $(\Sigma \sigma_m{}^3$, which is related to the muscle fatigue rate), the maximum compressive loading on the lumbar spine, and the critical value of the muscle stiffness parameter (q) required for the spine to be stable. Forcing antagonistic coactivation increased stability, but at the cost of an increase in $\Sigma \sigma_m{}^3$ and a small increase in maximum spinal compression.

Conclusions. These analyses provide estimates of the effects of antagonistic abdominal muscle coactivation, indicating that its probable role is to stabilize the spine. [Key words: biomechanics, coactivation, lumbar spine, muscles, stability] **Spine 1998;23:86–92**

There are a redundant number of strategies that can be used to activate the muscles of the trunk to achieve a desired task. The greatest difference between the activation strategy predicted by biomechanical models and that measured by electromyography (EMG) is that, in the models, the degree of coactivation of synergistic and

antagonistic muscles is underestimated.[21,22,24,30,32,35,37,39,42]

This apparently suboptimal and excessive use of muscle forces increases lumbar spine loads.[8,17,22,41] Thelen et al[41] estimated that the level of coactivation measured in EMG studies increases compressive load on the spine in extension efforts by 16–19% and by a greater amount in lateral bending efforts. For dynamic extension, the calculated increase in compressive load is greater, and the estimated shear forces increased by 70% because of this coactivation.[17] Hughes et al[19] calculated that the increase in spinal compression resulting from abdominal muscle coactivation could be as much as 5.52 times as much as the force increase in the abdominal muscles.

Biomechanical models are used to identify specific lifting tasks that may produce low back pain.[21] Therefore, models that underestimate muscle coactivation also underestimate *in vivo* spinal and muscular loadings. Besides simple mechanical overload, low back pain could be initiated by damage produced when muscles are not properly activated to maintain equilibrium and stability at all spinal levels.

The coactivation of abdominal muscles in extension efforts is associated with increases in intra-abdominal pressure, which in turn generates an extension moment. However, the flexion moment resulting from the muscle forces almost cancels out this effect, casting doubt on this possible advantage of antagonistic abdominal muscle activity.[33]

It appears that the only rational reason that coactivation occurs is to stabilize the trunk against the possible destabilizing effects of an unexpected perturbation.[2,4,7,9–11,14] Anticipatory activation of trunk muscles is apparently an example of simultaneous activation of synergistic and antagonistic muscles used to stabilize the spine.[23,28] Although coactivation has been attributed qualitatively to a need to increase the spinal stability, this has not been shown quantitatively. Stability is used in this report to mean the tendency of a structure to return to its equilibrium position after a perturbation.[1,36]

Spinal and muscle forces can be studied in two ways: in analytical models to calculate muscle forces required to perform a specified task, and EMG experiments to document the *in vivo* activities of the muscles. Electromyographic-assisted models combine both approaches.[7,8,26,27,29] The trunk has a redundant number of mus-

cles compared with the minimum number required to achieve a specific task. Analytical models are used to calculate an optimized activation strategy by assuming that the neuromuscular control system activates muscles to minimize a presumed physiologic cost function. Several such cost functions have been proposed: the compressive component of the intervertebral joint force, the shear component, muscle stress, or an exponential function of muscle stress.[3,16,19,20,21,34,38] However, models with these presumed physiologic cost functions do not adequately predict antagonistic activity.

Recently, biomechanical analyses have been developed to quantify the stabilizing effect of muscle forces and muscle stiffness. Bergmark[4] introduced the idea that muscle stiffness, which increases with activation, must exceed a critical value to prevent spinal buckling. Crisco[9-11] used similar buckling analyses to investigate the relative roles of short and long muscles in stabilizing the spine. Gardner-Morse et al[14] used a more anatomically realistic model to examine the roles of varying posture and spinal stiffness on spinal stability, and the associated buckling mode shapes. These models did not include antagonistic muscle activation. Cholewicki and McGill[7] investigated spinal stability with an EMG-assisted model, which included any muscle coactivation present in the recorded EMG signals, but the additional contribution to spinal stability of the coactivation was not determined.

Because muscle stiffness increases with activation, the authors of this report hypothesize that the neuromuscular control system sets the muscle activation and coactivation to ensure lumbar spine stability.[4,11,14] This would reduce the need for active neuromuscular control system responses and their inherent time delays.

The purpose of this study was to quantify the effects on spinal stability of forcing abdominal muscle coactivation in extension and lateral bending efforts. A previously published, anatomically realistic biomechanical model of the lumbar spine and its musculature was used.[14,40]

■ Methods

Analyses of spinal stability were performed using a three-dimensional, biomechanical, quasistatic model of the lumbar spine and its musculature.[14] The geometry was specified in terms of vertebral body positions, locations of muscle attachments, and physiologic cross-sectional areas of muscle.

The dorsal muscles, abdominal wall muscles, and psoas muscles were represented by 50 symmetric muscle pairs. Origins, insertions, and physiologic cross-sectional area of dorsal muscles and the psoas were obtained from the publications of Bogduk et al.[5,6] The same geometric information was inferred for the quadratus lumborum from serial, computed tomography, sectional data published by Han et al.[18] For the internal and external obliques, area values were obtained from Han et al,[18] and the muscle paths were obtained from Dumas et al.[13]

Three simplifying assumptions were made about all the muscles: 1) muscles were assumed to take a straight-line path

from origin to insertion, 2) muscle contractile stress (force/muscle area) was assumed to be between zero (muscles produce only tension) and an upper limit of 460 kPa, and 3) activated muscles were considered to have stiffness proportional to the level of activation and inversely proportional to muscle length.[4,5]

The spinal motion segments were represented by beam elements matched to experimentally derived linear stiffness data.[15] The thorax and sacrum/pelvis were considered to be rigid bodies, and the sacrum/pelvis was constrained. A static, vertical, compressive load of 340 N was imposed 50 mm anterior to T12 to represent upper-body weight.

In a preliminary step, the magnitudes of maximum efforts were calculated by solving the muscle force distribution in a linear program, in which the external effort was the cost function to be maximized, as described by Stokes and Gardner-Morse in a previous study.[40] Subsequent analyses simulated a person performing with submaximum (40%, 60% and 80%) efforts.

In the analyses of submaximum efforts, the sum of cubed muscle stresses ($\Sigma\sigma_m^3$) was minimized as the objective function for solving the muscle force distribution. This objective is thought to be related to the endurance of an activity or rate of muscle fatigue; minimizing it would minimize the overall muscle fatigue rate.[12] This cost function also has been found to result in data that are the most similar to experimental data for spinal muscle activities.[12,20,21,34]

For maximum efforts, the solutions were constrained to satisfy static equilibrium of each vertebra, physiologic limits on muscle forces, and physiologic limits on motion-segment translations and rotations (5 mm and 5° for the sagittal plane and 2 mm and 2° for the other planes, respectively).[40] For submaximum efforts the constraints on intervertebral motions were reduced by scaling them according to the percent effort, because this was considered more realistic. All the optimization calculations were performed using MINOS (Technical Report SOL83-20, Systems Optimization Laboratory, Stanford University, Stanford, CA).

The linear lumbar spine stability analysis used the muscle and motion-segment forces from the muscle force distribution (optimization) analysis. The stability analyses involved the determination of the critical amount of muscle stiffness required to stabilize the spine under the prevailing loading.

Muscles have negligible amounts of stiffness when not activated, but activation increases muscle stiffness.[4,11,14] This stiffness is a complex function of activation, length, and rate of shortening or lengthening. It was assumed in this study that the stability of the lumbar spine (for small perturbations) depended on the short-range or cross-bridge muscle stiffness represented by the linear formulation of Bergmark[4]:

$$k_m = q\frac{T}{l_m} \tag{1}$$

where k_m represents muscle stiffness (N/mm), T represents active muscle force (N) found in the muscle force distribution analysis, l_m represents muscle length (mm), and q is a dimensionless parameter that is assumed to be the same for all muscles.

There is a wide range of published values for the muscle stiffness parameter q.[11] In the analyses in the current study, q was treated as a variable, which allowed for the determination

of the lowest (critical) value of q required to ensure stability of the loading state predicted by the force distribution analysis (*i.e.*, the value of q that produces a metastable spine). Increases in the value of the critical q represent decreases in stability, because more muscle stiffness is required to stabilize the spine. Determination of the degree of muscle stiffness critical for stability required an iterative solution of an eigenvalue buckling analysis of the lumbar spine stability. The eigenvalue buckling problem was solved using the QZ algorithm ("eig" routine in Matlab, The MathWorks Inc, Natick MA). Details of this method are given in a previously published report by Gardner-Morse et al.[14]

Extension (resisting a flexion moment) and lateral bending efforts were studied without coactivation and with three different abdominal muscle coactivation patterns. The antagonistic muscles were forced to be active by raising the lower bounds on the forces of those muscles.

Increasing levels of the following three muscle coactivation patterns were studied:

1. coactivation of internal obliques at 2%, 4%, 6%, and 8% of maximum voluntary contraction (MVC);
2. coactivation of external obliques at 2%, 4%, 6%, and 8% of MVC; and
3. coactivation of internal and external obliques at 2% and 4% of MVC.

These levels of coactivation were based on available values from the literature. Thelen et al[41] reported that approximately 20% of MVC coactivation of internal and external obliques occurred during maximal exertions in extension and lateral bending. Therefore, a lesser degree of activation was considered for the submaximal efforts analyzed in the current study. Lavender et al[22] reported negligible coactivation of external obliques for small extension efforts and approximately 10% of MVC values in lateral bending efforts. Seroussi and Pope[39] reported increasing coactivation of external obliques with increasing effort in a series of experiments, in which participants lifted a weight requiring extension and lateral bending efforts.

■ Results

Results were quantified in terms of the sum of cubed muscle stresses ($\Sigma\sigma_m^3$), which is considered to be a measure of muscle fatigue rate, the maximum compressive loading of the six motion segments (T12–L1 through L5–S1), and the critical value of the muscle stiffness parameter q required for the spine to be stable. Table 1 and Figure 1 show the results for extension efforts, and Table 2 and Figure 2 show the results for lateral bending efforts.

Tables 1 and 2 give the values for four differing percentages of maximum efforts with no coactivation. For extension and for lateral bending efforts, spinal stability decreased with increasing effort up to the maximum efforts (increasing values of muscle stiffness required for stability). This decrease probably resulted from the spinal stiffness, which is independent of effort in these analyses, becoming less important relative to the required contribution from muscle stiffness. In fact, no muscle stiffness was required to stabilize the spine ($q = 0$) for

Table 1. Values of Critical q, Maximum Spine Compression, and the Sum of Cubed Muscle Stresses ($\Sigma\sigma_m^3$) for 40, 60, 80, and 100% of Maximum Extension Efforts With No Coactivation

% Effort	Critical q	Maximum Spine Compression (N)	$\Sigma\sigma_m^3$ (muscle fatigue rate) $(N/mm^2)^3$
40	0.62	682	0.24
60	1.95	976	0.63
80	3.17	1199	1.38
100	4.17	1553	3.70

6% and 8% of MVC coactivation of external oblique at 40% of maximum extension effort.

Figures 1 and 2 show the percentage changes from the "no coactivation" values for the three different coactivation patterns at 40% and 80% of maximum efforts. The stability increased progressively (smaller values of critical q) in all cases, but at the expense of greater summed
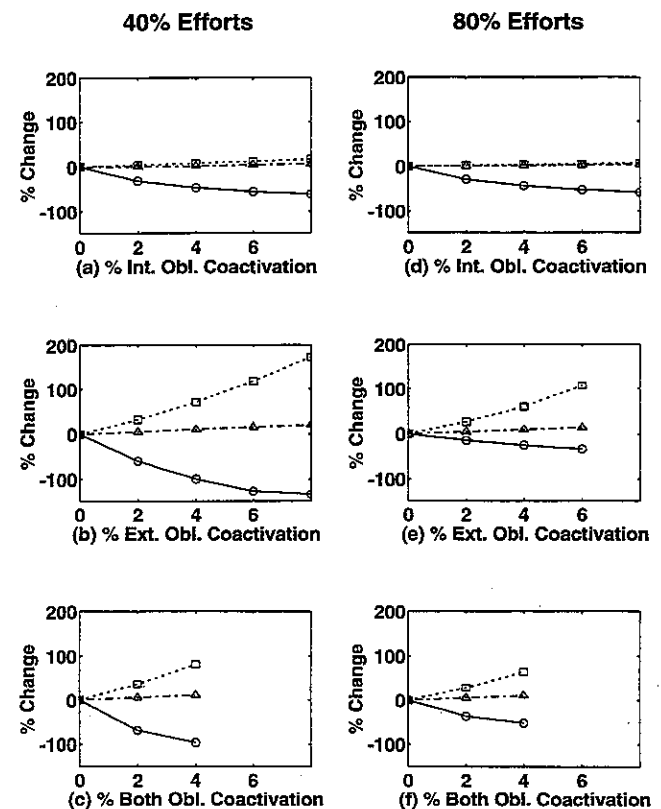

Figure 1. Percent changes from the no coactivation values (Table 1) in the critical muscle stiffness parameter q (o——o), maximum spine compression, ($\triangle$-•-•-$\triangle$) and the sum of cubed muscle stresses (related to muscle fatigue rate; ($\square$•••$\square$) for 40% (a–c) and 80% (d–f) of maximum extension efforts with increasing abdominal muscle coactivations. In (e) there was no feasible solution for the muscle force distribution for coactivation at 8% of maximum voluntary contraction, apparently because the magnitude of flexion moment produced by the external obliques overpowered the extensor muscles.

Table 2. Values of Critical q, Maximum Spine Compression, and the Sum of Cubed Muscle Stresses ($\Sigma\sigma_m^3$) for 40, 60, 80, and 100% of Maximum Lateral Bending Efforts With No Coactivation

% Effort	Critical q	Maximum Spine Compression (N)	$\Sigma\sigma_m^3$ (muscle fatigue rate) (N/mm^2)^3
40	2.49	914	0.67
60	5.65	1015	0.43
80	6.92	1081	0.38
100	3.93	1492	2.22

cubed muscle stresses ($\Sigma\sigma_m^3$). The small levels of coactivation of the obliques produced increased stability (decreases in the required critical muscle stiffness parameter q on the order of 50%). Increases in the maximum spine compression were small (less than 21%).

According to these analyses, the external obliques provided the greatest gains in stability (Figures 1B, 1E, 2B, and 2E), but at the cost of a greater percentage increase in the muscle fatigue rate ($\Sigma\sigma_m^3$). The internal oblique muscles (Figures 1A, 1D, 2A, and 2D) produced

40% Efforts   80% Efforts

Figure 2. Percent changes from the no coactivation values (Table 2) in critical muscle stiffness parameter q (o——o), maximum spine compression ($\triangle$-•-•-$\triangle$), and the sum of cubed muscle stresses (related to muscle fatigue rate; ($\square$-•-•-$\square$) for 40% (a–c) and 80% (d–f) lateral bending efforts with increasing abdominal muscle coactivations. In (b) there was no feasible solution for the muscle force distribution with this high level of antagonistic coactivation.

larger increases in stability relative to the increases in $\Sigma\sigma_m^3$. The analyses predicted an intermediate effect with coactivation of internal and external obliques.

■ Discussion

Antagonistic coactivation of the abdominal muscles increased lumbar spine stability. In these analyses, increased stability occurred at the cost of increased muscle fatigue rates and small increases in spinal compression. These results confirm that without active muscle stiffness (*i.e.*, with $q = 0$) the lumbar spine would generally be unstable (buckle) in response to small perturbations, even if the spine was in equilibrium. These findings support the hypothesis that activated muscles behave as stabilizing springs (and not just force generators); that active neuromuscular responses to small disturbances are, therefore, often not needed; and that coactivated synergistic and antagonistic muscles provide additional trunk stability.

For stability, the calculated critical muscle stiffness parameter was as much as 6.92 . The exact *in vivo* magnitude of active muscle stiffness remains unclear because of practical difficulties of measurement. Crisco and Panjabi[11] examined the literature and found values of q ranging from 0.5 to 42, with a mean of 10. The critical values found in the current study were at the lower end of this range of experimental values, implying that the spine has a reasonable margin of safety against buckling. This margin of safety is increased by coactivation. However, it is possible that under fatigue or pathologic conditions, lowered q values would result in an unstable spine that is liable to self-injury.

Thelen et al[41] estimated that coactivation increased spine compression by 16–19% in maximum extension efforts and by a greater amount in maximum lateral bending. The current study indicated a maximum increase of 21%, which was calculated for 40% of maximum extension effort with 8% of MVC coactivation of the external oblique muscle.

These findings depend on the geometric representation of the muscles and might be sensitive to the approximations made in the model. Although the muscular anatomy in this model was very detailed, some simplifications were made. Each abdominal muscle was represented as a single straight-line muscle, instead of a curved sheet of muscle. Single joint intertransversii and rotatores muscles were omitted, because they are relatively small and close to the spine. Intra-abdominal pressure effects and possible effects of the thoracolumbar fascia were not considered, because it appears that their effect on trunk forces is relatively small in neutral postures.[25,31]

The stiffness of the motion segments contributes to spinal stability, although these analyses confirm that, in general, motion-segment stiffness is not sufficient to prevent spinal buckling. The increase in spinal stability that occurred with decreasing efforts was probably a result of
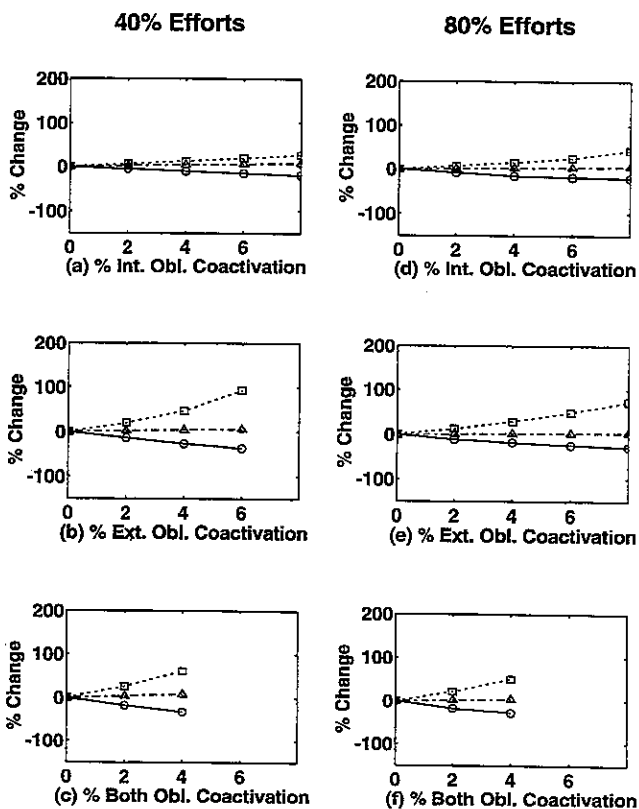
the spinal stiffness, which predominated when less effort and fewer active muscles were used. The amount of muscle stiffness required for stability was found in a previous study to be sensitive to spinal posture and spinal stiffness.[14]

The authors of the current study considered that the neuromuscular control system probably presets the muscle coactivation to ensure stability, which would reduce the need for active neuromuscular control. These static stability analyses apply to cases of small perturbations from static or quasistatic equilibrium and to the time frame before any reflex or voluntary muscle responses. Dynamic stability would be important under changing loads, large postural changes, or neural feedback.

Validation of these analyses using human experimentation is risky because of the potential for experimentally inducing spinal buckling under loaded conditions. A further difficulty in validating these analyses is the inability to measure *in vivo* spine and muscle forces. Also, muscle activation patterns can be quite variable in different individuals and under differing circumstances, although in controlled lab experiments it has been found that the activation pattern is relatively constant with increasing effort.[22,24,32] The validity of the analyses depends on whether accurate or plausible model inputs and model formulation were used.

The exact pattern of activation used has implications for the loading of the spine and surrounding tissues. The results of this study indicate that *in vivo* there is a compromise to be made either in trunk stability or in the magnitude of the trunk muscle fatigue and spine compression (the latter to a small extent for the efforts studied here). The neuromuscular system must balance the competing requirements of these factors. The three coactivation patterns used in this study were partly based on EMG findings. However, the simulated magnitudes of abdominal coactivations may represent a suboptimal strategy in terms of increasing stability, while seeking to minimize muscle fatigue rate and spinal loading.

Previously it was proposed that antagonistic abdominal muscle coactivation pressurized the abdomen and reduced spinal compression. The analyses in the current study indicate that its probable role is to stabilize the spine. Spinal biomechanical analyses that consider stability help to explain the role of antagonistic muscle coactivation. Future biomechanical models that include stability either as a constraint or as part of a physiologic cost function may provide more physiologic coactivation patterns and better estimates of safe postures and loads for handling tasks.

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