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Role of Muscles in Lumbar Spine Stability in Maximum Extension Efforts

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Summary: Many problems of the lumbar spine that cause pain are attributed to instability. The ligamentous spine (without muscles) is unstable at very low compressive loads. This study examined the hypothesis that instability of the lumbar spine is prevented under normal circumstances by the stiffness of spinal musculature, without active responses from the neuromuscular control system. The effect of muscle activity (force and stiffness) on the stability of the lumbar spine was analyzed for maximum voluntary extension efforts with different spinal postures in the sagittal plane. The analysis included realistic three-dimensional representation of the muscular anatomy with muscles crossing several motion segments. The stiffness of motion segments was represented using *in vitro* measured properties. Under a range of conditions with maximum extension effort, active muscle stiffness was required to prevent the lumbar spine from buckling. The dimensionless value of the muscle stiffness parameter q as a function of activation and length had to be greater than a critical value in the range of 3.7-4.7 in order to stabilize the spine. Experimentally determined values of q ranged from 0.5 to 42. These analyses demonstrate how changes in motion segment stiffness, muscle activation strategy, or muscle stiffness (due to degenerative changes, injuries, fatigue, and so on) might lead to spinal instability and "self-injury."

In vitro, the ligamentous lumbar spine is unstable at compressive loads of 88 N (8); however, *in vivo* the compressive force acting on the lumbar spine exceeds 2,600 N, as demonstrated by measurements of intradiscal pressure (25) and biomechanical models of lifting (32). Thus, the spinal column is inherently unstable, but it is believed that *in vivo* a combination of muscle forces and muscle stiffnesses stabilize it. Spinal instability or segmental instability is a concept used clinically to describe a situation in which motion segments apparently are hypermobile (10). Although this clinical condition is notoriously poorly defined (1), it generally is considered a degenerative condition of the motion segments. Most biomechanical studies have assumed that it is simple overload of the spine that causes injuries (5) and their painful consequences. Therefore, prevention of injury and effective rehabilitation has been directed primarily at minimizing spinal forces. This viewpoint does not take into account that muscles are required to stabilize the ligamentous spine or the possible destabilizing effects of poor neuromuscular coordination.

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Muscle and ligamentous forces increase the compressive load on the spine, increasing its tendency to buckle; however, the muscle forces are required to stabilize the spine. The relative magnitude of these competing effects must be resolved with quantitative analyses.

If muscles acted as pure force generators, then the nervous system would be responsible for stabilizing the spine by producing an appropriate response to every small disturbance. Instead, it appears that activated muscles act not only as force generators but also as stabilizing springs (2,6,7). The amount of muscle stiffness depends on the degree of muscle activation, so it can be set or modulated in advance under control of the nervous system. This regulation of muscle stiffness by activation (and more commonly coactivation of antagonist muscles) is used in targeted movements to control the trajectory of a body segment (15).

Previous investigations of the stability of the spine started with the observation that the ligamentous spine is highly unstable (8,21,33). Bergmark (2) examined the possibility that muscle stiffness stabilizes the lumbar spine for extension efforts using a model with simplified muscle anatomy and motion segments. The distribution of muscle force was reduced to static determinacy by a number of simplifying assumptions; however, it was found that the muscle stiffness required for stability was high compared with values

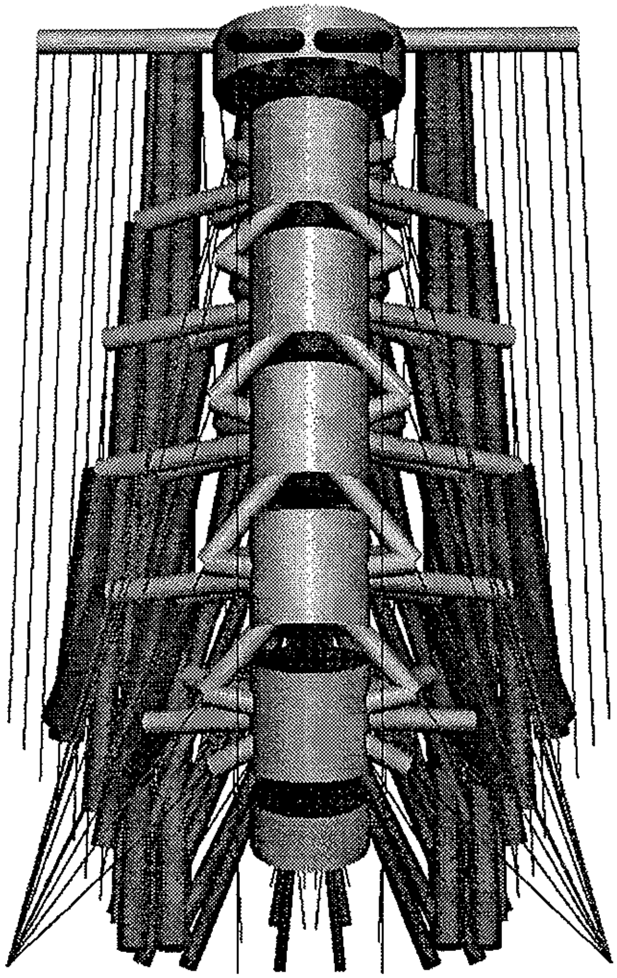

FIG. 1. Anteroposterior view of the lumbar spine model. The lighter-gray cylinders represent the lumbar vertebrae, with rigid connections supporting muscle attachments. The thorax is a rigid body to which all thoracic muscles attach. The position of S1 is indicated by a simple cylinder. The rest of the sacrum and the pelvis (which were fixed) are not shown. Active muscles (activity corresponding to the maximum extension effort) are shown as cylinders with diameters proportional to their physiologic cross-sectional areas, and inactive muscles are shown as thin lines.

subsequently reported by Crisco and Panjabi (6,7) from a review of the literature on muscle physiology. This leaves open the question of how muscle stiffness stabilizes the spine.

Crisco and Panjabi (7) explored the critical muscle stiffness required to maintain stability in five different hypothetical muscle architectures attached to a straight spine but did not establish how this would apply to realistic anatomy or how the required stiffness might relate to properties of active muscle. Dietrich et al. (9) modeled the musculature of the spine as solid finite elements with linear passive and active stiffness. The model buckled, but they did not establish how such buckling is avoided *in vivo*.

Because of the approximations and simplifying assumptions in previous studies, it has not been established quantitatively whether muscle stiffness alone is adequate to stabilize the spine in response to small perturbations. We analyzed stability using realistic data for spinal geometry, motion segment stiffness, and muscle anatomy. The overall hypothesis was that damage occurs in the low back when muscles are not sufficiently well coordinated to maintain both equilibrium and stability at all spinal levels. We addressed maximum extension efforts because they are considered more challenging to stability and have unique solutions in the analysis of distributions of muscle force. This avoided a major simplifying assumption of previous studies. We hypothesized that the lumbar spine normally is stabilized in response to small disturbances from the maximum effort state by the stiffness of spinal musculature. In this way, it would be "self-stabilized" without active responses from the neuromuscular control system, which has inherent time delays.

METHODS

The analysis of lumbar spine stability involved two sequential steps: (a) an analysis of distribution of muscle force, which involved linear programming with equilibrium and physiologic constraints, was used to calculate the maximum extension effort and corresponding motion segment and muscle forces, and (b) an eigenvalue buckling analysis of spinal stability was done next, using the motion segment and muscle forces found in the first step.

The geometry of the lumbar spine model was specified in terms of vertebral body positions, locations of muscle attachment points, and physiologic cross-sectional areas of muscle (3,34) (Fig. 1). The dorsal musculature, rectus abdominis, and psoas were represented by 66 symmetric muscle pairs. The same three simplifying assumptions were made about all of the muscles: (a) they take a straight line path from origin to insertion, (b) their contractile stress (force/muscle area) was between 0 (muscles produce only tension) and 460 kPa (3,34), and (c) they have active stiffness proportional to activation and inversely proportional to muscle length (2). The model used beam elements (11) matched to the experimentally derived data on linear stiffness given by Panjabi et al. (27) to represent the spinal motion segments. The thorax and sacrum/pelvis were both considered as rigid bodies. The sacrum/pelvis was constrained from moving. A static vertical compressive load of 340 N was imposed at the center of T12 in the thorax rigid body to represent upper body weight.

A previously published method (34) was used to perform the calculations of muscle force for maximum efforts. A redundant set of equations had to be solved in order to find the muscle and joint forces corresponding to a maximum voluntary extension effort. Since by equilibrium the maximum effort is a linear function of the muscle and spinal forces, the problem was solved with use of linear programming. The objective function was to maximize the extension effort moment at the T12 vertebral body center as a function of the 132 muscle forces and 36 vertebral displacements (168 variables total). The constraints on the solution were force and moment equilibrium of the thorax and each vertebra, bounds of muscle stress (0-460 kPa), the physiologic range of motion permitted in the joints (5 mm and 5° for the sagittal plane; 2 mm and 2° for the other planes), and additional displacement constraints to impose symmetry of the sagittal plane, which resulted in a smaller linear program and symmetric muscle forces. By analyzing maximum efforts, we avoided the unsolved problem of understanding physiologic strategies for muscle activation at submaximum loads. The calculation was performed using an active set projection method that is a variation of the simplex method (the "lp" routine in the optimization toolbox of Matlab; The MathWorks, Natick, MA, U.S.A.).

By the considerations of potential energy, a structure is stable if the work done by a small displacement produces a restoring force or an overall increase in the potential energy stored in the structure (20). The potential energy of a structure depends on its stiffness. This stiffness has two components: elastic stiffness (dependent on the structure's elastic properties) and geometric stiffness associated with the internal forces in the structure. Here, the internal forces are those acting on the motion segments and the forces generated by muscles.

The stability of a structure under conservative loads can be determined with use of an eigenvalue buckling analysis. Mathematically, the lowest positive eigenvalue λ, which is a nontrivial solution of the following equation, indicates the stability of the spine, and the associated eigenvector v indicates the buckling mode:

$$(K_S + K_M)v + \lambda(G_S + G_M)v = 0 \quad (1)$$

where K_S is the global spine stiffness, K_M is the global muscle stiffness matrix, G_S is the global geometric stiffness matrix of the spine, and G_M is the global geometric stiffness matrix of the muscles. The spine is unstable if the value of the lowest positive eigenvalue λ is less than 1, metastable if it is equal to 1, and stable if it is greater than 1. Since the model is three-dimensional, the shape of the buckling mode potentially is three-dimensional.

In these analyses of stability, the elastic stiffness of the spine was represented by the same beam elements used in the analysis of distribution of muscle force. The geometric stiffness of the spine was represented by a linearized approximation of the geometric stiffness of beam elements (26,29). The spinal loads were determined by the analysis of muscle force distribution.

Muscles have two components of axial stiffness: passive (nonlinear elastic) and active (6,7,12,14,24,30,31). The passive stiffness was assumed to be small in comparison with the active component. The active component is a complex function of activation, length, and rate of shortening or lengthening (14,16,17,19,30). There is disagreement in the literature about what is an adequate representation of active muscle stiffness (36). Here, the simplified linear formulation based on that of Bergmark (2) was used:

$$k_m = q\frac{T}{l_m} \quad (2)$$

where k_m represents axial muscle stiffness (Newton-millimeters), T represents active muscle force (Newtons) found in the analysis of muscle force distribution, l_m represents muscle length (millimeters), and q is a dimensionless parameter that is assumed to be the same for all muscles. Thus, the global muscle stiffness (K_M) in Eq. 1 depends on the forces in the muscles as well as the value of the active muscle stiffness parameter q, for which there is a wide range of published values (6,7). In these analyses, q was treated as a variable and the critical value necessary for spinal structural stability was determined.

The active muscle stiffness (k_m) in Eq. 2 was modeled with use of truss elements. The action of this axial muscle stiffness was transformed from the muscle attachments to the vertebral body centers with use of a rigid offset transformation (35). The geometric stiffness of the muscles was incorporated with use of the geometric stiffness matrix of truss elements (29), taking into account their offsets from the spine.

The generalized eigenvalue buckling problem (Eq. 1) was solved with use of the QZ algorithm (the "eig" routine in Matlab; The MathWorks). This analysis permitted us to determine the lowest (critical) value of q that was required to ensure stability of the loading state predicted by the analysis of force distribution (the value of q that produces a metastable spine [eigenvalue $\lambda = 1$]).

TABLE 1. *Critical q values of active muscle stiffness for a metastable spine and predicted maximum extension efforts (moments) at T12*

	Critical q value (dimensionless)	Maximum extension effort (Nm)
Neutral posture with nominal spine stiffness	4.5	60
Posture		
Extended 2.5° per vertebral level	4.6	57
Flexed 2.5° per vertebral level	4.6	65
Spine stiffness		
10% reduction	4.7	56
10% increase	3.7	64

The sensitivity of the critical value of active muscle stiffness parameter q was examined by varying either the posture or the motion segment stiffness (K_S). Changes in the muscle forces and critical value of q were examined in slightly flexed and extended postures about the neutral (upright standing) posture. This posture was altered by application of 2.5° rotations to each vertebral level in turn, and the new positions of all vertebrae and muscle attachment points above the rotated vertebra were calculated (Fig. 2). The 340 N preload was rotated to maintain the same alignment relative to the centers of T12 and S1. The forces associated with flexion or extension of the spine to a new posture were ignored because they do not affect the internal muscles forces or the calculations of stability. Changes in the critical value of active muscle stiffness parameter q were examined for changes of 10% in motion segment stiffness (K_S).

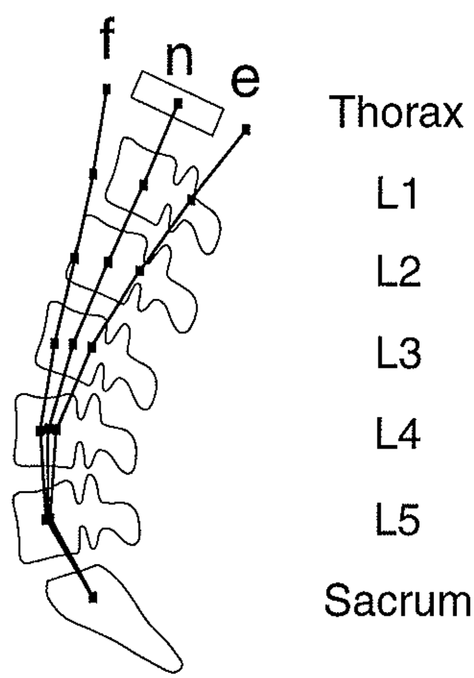

FIG. 2. Lateral view of the spine in the neutral upright standing posture (n), extended posture (e), and flexed posture (f). The extended and flexed postures were produced by 2.5° of extension or flexion from the neutral posture at each vertebral level.

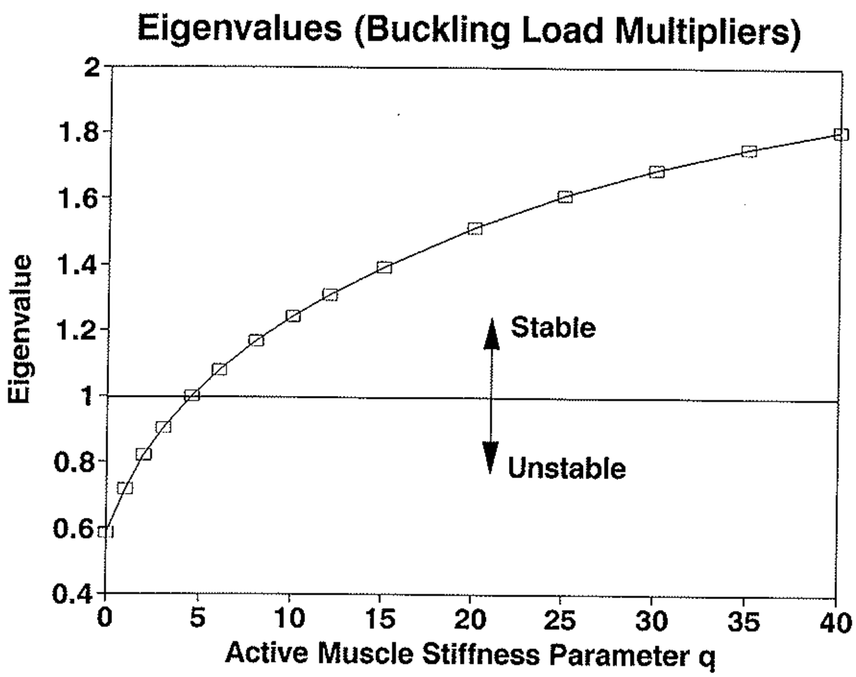

FIG. 3. Eigenvalues (buckling load multipliers) for the lumbar spine in the neutral posture. Eigenvalues greater than 1 indicate stability. The stability increases as q (the active muscle stiffness parameter) increases. With q = 0, the spine is unstable. The spine is stable for all values of q greater than 4.5 (the critical q value). The experimentally determined range of the physiologic muscle stiffness parameter q is 0.5-42 (6,7).

RESULTS

The eigenvalues (buckling load multipliers) from the stability analyses for the initial posture are shown in Fig. 3. It can be seen that in its neutral posture the spine would be liable to buckling if the muscle stiffness parameter q is less than 4.5 (the critical value of q). As q is increased, the spine becomes more stable, but there is a diminishing (nonlinear) effect with increasing values of q.

The spine was metastable ($\lambda = 1$) in its extended and flexed postures for q = 4.6. These modified spinal postures were associated with changes in the maxi-

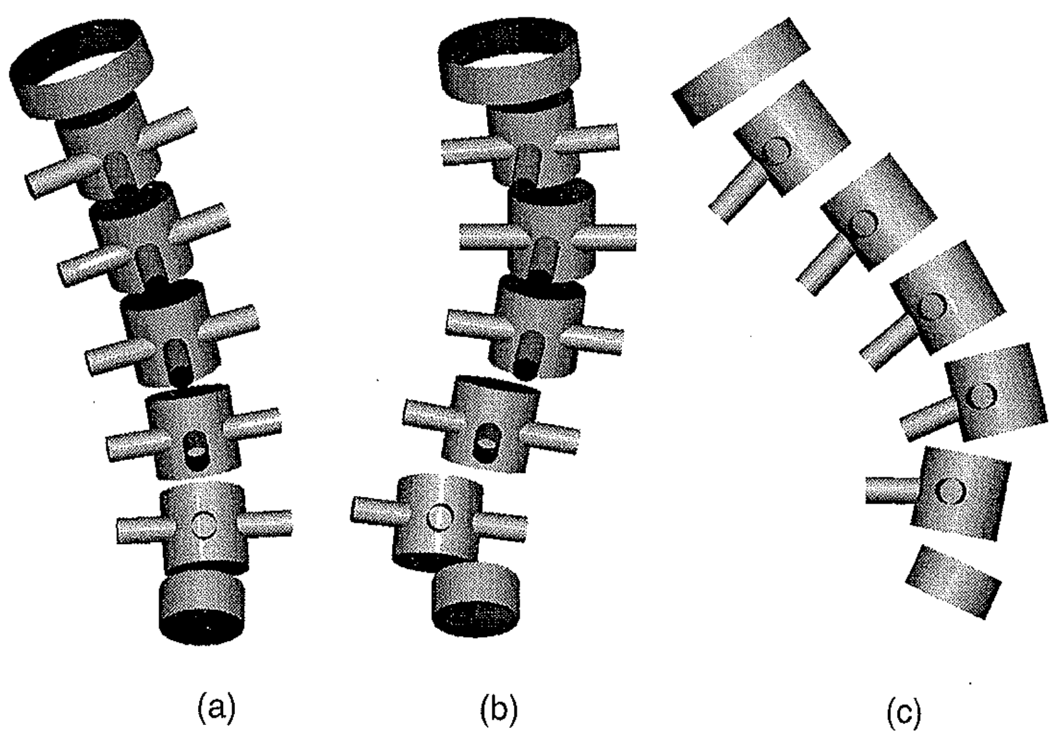

FIG. 4. Views of the first three mode shapes of the lumbar spine model in a maximum extension effort. **a:** First buckled mode shape for the neutral posture (posterior view) with q = 4.5. In this shape, there was 0.25° of axial rotation per degree of lateral bending of the thorax. **b:** Second mode shape, occurring at 1.65 times the loading required for the first mode (posterior view). **c:** The third mode shape also occurs at 1.65 times the loading of the first mode (lateral view). In all cases, the cylinders with processes represent the positions of the lumbar vertebrae; simple cylinders at the top and bottom represent the thorax and the sacrum, respectively.

TABLE 2. *Simulated muscle forces as percentage of physiologic maximum for maximum extension efforts*

Muscle name[a]	Muscle physiologic cross-sectional area (mm²)	Neutral posture with nominal spine stiffness[b]	Posture[b] Extended	Posture[b] Flexed	Spine stiffness[b] Reduced	Spine stiffness[b] Increased
Thoracic multifidus with insertions in lumbar vertebrae L1-L5 (six muscle slips)						
tmf L1-L5[c]	480	100	100	100	100	100
Muscles with origins at L1						
m1s	40	100	100	0	0	100
m1t.1	42	100	100	0	0	0
m1t.2	36	0	0	0	0	0
m1t.3	60	0	0	0	0	0
i1	108	100	0	100	100	100
l1	79	0	0	0	0	0
Muscles with origins at L2						
m2s	39	0	100	0	0	100
m2t.1	39	0	0	0	0	0
m2t.2	50	100	100	0	44	100
m2t.3	50	0	55	0	0	0
i2	154	0	0	47	0	0
l2	91	100	0	100	100	100
Muscles with origins at L3						
m3s	54	0	0	0	0	0
m3t.1	52	100	100	100	100	100
m3t.2	52	100	100	100	100	100
m3t.3	52	100	100	100	100	100
i3	182	100	100	100	100	100
l3	103	100	100	100	100	100
Muscles with origins at L4						
m4s	47	0	0	0	0	0
m4t.1	47	100	100	100	100	100
m4t.2	47	100	100	100	100	100
m4t.3	47	0	100	0	0	0
i4	189	100	100	100	100	100
l4	110	100	100	100	100	100
Muscles with origins at L5						
m5s	23	0	0	0	0	0
m5t.1	23	100	100	100	100	100
m5t.2	23	100	100	100	100	100
m5t.3	23	0	0	0	0	0
l5	116	100	100	100	100	100
Longissimus thoracis LT1-LT6, which insert in lumbar vertebrae (six muscle slips)						
LT1-LT6[c]	295	100	100	100	100	100
Longissimus thoracis LT7-LT12, which insert in sacrum						
LT7	78	0	0	0	0	0
LT8	125	0	0	0	0	0
LT9	146	0	0	0	0	0
LT10	160	0	0	23	0	0
LT11	167	54	7	100	20	88
LT12	138	100	100	100	100	100
Iliocostalis thoracis, which insert in iliac crest (IT7-IT12 are six muscle slips)						
IT5	23	100	0	100	100	100
IT6	31	0	0	100	0	0
IT7-IT12[c]	493	0	0	0	0	0

All muscle forces are expressed as a percentage of the physiologic maximum based on their physiologic cross-sectional area and an assumed effective maximum muscle stress of 460 kPa (3).

[a] The muscle names are based on the notation of Bogduk et al. (3). tmf = thoracic multifidus, m = multifidus, i = iliocostalis lumborum pars lumborum, l = longissimus thoracis pars lumborum, LT = longissimus thoracic, and IT = iliocostalis lumborum pars thoracis. The psoas major and rectus abdominis were inactive and are not listed.

[b] The muscle forces were symmetric; therefore, only the forces for one side are given in the table.

[c] These muscles were grouped and their physiologic cross-sectional areas were added for this table because their activations were the same in all cases.

mum efforts, but the critical q values were less sensitive (by three times) to these changes in posture.

The spinal stability was more sensitive to change of the spinal stiffness. A 10% decrease or increase in stiffness produced critical values of q of 4.7 and 3.7, respectively (Table 1). Despite the lower maximum effort and spinal loading resulting from the reduction in spinal stiffness, the spine was less stable, requiring a higher critical q value. The opposite was true with the increase in spinal stiffness.

The buckled shape of the spine (the eigenvector associated with the lowest eigenvalue from the buckling analysis) was primarily lateral bending with a small amount of torsional twisting of the spine (Fig. 4a). This suggests that the coactivation of lateral muscles observed in symmetric activities of the sagittal plane might serve to increase spinal stability. Lower physiologic values of q with active lateral stabilizing muscles might permit higher order buckling modes as illustrated in Fig. 4b and c.

The flexed (straighter) posture produced higher maximum efforts than the extended (lordotic) posture (Table 1). The active muscles and their level of activation as a percentage of maximum are given in Table 2. The active muscles for the neutral posture are illustrated in Fig. 1. Not all extensor muscles could be activated because this would preclude simultaneous equilibrium of all vertebrae.

The maximum compressive loads were at L4-L5. The maximum compressive force was 1,600 N in the neutral posture, 1,400 N in the extended posture, and 1,740 N in the flexed posture. The maximum compressive loads in the spine changed by 5% with the 10% change in spinal stiffness. These compressive loads are within the range determined from *in vivo* intradiscal pressures (25) but are much greater than the experimentally and analytically determined buckling loads for the ligamentous spine (8,21,33).

DISCUSSION

It has been assumed on a qualitative basis that it is the musculature that stabilizes the spine *in vivo*. These results show that at maximum efforts without active muscle stiffness (i.e., with $q = 0$), the lumbar spine would be unstable (buckle) in response to small perturbations, even though the spine was in equilibrium. These findings support the hypothesis that activated muscles must behave as stabilizing springs (and not just force generators) to avoid the need for active neuromuscular responses to small disturbances.

For stability, the parameter of muscle stiffness had to be greater than 3.7 in these analyses (Table 1). The exact *in vivo* magnitude of active muscle stiffness remains unclear because of practical difficulties of measurement. Crisco and Panjabi (6,7) examined the literature and found values of q from 0.5 to 42, with a mean of 10. The critical values found here were all greater than the lower end of this range of experimental values. This implies that the lumbar spine requires muscle stiffness values closer to the mean and that abnormal q values would result in an unstable spine, liable to "self-injury."

Clinically, instability of the spine commonly has been associated only with degeneration of the motion segment. This study shows that stability of the spine is reduced by "softening" (10% reduction in stiffness) of motion segments but also depends on muscle stiffness (which is a function of activation) remaining within normal ranges. Thus, spinal instabilities could result from degenerative changes reducing the stiffness of motion segments or from changes in muscle activation strategies (due to injuries, fatigue, and so on).

The finding here that "softening" of motion segments increases the requirement of the muscles to maintain a stable spine raises the importance of knowledge of the stiffness (or flexibility) behavior of motion segments. In these analyses, *in vitro* test results for this mechanical property were used. Panjabi et al. (28) proposed that motion segments have a "neutral zone" in which the stiffness is negligible for small rotations. Experimental work of Janevic et al. (18) and theoretical studies of Broberg (4) both point to increases in stiffness of the motion segment in the presence of compressive load ("preload"). Our analyses show that spinal stability is sensitive to joint stiffness, so by using data obtained from motion segments without preload we may have underestimated the degree of stability. On the other hand, the existence of lower stiffness close to the resting position "neutral zone" might offset this effect.

Although the muscular anatomy in this model was very detailed, some simplifications were made. Single joint intervertebral muscles (intertransversarii and rotatores) were omitted because they are relatively small and close to the spine. The oblique and transverse abdominal muscles and quadratus lumborum were omitted because of their negligible role in extension efforts. Effects of intraabdominal pressure and possible effects of the thoracolumbar fascia (13) were not considered, since it appears that their effect on trunk forces is relatively small (22,23).

In vivo, the trunk undergoes large displacements and has nonlinear properties, but in this study small displacements and approximations of linear material properties were used. The approximation of small displacements was considered to be reasonable for the static maximum extension efforts analyzed here. In general, large displacements tend to produce lower buckling loads. Nonlinear material properties may increase or decrease stability depending on the form of the nonlinearity.

Validation of these analyses using human experimentation is very difficult to imagine because of the risks of experimentally inducing spinal buckling under maximum efforts. The validity of the analyses depends on having accurate or plausible model inputs and model formulation. Greater efforts could be produced with a flexed as opposed to extended posture (65 and 57 Nm). The spine was equally stable in both postures; this implied that a flexed posture (straight back) is preferable for lifting.

Overall, these analyses provided two new insights into the significance of instability in the lumbar spine. First, the need to retain equilibrium at all levels of the spine was a limiting factor on the number of muscles that could be simultaneously activated in a maximum effort. Second, the spine could be in equilibrium but unstable, depending on the magnitude of the active muscle stiffness. Both of these findings suggest mechanisms by which poor neuromuscular control of the lumbar spinal musculature as well as a pathological reduction of motion segment or muscle stiffness could produce inappropriate combinations of spinal loading and possibly result in "self injury." Under normal circumstances at maximal efforts, muscle stiffness can stabilize the lumbar spine without the need for active feedback control.

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