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Distribution of tissue loads in the low back during a variety of daily and rehabilitation tasks

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Abstract—Successful rehabilitation programs arise from the balance of wisdom obtained through scientific laboratory experiment, and the "art" developed from clinical experience. Specifically, choosing the best exercises is enhanced by knowledge of the resultant tissue loads to reduce the risk of injury exacerbation and to strengthen healthy supporting tissues. The following report attempts to briefly describe a technique to obtain tissue load distribution, together with examples of tissue loads during the performance of some selected tasks. For example, it appears that those persons with shear injury to the vertebral joint (including facet, neural arch damage, or spondylolisthesis) or posterior ligament damage should avoid fully flexed postures given the resultant tissue loading. Specific data are provided to guide selection of various abdominal and low back exercises. Finally, some hypotheses and opinions are offered for possible improvements in clinical success.

Key words: *EMG, low back injury, lumbar spine, muscle forces, passive forces, rehabilitation.*

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INTRODUCTION

Throughout the course of daily activity, the low back system is subjected to loading from external forces and from forces produced by the internal tissues needed to create movement and maintain static postures. The fact that injury to the low back can only be caused by excessive loading of any given tissue cannot be easily dismissed. Unfortunately, the low back is not a simple mechanical system, and failure or injury cannot be described in a simplistic way. But fortunately, sophisticated techniques are being developed that facilitate investigation of the loads that lead to injury in a variety of injury sites. Knowledge of the tissue loads, therefore, is necessary to enable testing of hypotheses designed to reduce the risk of injury from a preventative standpoint and to optimize the loading that results from various rehabilitation programs for the injured. There is no doubt that once the injury has occurred, a plethora of variables, including psychosocial interactions, modulate the perception of pain and rate of recovery from the injury. Given these nonmechanical variables, it is often difficult to relate specific physical tissue damage with subsequent pain and compromise in function. Nonetheless, it is safe

to state that maintaining healthy, nontraumatized tissues and restoring damaged tissue is the objective of all in the rehabilitation field.

Several hypotheses may be considered to explain the role of movement/exercise and the resultant tissue loading on maintaining tissue health and optimizing the repair process. There is powerful evidence demonstrating that tissue loading stimulates tissue hypertrophy and slows—and possibly reverses—several degenerative conditions (1,2). However, the reported effectiveness of various training and rehabilitation programs is quite variable, with some claiming great success while others report no, or even negative, results (3,4). It is the opinion of this author that the prescription of optimal rehabilitation tasks is hindered by lack of knowledge of the loading that results in the various tissues. Therapists sometimes unknowingly formulate programs that create excessive loads and exacerbate the damage. The following report attempts to briefly describe a technique used to obtain low back tissue load distribution, together with examples of tissue loads, during the performance of some selected tasks.

The specific purpose of this review is to briefly explain the process of determining the load-time histories of tissues that are susceptible to injury, and provide some examples to quantify the risk of injury when people perform different tasks, particularly those tasks that may be prescribed as part of a rehabilitation program. The objective of therapy is to stress both damaged tissue and other healthy supporting tissues to the optimum level, which fosters tissue repair and strengthening, while avoiding excessive loading, which can exacerbate an existing structural weakness. However, we must humbly admit that while knowledge of tissue loads is important to avoid further injury, choosing the optimal load—not too much or too little—constitutes the blend of "art" and "science" attributes that characterize the best rehabilitation professionals. The professional challenge for all is to make wise decisions from the balance of laboratory and clinical experience.

How Tissue Loading Leads to Injury

Injury shall be defined, for the purposes of this review, as the full continuum from the most minor of tissue irritation (but microtrauma nonetheless) to the grossest level of tissue failure, for example, vertebral fracture or ligament avulsion. There is a tendency among those reporting or describing their back injury to identify a single specific event, such as lifting and twisting with a box, as causing forces sufficient to damage a structure in their back. While this description of low back injury is common, particularly among the occupational/medical community who are required to identify a single event when filling out injury reports, it is the contention of this author that relatively few low back injuries occur from this mechanical scenario. Rather, the culminating injury event is more commonly preceded by a history of excessive loading, which gradually but progressively reduced the tissue failure tolerance. Thus, it would appear that other scenarios where sub-failure loads can result in injury are probably more important, particularly for those charged with the responsibility of prescribing rehabilitation programs designed to stress tissues for strengthening while at the same time avoiding further injury. For example, the ultimate failure of a tissue (i.e., injury) may result from accumulated trauma produced by either repeated application of load (and failure from tissue fatigue) or of a sustained load that is applied for a long duration (and tissue failure from deformation and strain). Hence, the injury process need not be associated with very high magnitudes of loads but rather relatively low loads that are repeated or sustained, such as prolonged stretching and sitting.

Issues Relevant for Tissue Load Prediction

It is important to understand the limitations, and conversely the assets, of scientific laboratory approaches for investigating tissue loading *in vivo*. Because the low back system is an extremely complex mechanical structure, and direct measurement of tissue loads *in vivo* is not feasible, the only tenable option for tissue load prediction is to utilize sophisticated modeling approaches. However, there are several issues that must be addressed: anatomic detail; a method to solve for the inherent indeterminacy from so many unknown forces among the significant load-bearing structures; development of methods that enable the prediction of loads in deep (and inaccessible) muscles and supporting ligaments; and sensitivity to detect migration of load between supporting tissues during longer duration tasks.

Pain and disability have been documented to result from damaged ligaments, discs, vertebral end plates, vertebral bodies, various muscle injuries, and several other tissues (5). This requires a modeling approach that incorporates sufficient anatomical detail, together with a method sensitive to the many different ways in which

individual people utilize their muscles and various ligaments to perform tasks. Two basic approaches have been used to partition the supporting duties among the many components of the trunk musculature and ligamentous system: optimization approaches and biological approaches that utilize biological signals obtained from each subject (for example, measurements of muscle EMG and spine kinematics). The optimization approach attempts to satisfy the reaction moment requirements needed to support a posture by recruiting the various muscles based on an optimization criterion, such as the minimization of joint compression and shear load (6) or the minimization first of muscle contraction intensity and then spine compression load (7). Generally, optimization assumes that the motor control system operates to fulfill objectives that can be mathematically defined and, in so doing, ignores individual variability, predicting the same tissue load distribution for all subjects performing a certain task. Furthermore, most currently reported optimization approaches are very poor at predicting patterns of muscle cocontraction so characteristic of three-dimensional (3-D) spine motion (8,9).

On the other hand, a modeling approach that uses biological signals obtained directly from each subject is inherently sensitive to the individual ways that people load their low back tissues and hence is better suited to investigations of injury. For example, McGill and Norman proposed a dynamic model that attempted to determine the significant forces in many muscles in the low back, based in part on their neural drive measured through calibrated surface EMG and in the passive structures based on estimates of strain from directly measured spine kinematics (10). While the major asset of the biological approach is that both muscle cocontraction and individual differences in movement are fully accommodated, estimations of muscle force based, in part, on myoelectric signals are problematic as the force potential per muscle cross-sectional area must be assumed together with other variables that are known to modulate muscle force production. Furthermore, one must rely on anatomical accuracy to satisfy the moment requirements about all three joint axes and about several joints simultaneously. Recent work comparing the assets and liabilities of an EMG and spine kinematics approach with an optimization approach to obtain tissue force distribution profiles, concluded a greater suitability of the biological-EMG approach for investigation of injury mechanisms (11).

Nonetheless, a major drawback of the EMG-based approach is the myoelectric inaccessibility of the deeper torso muscles (e.g., psoas, quadratus lumborum, and three layers of the abdominal wall). In an attempt to address this drawback, McGill, Juker, and Kropf utilized indwelling intramuscular electrodes with simultaneous surface electrodes sites to evaluate the possibility and validity of using surface activity profiles as surrogates to activate deeper muscles over a wide variety of tasks and exercises, such as situps, curlups, leg raises, pushups, some spine extensor tasks, and lateral bending and twisting tasks common to rehabilitation programs (12). Prediction of these deeper muscles is possible from well chosen surface electrodes within the criterion of 15 percent of MVC (RMS difference) or less.

Finally, during prolonged tasks, it is sometimes important to predict tissue loads throughout the performance rather than trying to select a single event in time for analysis. For example, subtle shifts in tissue load distribution during the performance of lighter loading tasks, but of longer duration, can lead to situations where loads in a single tissue may rise to unreasonable levels, as can occur during prolonged sitting or flexion where ligamentous creep transfers more load and strain to the posterior annulus.

A Brief Description of the Laboratory-modeling Approach

Individual tissue loads have been predicted from a laboratory technique and model developed over the past 14 years. The model is composed of two distinct parts. First, a 3-D, linked-segment representation of the body was constructed, using the dynamic load in the hands as input; working through the arm and trunk linkage, reaction forces and moments about a joint in the low back (usually L4/L5; see **Figure 1**) are computed as described in McGill and Norman (13). Joint displacements are recorded on two or more video cameras at 30 Hz to reconstruct the joints and body segments in 3-D. This first model produces the three reaction forces and corresponding moments about the three axes of the low back (flexion-extension, lateral bend, and axial twist). The second anatomically detailed model enables the partitioning of the reaction moment obtained from the linked-segment model into the substantial restorative moment components (supporting tissues) using an anatomically detailed 3-D representation of the skeleton, muscles, ligaments, nonlinear elastic intervertebral discs, and so forth. This part of the model was first described in McGill and Norman (10), with full 3-D methods described in McGill (14), and the most recent update provided by Cholewicki and McGill where 90 low back and torso muscles are rep-

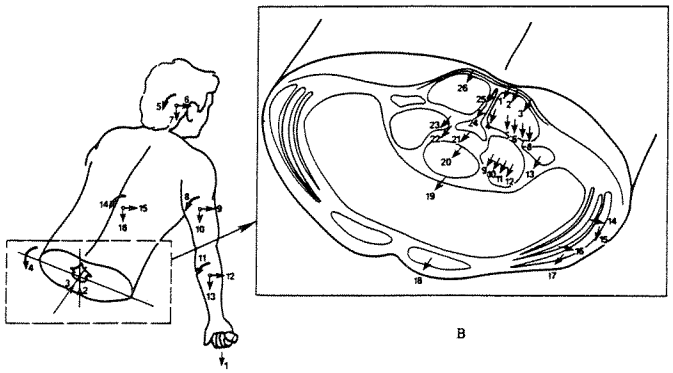

Figure 1.
The tissue load prediction approach required 2 models. The first (left) is a dynamic, 3-D, linked-segment model to obtain the 3 reaction moments about the low back; the second model (right) partitions the moments into tissue forces: muscle forces 1–18, ligaments 19–26, and moment contributions from deformed disc, gut, and skin in bending.

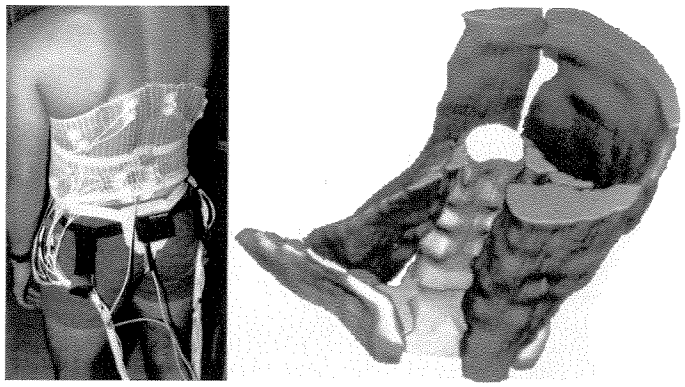

Figure 2.
Subject (left) monitored with EMG electrodes and an electromagnetic device to obtain 3 axes of lumbar motion. The modeled spine (right) moves in accordance with the spine of the subject, its muscles activated by the subject. (Only some muscles are shown for clarity.)

resented in total (15). Very briefly, first the passive tissue forces are predicted by assuming stress-strain or load deformation relationships for the individual passive tissues. Relationships are calibrated for the differences in flexibility of each subject by normalizing the stress-strain curves to the passive range of motion of the subject, detected by electromagnetic instrumentation that monitors the relative lumbar angles in 3-D. Then the remaining moment is partitioned among the many laminae of muscle, where muscle force estimates are based on their myoelectric profile and their physiological cross-sectional area. This is followed by further force modulation with known relationships for instantaneous muscle length and for either shortening or lengthening velocity. Recent improvements of the force/velocity relationship have been described in Sutarno and McGill (16). The method of using biological signals to solve the indeterminacy of multiple load-bearing tissues (see **Figure 2**) facilitates the assessment of the many ways that we choose to support loads: this objective is necessary for evaluation of injury mechanisms and the evaluation of various tasks prescribed in rehabilitation programs.

RESULTS

Examples of Tissue Loading

Several data sets will be presented and discussed to demonstrate tissue loads and provide clinical relevance.

The Case of Full Lumbar Flexion

Several studies have attempted to evaluate the issue of stoop versus squat lifting postures, based mostly on comparisons of low back compression, but these were unable to uncover a clear biomechanical rationale for the promotion of either method. Perhaps the issue is much more complex than has been realized. Using the tissue load distribution perspective, the following example demonstrates the shifts predicted from our modeling approach in tissue loading, which has quite dramatic effects on shear loading of the intervertebral column and lends insight into the stoop/squat lifting debate. First, the dominant direction of the pars lumborum fibers of longissimus thoracis and iliocostalis lumborum are noted to act obliquely to the compressive axis of the lumbar spine, producing a posterior shear force on the superior vertebra. In contrast, the interspinous ligament complex acts with the opposite obliquity to impose an anterior shear force on the superior vertebra (17; see **Figure 3**). This is one example where spine posture determines the interplay between passive tissues and muscles, which ultimately modulates the risk of several types of injury. For example, if a subject holds a load in the hands with the spine fully flexed sufficient to achieve myoelectric silence in the more superficial extensors (reducing their tension), and with all other joints held static so that the low back moment remains the same, then the tensioned ligaments will add to the anterior shear to levels well over 1,000 N. This is of great concern from an injury risk viewpoint (see **Figure 4**). It is acknowledged that deeper quadratus lumborum activity, noted by Andersson et al.

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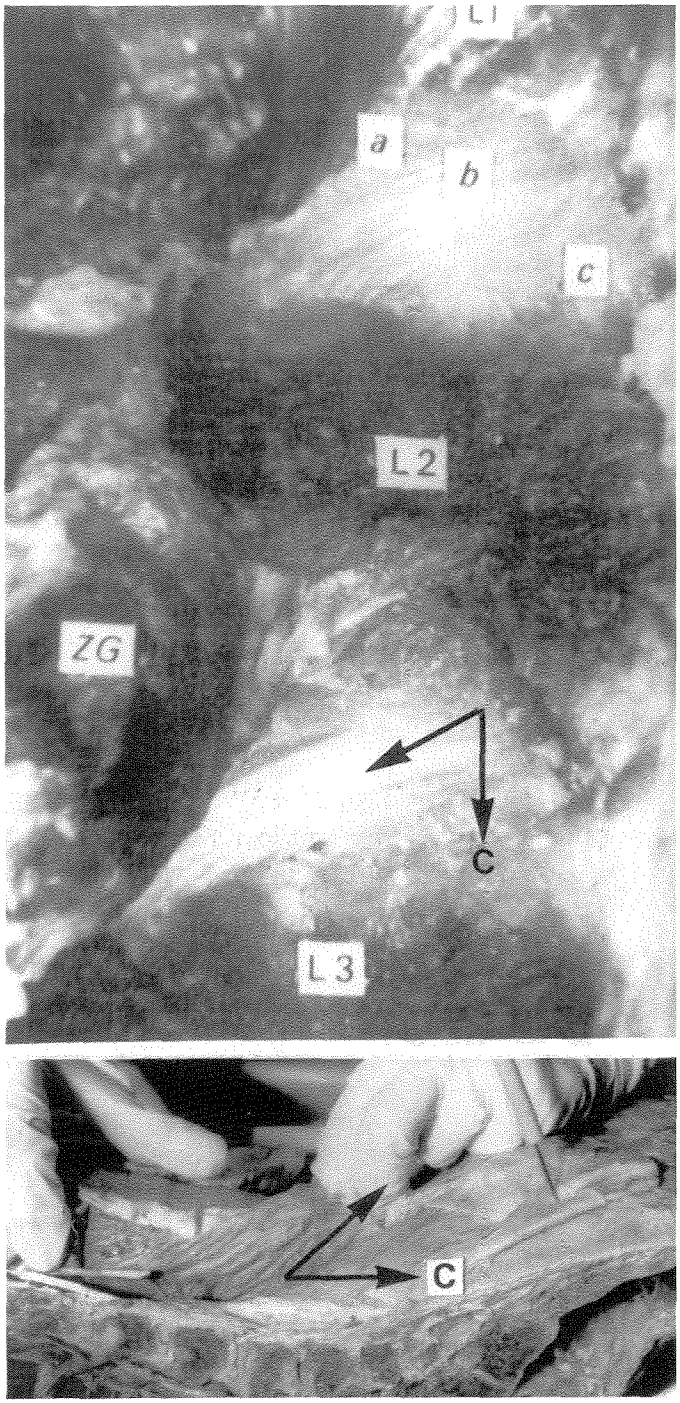

Figure 3.
Pars lumborum fibers (a) of iliocostalis lumborum and longissimus thoracis create a posterior shear force on the superior vertebra compared to the compressive axis **C**, while the interspinous ligament (b) imposes an anterior shear when strained in flexion. *Published by permission from J Anat Heylings* (17). At bottom: the general oblique line of action of the muscle and ligament, which causes shear loading, is shown compared to the compressive axis **C**.

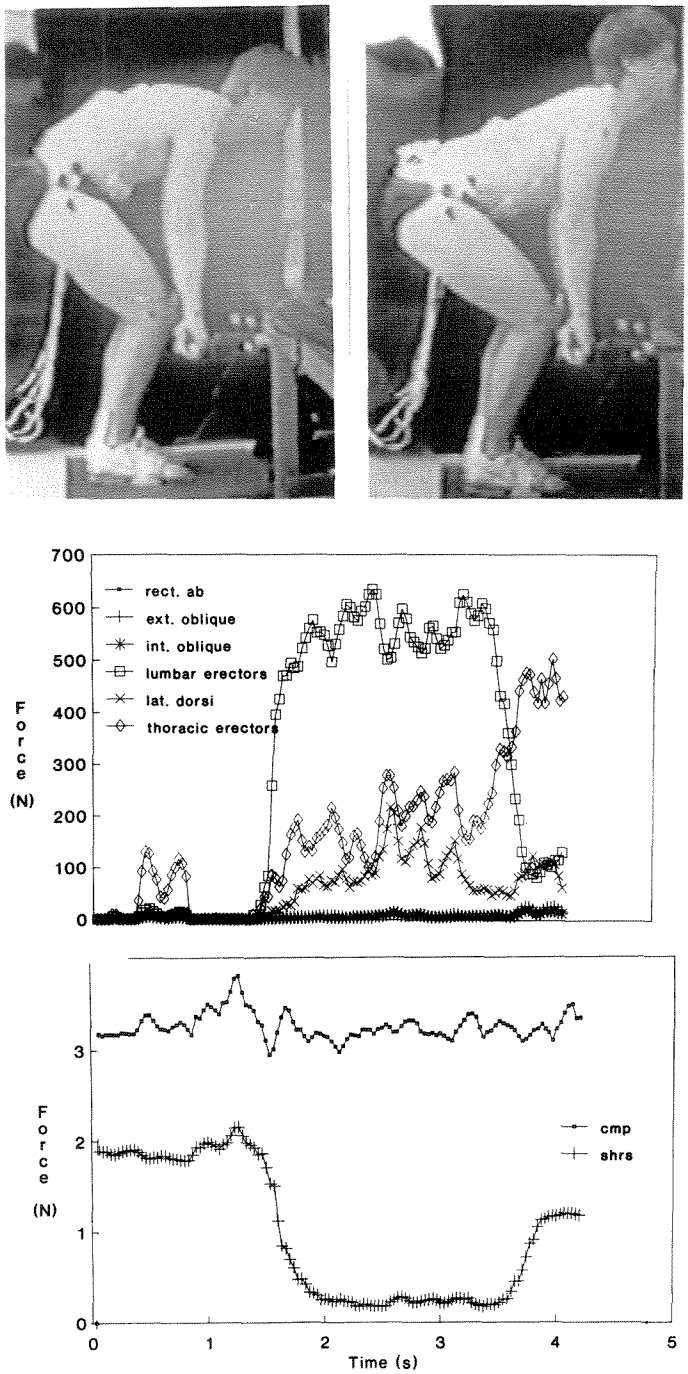

Figure 4.
The computer digitized images at top show that the fully flexed spine (left) is associated with myoelectric silence in the back extensors and loaded posterior passive tissues, and high shearing forces on the lumbar spine. At right, more neutral posture recruits the shear supporting pars lumborum extensors, disables the interspinous ligaments, minimizing their shear producing forces, and reduces the net shear (Shrs) on the spine. Compression (CMP) remains essentially unchanged.

(18), in this posture may provide minor shear support. However, if a more neutral lordotic posture is adopted, the extensor musculature responsible for creating the extensor moment will at the same time support the anterior shearing action of gravity on the upper body and hand-held load.

Disabling the ligaments greatly reduced shear loading. In another example of two similar subjects holding a load—one with the lumbar spine fully flexed, the other with a near neutral spine—the shift in tissue loading (demonstrated in **Table 1**) has smaller consequences on joint compression even though there was a noticeable amount of cocontraction (3,145 vs. 3,490 N) but much larger effects on joint anterior-posterior shear (954 vs. 269 N). Specifically, the major difference in shear load occurs as the pars lumborum fibers of erector spinae become disabled, removing their shear support, and the moment responsibility shifts to the interspinous/supraspinous complex, increasing joint shear. Full flexion postures have injury implications on strained posterior tissues and also on structures (e.g., facet joints, neural arch, or conditions of spondylolisthesis) affected by large shear loads.

Using knowledge of tissue loads, one could take the position that the important issue is not whether it is better to lift stooping or squatting, but rather the emphasis should be on placing the load close to the body to reduce the reaction moment (and the subsequent extensor forces and resultant compressive joint loading) and to avoid a fully flexed spine to minimize shear loading. In fact, sometimes it may be better to squat to achieve this, or in cases where the object is too large to fit between the knees, it may be better to stoop, flexing at the hips but always avoiding full flexion to minimize posterior ligamentous involvement. (For a more comprehensive discussion see 19–22).

Tissue Loading During Daily Activities and Rehabilitation Exercises

We have all been aware of the suggestion that situps and other flexion exercises should be performed with the knees and hips flexed. Several hypotheses have suggested that this disables the psoas and/or changes the line of action of psoas. Recent MRI-based data of Santaguida and McGill (23) demonstrated that the line of action of the psoas does not change due to alterations in lumbar or hip posture (except at L5/S1), as the psoas laminae attach to each vertebrae and "follow" the changing orientation of spine. There is no doubt that the psoas is shortened with the flexed hip, modulating force production. But the question remains, is there a reduction in spine load with the legs bent? Recently, McGill (24) examined 12 young men with the laboratory technique described previously, and observed no major difference in lumbar load as the result of bending the knees (average moment of 65 Nm in both straight and bent knees, compression: 3,230 N for straight legs, 3,410 N for bent knees; shear: 260 N for straight legs, 300 N for bent knees). Compressive loads in excess of 3,000 N certainly raise questions of safety. It appears that the issue of performing situps using bent knees or straight legs is probably not as important as the issue of whether to prescribe situps at all. This finding motivated us to examine a wide variety of rehabilitation tasks involving the torso musculature.

Our recent work at the University of Bern was directed toward obtaining normalized and calibrated muscle activation profiles from deeper muscles of the torso using intramuscular electrodes (namely psoas, quadratus lumborum, external oblique, internal oblique, and transverse abdominis), over a wide variety of different tasks and training exercises (25). Surface electrodes also monitored rectus abdominis, external oblique, internal oblique, rectus femoris, and lumbar erector spinae for comparison and to facilitate modeling analysis. EMG amplitudes, measured when the largest moment occurred in each task (**Table 2**), assist in interpretation of several important clinical and rehabilitation issues (cf. 26).

Psoas muscle activity is lowest during curlups, followed by higher levels during isometric side support; once again, bent knee situps were characterized by larger psoas activation than straight leg situps, through to leg raises, and hand-on-knee flexor isometric exertions. It is interesting to note that the "press-heels" situp which has been hypothesized to activate hamstrings and neurally inhibit psoas (27), actually increased psoas activation. Graded activity in rectus abdominis and each of the components of the abdominal wall requires reordering of each of these exercises demonstrating that there is no single best task for the collective "abdominals." Clearly, curlups excel at activating the rectus abdominis but result in low oblique activity. A very wise choice for abdominal exercises in the early stages of training or rehabilitation would consist of several variations of curlups for rectus abdominis and isometric, horizontal side support, with the

Table 1. Individual muscle and passive tissue forces and the lumbar moments, compression, and shear forces during full flexion together with just the forces in a more neutral lumbar posture demonstrating the shift from muscle to passive tissue and the resultant effects on joint compression and shear. The extensor moment with full lumbar flexion was 171 N.m producing 3145N of compression and 954N of anterior shear-in the more neutral posture of 170 N.m produced 3490 N of compression and 269 N of shear.

	Fully Flexed Lumbar Spine							Neutral Lumbar Spine
	Force	Moment (Nm)				Shear(N)		Forces
	(N)	Flexion	Lateral	Twist	Compression (N)	Anteroposterior	Lateral	(N)
Muscle								
R rectus abdominis	16	-2	1	1	15	5	-4	39
L rectus abdominis	16	-2	-1	-1	15	5	4	62
R external oblique 1	10	-1	1	1	8	7	-3	68
L external oblique 1	10	-1	-1	-1	8	7	3	40
R external oblique 2	7	-1	1	0	6	2	-3	62
L external oblique 2	7	-1	-1	-0	6	2	3	31
R internal oblique 1	35	0	3	-2	21	-19	20	130
L internal oblique 1	35	0	-3	2	21	-19	-20	102
R internal oblique 2	29	-2	2	-3	8	-17	21	116
L internal oblique 2	29	-2	-2	3	8	-17	-21	88
R pars lumborum (L1)	21	2	1	0	21	6	2	253
L pars lumborum (L1)	21	2	-1	-0	21	6	-2	285
R pars lumborum (L2)	27	2	1	0	26	8	2	281
L pars lumborum (L2)	27	2	-1	-0	26	8	-2	317
R pars lumborum (L3)	31	1	1	0	29	-4	6	327
L pars lumborum (L3)	31	1	-1	-0	29	-4	-6	333
R pars lumborum (L4)	32	1	1	-0	30	-7	6	402
L pars lumborum (L4)	32	1	-1	0	30	-7	-6	355
R iliocostalis lumborum	58	5	4	1	57	14	-1	100
L iliocostalis lumborum	58	5	-4	-1	57	14	1	137
R longissimus thoracis	93	7	4	0	91	23	-6	135
L longissimus thoracis	93	7	-4	-0	91	23	6	179
R quadratus lumborum	25	1	2	-0	25	-1	1	155
L quadratus lumborum	25	1	-2	0	25	-1	-1	194
R latissimus dorsi (L5)	15	1	1	-0	14	-1	-6	101
L latissimus dorsi (L5)	15	1	-1	0	14	-1	6	115
R multifidus 1	28	1	1	1	26	6	9	80
L multifidus 1	28	1	-1	-1	26	6	-9	102
R multifidus 2	28	1	1	0	28	6	0	87
L multifidus 2	28	1	-1	-0	28	6	0	90
R Psoas (L1)	25	1	2	0	24	0	6	61
L Psoas (L1)	25	-1	-2	-0	24	0	-6	69
R Psoas (L2)	25	-1	2	0	24	0	6	62
L Psoas (L2)	25	-0	-2	-0	24	0	-6	69
R Psoas (L3)	25	-0	1	0	24	0	7	62
L Psoas (L3)	25	-0	-1	-0	24	0	-7	69
R Psoas (L4)	25	-0	1	1	24	0	8	61
L Psoas (L4)	25	-0	-1	-1	24	0	-8	69
Ligament								
Anterior Longitudinal	0	0	0	0	0	0	-	0
Posterior Longitudinal	86	2	0	0	261	44	-	0
Ligamentum flavum	21	1	0	0	21	2	-	3
R intertransverse	14	0	0	0	13	3	-	0
L intertransverse	14	0	-0	-0	13	3	-	0
R articular	74	2	1	1	65	40	-	0
L articular	74	2	-1	-1	65	40	-	0
R articular 2	103	3	2	2	84	-3	-	0
L articular 2	103	3	-2	-2	84	-3	-	0
Interspinous 1	301	18	0	0	273	142	-	0
Interspinous 2	345	14	0	0	233	268	-	0
Interspinous 3	298	10	0	0	194	238	-	0
Supraspinous	592	41	0	0	591	79	-	0
R lumbodorsal fascia	122	8	1	-0	109	-1	-	0
L lumbodorsal fascia	122		-1	0	109	-1	-	0
Passive tissue								
Disc	-	9	0	0	-	-	-	1
Gut, etc.	-	11	0	0	-	-	-	2

Table 2. Subject averages (5 men, 3 women) of EMG activation normalized to 100% MVC - mean and (standard deviation). Note Psoas channels, External Oblique, Internal Oblique, Transverse Abdominis, Quadratus Lumborum, are intramuscular electrodes while Rectus Abdominis, Rectus Femoris, Erector Spinae are surface electrodes. (Quadratus lumborum was only collected from 4 subjects.)

TASK	Psoas 1	Psoas 2	EOi	IOi	TAi	QLi	RAs	RFs	ESs
Flexion dominant tasks:									
Straight-leg situps	15(12)	24(7)	44(9)	15(5)	11(9)		48(18)	16(10)	4(3)
Bent-Knee situps	17(10)	28(7)	43(12)	16(14)	10(7)	12(7)	55(16)	14(7)	6(9)
Press-heel situps	28(23)	34(18)	51(14)	22(14)	20(13)		51(20)	15(12)	4(3)
Bent-knee curlup	7(8)	10(14)	19(14)	14(10)	12(9)	11(6)	62(22)	8(12)	6(10)
Bent-knee leg raise	24(15)	25(8)	22(7)	8(9)	7(6)	12(6)	32(20)	8(5)	6(8)
Straight leg raise	35(20)	33(8)	26(9)	9(8)	6(4)	9(2)	37(24)	23(12)	7(11)
Isom. left hand-to right-knee	16(16)	16(8)	68(14)	30(28)	28(19)		69(18)	8(7)	6(4)
Isom. right-hand-to-left-knee	56(28)	58(16)	53(12)	48(23)	44(18)		74(25)	42(29)	5(4)
Cross curlup right shoulder across	5(3)	4(4)	23(20)	24(14)	20(11)	6(4)	57(22)	10(19)	5(8)
Cross curlup left shoulder across	5(3)	5(5)	24(17)	21(16)	15(13)	6(4)	58(24)	12(24)	5(8)
Pushup from feet	24(19)	12(5)	29(12)	10(14)	9(9)	4(1)	29(10)	10(7)	3(4)
Pushup from knees	14(11)	10(7)	19(10)	7(9)	8(8)		19(11)	5(3)	3(4)
Lateral bending moment tasks:									
Isom. side support	21(17)	12(8)	43(13)	36(29)	39(24)	54(28)	22(13)	11(11)	24(15)
Dyn. side support	26(18)	13(5)	44(16)	42(24)	44(33)		41(20)	9(7)	29(17)
Extension-lifting tasks:									
Lift light load (18kg)	9(10)	3(4)	3(3)	6(7)	6(5)	43(24)	14(21)	6(5)	37(13)
Lift heavy load (50-100kg)	16(18)	5(6)	5(4)	10(11)	10(9)	74(21)	17(23)	6(5)	62(12)
Twisting moment tasks:									
Seated Isom. twist CCW	30(20)	17(15)	18(8)	43(25)	49(35)	11(6)	17(22)	7(4)	14(6)
Seated Isom. twist CW	23(20)	11(8)	52(13)	15(11)	18(19)	9(6)	13(10)	9(10)	13(8)
Hip rotation tasks:									
Standing hip internal rotation	21(18)	10(9)	18(12)	24(23)	33(20)	32(18)	13(9)	9(7)	18(6)
Standing hip external rotation	27(20)	22(19)	17(13)	21(19)	31(17)	13(12)	13(8)	19(11)	17(9)
Sitting hip internal rotation	19(15)	21(18)	36(31)	30(30)	31(29)	6(1)	18(8)	20(19)	12(8)
Sitting hip internal rotation	32(25)	25(20)	11(9)	15(17)	16(13)	9(5)	15(9)	16(13)	8(8)
Other:									
Sitting upright	12(7)	7(5)	3(6)	3(3)	4(2)	6(4)	17(9)	4(2)	5(8)
Sitting slouched/relaxed	4(4)	3(3)	2(5)	2(2)	4(3)	5(5)	17(11)	3(2)	5(8)
Quiet standing	2(1)	1(1)	3(4)	5(3)	4(2)		5(5)	3(3)	11(11)
Symmetric bucket hold 0kg	2(4)	1(1)	7(4)	5(3)	5(1)	3(1)	10(7)	3(3)	3(6)
20kg	3(4)	1(1)	9(5)	6(4)	6(1)	14(12)	10(8)	3(3)	4(7)
30kg	3(5)	1(1)	10(6)	8(6)	6(2)	15(17)	10(8)	3(3)	3(2)
40kg	1(2)	0(1)	2(1)	2(2)	1(1)	19(23)	10(9)	2(1)	2(1)

body supported by the feet and upper body supported by one elbow on the floor, to challenge the abdominal wall in a way that imposes minimal compressive penalty to the spine (**Figure 5**).

Recent work (28) has calculated the distribution of tissue loads during the recommended side support exercise (**Table 3**). Other clinically relevant findings from these two data sets include notions that psoas activation is dominated by hip flexion demands and that psoas activity is not consistent with either lumbar sagittal moment or compression demands. We question the often cited notion that psoas is a lumbar spine stabilizer; quadratus lumborum activity is consistent with lumbar sagittal moment and compression demands, suggesting a larger role in stabilization (29); psoas activation is relatively high (greater than 25 percent MVC) during pushups, suggesting concern for persons with low back injury given the spine compression penalty associated with activation; there is no dominance of psoas activation for either internal or external hip rotation; psoas is more active during upright, unsupported sitting than during relaxed and "slouched" unsupported sitting.

DISCUSSION

The rehabilitation professional enhances the chance for success when designing custom programs by blending both clinical experience and scientific data. Some of the techniques and subsequent data reported here will hopefully contribute to the selection of scientifically justifiable tasks and exercises. In particular, the availability of tissue load-time histories enables the selection of tasks that are more suitable for certain individuals, but yet may be contraindicated for others. The bottleneck occurs in the inability to conclusively diagnose the tissue that has been damaged. Better diagnostic techniques will lead to improved identification as to which tissues are damaged, the extent of the damage, and subsequent prescription of rehabilitation tasks to sufficiently challenge the injured tissue and strengthen supporting tissues.

Several generalized limitations apply to the approach for obtaining tissue load-time histories described in this review, together with the interpretation of results. For example, predicting muscle forces from surface electromyography remains problematic. However, the ability to predict measured moments from biologically driven models adds some degree of validity to the modeling approach. Furthermore, animal studies involving the direct measurement of tendon forces, coupled with similar predictions of muscle force, from EMG-based approaches, increase the validity of the method (30–32). Another weakness in all modeling approaches is the need for improved data on tissue behavior (both injured and intact). Data on the effects of prolonged loading and repeated loading, the effects of rest periods for tissue healing and adaption, and the effects of individual parameters, such as age and gender, are sparse and both need and deserve attention.

In summary, as improvements to current approaches are made, and as new approaches are developed, availability of customized rehabilitation programs for every individual, and for all types of low back tissue injury, will improve. The approach and results reported in this review constitute only a beginning in this direction. Veterans and workers of the industrialized world can ill afford the assumption that simplistic qualitative approaches will lead to significant improvements in the rehabilitation of low back injury. The scientific community must continue to press forward with more comprehensive approaches to understand the biomechanical loading of tissues that, when excessive, lead to injury, but when absent, lead to detrimental deterioration of tissues. The most healthy optimum load will only be achieved when a thorough understanding of the biomechanical force parameters is obtained and wisely applied.

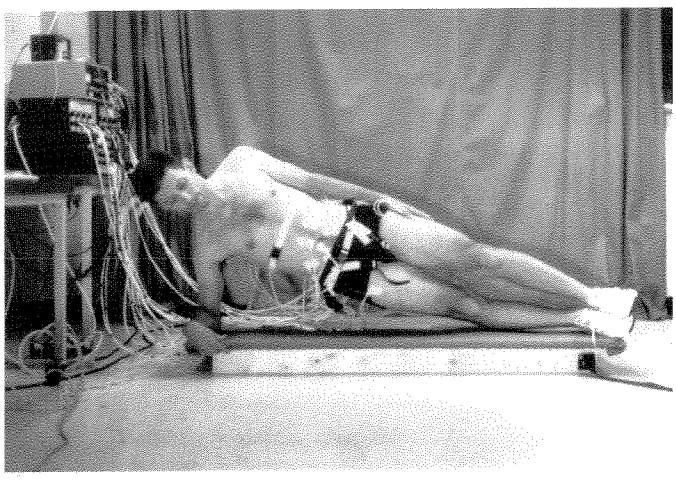

Figure 5.
The isometric side-support exercise. Modifying this isometric hold by supporting the lower body with the knees on the floor reduces the demand further for those cases of more serious concern.

Table 3. An example of tissue load distribution during the horizontal isometric side support exercise. (The lumbar spine was in a neutral posture minimizing passive tissue deformation). Lumbar lateral moment: 107Nm, Compression: 2772N.

	Force (N)	Moment (Nm)			Compression (N)	Shear (N)	
		flexion	lateral bend	twist		A-P	LAT
Muscle							
R rectus abdominis	231	-27	12	5	227	44	-32
L rectus abdominis	52	-6	-3	-1	51	10	7
R external oblique 1	107	-0	10	5	99	47	5
L external oblique 1	9	-0	-1	-0	8	4	-0
R external oblique 2	352	-34	31	25	266	202	-131
L external oblique 2	30	-3	-3	-2	23	17	11
R internal oblique 1	116	2	11	-8	97	-57	12
L internal oblique 1	28	0	-3	2	24	-14	-3
R internal oblique 2	249	-20	17	-18	176	-49	166
L internal oblique 2	60	-5	-4	4	43	-12	-40
R transverse abdominis	92	-5	-0	-9	5	-57	71
L transverse abdominis	10	-1	0	1	1	-6	-8
R pars lumborum (L1)	64	5	3	1	62	12	8
L pars lumborum (L1)	17	1	-1	-0	17	3	-2
R pars lumborum (L2)	79	6	4	1	78	15	11
L pars lumborum (L2)	22	2	-1	-0	21	4	-3
R pars lumborum (L3)	91	5	4	0	84	-21	25
L pars lumborum (L3)	25	1	-1	-0	23	-6	-7
R pars lumborum (L4)	96	4	4	-1	81	-42	24
L pars lumborum (L4)	26	1	-1	0	22	-12	-6
R iliocostalis lumborum	141	11	10	-0	141	8	-4
L iliocostalis lumborum	93	7	-6	0	93	5	3
R longissimus thoracis	225	18	9	-2	223	13	-24
L longissimus thoracis	149	12	-6	1	148	9	16
R quadratus lumborum	83	3	6	2	78	32	4
L quadratus lumborum	23	1	-2	-1	21	9	-1
R latissimus dorsi (L5)	25	1	1	-1	21	4	-12
L latissimus dorsi (L5)	13	1	-1	0	11	2	6
R multifidus 1	84	4	2	2	76	7	37
L multifidus 1	23	1	-1	-1	21	2	-10
R multifidus 2	84	4	2	0	79	31	0
L multifidus 2	23	1	-1	-0	22	8	0
R psoas (L1)	91	-2	5	2	90	16	20
L Psoas (L1)	22	-0	-1	-0	21	4	-5
R Psoas (L2)	91	-1	5	2	90	16	22
L Psoas (L2)	22	-0	-1	-0	21	4	-5
R Psoas (L3)	91	-1	5	1	91	16	25
L Psoas (L3)	22	-0	-1	-0	21	4	-6
R Psoas (L4)	91	-1	4	1	91	16	29
L Psoas (L4)	22	-0	-1	-0	21	4	-7
Ligament							
Anterior Longitudinal	0	0	0	0	0	0	0
Posterior longitudinal	0	0	0	0	0	0	0
Ligamentum flavum	3	0	0	0	3	0	0
R Intertransverse	0	0	0	0	0	0	0
L intertransverse	0	0	0	0	0	0	0
R articular	0	0	0	0	0	0	0
L articular	0	0	0	0	0	0	0
R articular 2	0	0	0	0	0	0	0
L articular 2	0	0	0	0	0	0	0
Interspinous 1	0	0	0	0	0	0	0
Interspinous 2	0	0	0	0	0	0	0
Interspinous 3	0	0	0	0	0	0	0
Supraspinous	0	0	0	0	0	0	0
R lumbodorsal fascia	0	0	0	0	0	0	0
L lumbodorsal fascia	0	0	0	0	0	0	0
Passive tissues							
Disc	0	1	0	0	0	0	0
Gut, etc.	0	2	0	0	0	0	0

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