

The assessment of spine movement dysfunction by a  
commercial dynamometer, EMG and an EMG assisted model.

by

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## Abstract

The purpose of this thesis was to determine whether the understanding of spine movement dysfunction, as indicated by abnormal displacement, velocity, and torso moment data measured by a clinical evaluation system (Isostation B-200), was augmented by a flexion-extension task and the knowledge of the EMG activity from select abdominal and back extensor muscles and/or by knowledge of individual torso tissue forces, estimated from an EMG assisted, dynamic, three dimensional spine model. Because individuals symptomatic for low back pain could not produce true Maximal Voluntary Contractions (MVCs) which were needed for scaling the model's EMG inputs, a method was developed which successfully replaced the MVC scaling factor by an EMG-to-Force ( $EMG_{Force}$ ) scaling factor. Model outputs using the MVC and  $EMG_{Force}$  methods were compared for 10 asymptomatic and 4 symptomatic males for a freestyle flexion-extension task. The  $EMG_{Force}$  method produced significantly lower compressions and flexor muscle forces, but no difference was found in extensor muscle force.

The  $EMG_{Force}$  method was then used to investigate the understanding of spine movement dysfunction. Four males symptomatic (SYMP) for recurrent low back pain and 10 asymptomatic (ASYMP) males were each evaluated on two days. The SYMP group were tested on days they identified as "Good" or "Bad". Each day they were tested using the Isostation B-200 and performed a flexion-extension task with hand loads of 0, 5 and 10 kg. The B-200, EMG and model output data from the ASYMP group were used to develop custom profiles for each of these methods. Muscle force data, presented as an Amplitude Probability Distribution Function (APDF) quantified differences between the groups by determining the amount of time that the forces were above a criterion level. Improved functionality was associated with decreases in

excessive spinal flexor and/or extensor muscle force production and the flexion component of the flexion-extension task was better for distinguishing an SYMP individual from the ASYMP group. The EMG profiles (mV, %MVC) did not distinguish the SYMP group or reveal the improved function. Performance profiles for the B-200, EMG and the model were found to augment the understanding of spine movement dysfunction.

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## **Dedication**

To:

Rebecca Ann Frazer: hundred - hundred.

Gerry and Gloria Frazer: You taught me how to love.

Beverly Mae Frazer: I think of you often and wonder, "What if ... ?".



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# **Chapter I**

## **Introduction**

### **Overview of Thesis Format**

This thesis addresses the issue of spine movement dysfunction and was approached through a series of three investigations. Each investigation is intended to be a stand alone publication and is reported in an individual chapter. This approach produces a certain degree of repetition, especially in the methods section .

The overall purposes of the studies were to determine whether the understanding of spine movement dysfunction, as indicated by abnormal displacement, velocity, and torso moment data measured by a clinical evaluation system (Isostation B-200), was augmented by a flexion-extension task and the knowledge of the EMG activity from select abdominal and back extensor muscles and/or by knowledge of individual torso tissue forces estimated from an EMG assisted, dynamic, three dimensional spine model. Chapter I introduces the problem and defines the issues. Chapter II is a more thorough review of the literature pertaining to these issues.

In order to use the biological model developed by McGill (1992) it was necessary to find an alternative to the maximal voluntary contraction (MVC) method used for obtaining a muscle's maximal EMG amplitude. The MVC method is problematic because individuals symptomatic for low back pain cannot produce the true MVCs which are necessary for scaling the model's EMG inputs. Chapters III and IV report on two approaches investigated to overcome this particular problem.

Chapter V is an extensive investigation which directly addresses the purpose of the thesis. The three studies are then discussed in Chapter VI. This chapter also contains a summary and list of suggestions for future research.

### **Introduction**

The mechanisms of back injury are not well understood. Knowledge of the distribution of tissue forces in injured and uninjured spines in response to external loading is required if injury mechanisms are to be comprehended. The understanding of injury mechanisms is prerequisite to the development of effective prevention and rehabilitation methods (Norman, 1992).

At some point in their life, 80% of the adult population will have an episode of acute low back injury. Typically, 70-80% of these episodes will resolve within six weeks of injury (Nachemson, 1976, Spitzer et al., 1987). The probability of work return decreases from 50% after 6 months absence to practically 0% after 2 years absence (Rowe, 1983). The economic cost of low back injury in the United States has been estimated to be between \$16 and \$50 billion each year (Frymoyer, 1990). These costs are not normally distributed as over 80% are accounted for by only 10% of the injured population (Spengler et al., 1986).

Generating a specific diagnosis, and identifying the source of spine movement dysfunction, from symptoms is difficult. At least eleven structures for each motion unit are capable of producing low back pain in response to mechanical injury and/or chemical irritation (White and Panjabi, 1990). As a result, the diagnostic process has become overwhelmed by the sheer number of "diagnoses". These terms are not well defined and the specificity of some

"diagnostic" tests is questionable (Nachemson, 1992). Patients may receive two or three diagnoses for the same symptoms depending on the focus of the health care practitioner (Spitzer et al., 1987). Bigos et al., (1986) reported that only 12 - 15% of back problems had physical findings that indicated the exact cause of symptoms. The other 85% were classified as "idiopathic" or "nonspecific". Triano et al. (1993) used the forced descriptive categories of entrapment, mechanical or muscular back pain to avoid the dilemma of determining the exact pathoanatomical basis of a patient's complaint. Marras et al. (1993 and 1995) have used the motion parameters of trunk velocity and acceleration in conjunction with a specific test protocol as a means to quantify and classify spine movement dysfunction. Stage one of their two stage model correctly classified more than 94% of 510 individuals as either being healthy or having a low back disorder. The second stage of the model was found to reasonably classify (30% error rate) the individuals suffering from a low back disorder into one of ten low back disorder classification groups.

Regardless of the classification system used, the "traditional" treatment of acute low back pain with decreased movement (i.e. prescribed bed rest) greater than 1-2 days appears to be of little use (Deyo et al., 1986). Rather, maintaining or achieving movement of the structures in the lower back, is now the desired rehabilitation outcome (Deyo et al., 1986, Waddell, 1987). A common clinical goal that conservative treatment methods have focused on is the return of an individual's "abnormal" function to "normal". Typically this is achieved through the use of modalities such as flexibility, strengthening and/or manipulation (specific, controlled movements of the spine) as in the chiropractic approach (Vernon, 1991). Therefore, some

clinicians use the degree of normal movement or obvious movement disability as "measures" of treatment outcome rather than dwelling on the pathoanatomy per se.

Decreased spinal strength and velocity of movement are characteristic observations made of individuals symptomatic for low back pain. Commercial, computerized dynamometers have been designed to measure and quantify these parameters for the lumbar spine. They are becoming a routine component of the assessment and rehabilitation procedure (Spengler and Szpalski, 1990). A "Back Dysfunction" rating, to help classify individuals, can be produced, by utilizing a custom clinical evaluation system software package in conjunction with a specific dynamometer, the Isostation B-200 (Deutsch, 1991). The Back Dysfunction rating and other performance data may also be used as a guide in the rehabilitation process, to assist in determining when an individual has regained "normal" function.

The "Back Dysfunction" rating must be interpreted with caution. Back dysfunction, is determined by abnormal displacement, velocity and torso moment data output during specific directions of movement (e.g. flexion, rotation to the right). The rating is not a diagnostic measure, but simply a composite outcome measure of an individual's response, given their functionality at the time of the test, to working against external loads. By concentrating on the magnitude of the peak moment and velocities of each test, it ignores the moment time history that produced the movements. Also, the software compares absolute, not relative peak moments when quantifying back dysfunction. Individuals symptomatic for low back pain may have decreased strength. Thus, an individual may have an absolute value for a peak torque which is considered abnormal, but when expressed as a percentage of their own strength, it may indeed be in the normal range. Also, in trying to understand the changes that occur with

regaining normal function, analysis of the moment-time histories may be more important than simply comparing peak values.

Using a dynamometer may aid in the identification of dysfunctional spine movements, but the location of the dysfunction (e.g. right side) and the cause(s) of the abnormal displacements, velocities and torso moments remain unidentified. To answer these questions requires knowledge of the functional nature of the muscles that contribute to the movement.

Electromyography (EMG) has been used to study the response of the extensor musculature to sustained isometric muscle contractions for individuals asymptomatic and symptomatic for low back pain. The observation of electrical silence (flexion-relaxation phenomenon) at the end range of flexion found in asymptomatic individuals has been found absent in 20 - 45% of individuals with low back pain (Floyd and Silver, 1955, Triano and Schultz, 1987).

The assessment of EMG amplitudes in the spinal musculature of symptomatic and asymptomatic low back pain individuals during the performance of static and dynamic tasks has produced mixed findings. In comparing the EMG amplitudes between these two groups, researchers have found no differences (Nouwen et al., 1987), increased amplitude (Arena et al., 1989) and decreased amplitude (Ahern et al., 1988) for the asymptomatic group. Each of these studies represented the task by a single EMG value (e.g. mean EMG), which ignores the time history of the EMG signal. Sutarno (1993) investigated the kinematic and EMG time histories of symptomatic and asymptomatic people performing a flexion-extension task, but had difficulty categorizing the low back pain individuals. This may have been due to the size of the load and the fact that only one subject was in pain at the time of testing. The assessment of

individuals at different periods during the time course of their symptomatic period may enhance both the classification of individuals and provide more insight into muscular function and low back pain.

Changes in the EMG power spectrum have been used to differentiate between individuals symptomatic and asymptomatic for low back pain. Moritani et al., (1992) found that the mean power frequency declined faster for those with a history of low back pain than for controls. Back pain subjects also had an asymmetry in fatigue rates between their left and right lower erector spinae. Unfortunately, the relationship between the asymmetry and the site and/or side of the low back pain was not explored. De Luca and colleagues have also utilized sustained isometric contractions and the power spectrums from multiple electrode sites to successfully distinguish between healthy subjects and individuals with low back pain (Roy et al., 1989, Roy et al., 1990, De Luca, 1993).

It appears then, that the EMG power spectrum may be used to classify individuals with and without low back pain. It may also assist in localizing the problem area and augmenting the understanding of the effects of injury on some aspects of muscular performance. However, this method is limited to the performance of static contractions. To-date, it cannot be used to identify the specific impairment associated with the dynamic activities of daily living, such as flexion-extension.

Monitoring the force time histories of the spinal musculature is another method that could be used to assess muscle function. Tissue forces in the lumbar spine may be estimated using sophisticated computerized spine models (McGill and Norman, 1986, McGill, 1992, Marras and Sommerich, 1991a, 1991b). McGill (1992), developed a three dimensional

dynamic model which uses EMG as a biological input signal in order to partition the restorative moment into force/time histories for 50 muscle fascicles, 12 ligament vectors and the compression and shear forces acting on the L4/L5 motion unit. An earlier version of the model (McGill and Norman, 1986), found motion unit compression to be appreciably reduced to well below failure levels as compared to compression predicted from a rudimentary, single equivalent erector tissue model with a 5 cm moment arm. Output from this model was also used as the basis for a hypothesis about the source of sacroiliac pain (McGill, 1987).

Knowledge of the force/time histories for ligament and muscles may allow the determination of a more precise identification of dysfunctional structures in individuals with low back pain. However, because maximal voluntary contractions are required to normalize the EMG input, the model has never been utilized with a spine movement dysfunction population symptomatic for low back pain. Therefore, it is not known if individuals with spine movement dysfunction have tissue force/time histories different than normals, or what the effects of recovery would be on the force/time histories.

The goal of this thesis is to integrate the commercial dynamometer, electromyography and a sophisticated model of the spine to learn more about spine movement dysfunction in individuals asymptomatic and symptomatic for low back pain. Assessment of the entire moment time history produced during low back dynamometer testing will allow an enhanced assessment of the "normal" parameters and facilitate the identification of "abnormal" patterns. Assessment of EMG time histories during the performance of a dynamic activity of daily living task may provide further insight into the muscular function. The spine model, which incorporates EMG as an input, will allow muscle force-time history patterns to be measured for



dynamic activities. By evaluating asymptomatic individuals twice, the amount of variability between test sessions may be documented for each of the evaluation techniques. This will assist in evaluating the responses of the symptomatic individuals when measured at intervals during the recovery period. The application of these measures may provide insight into the muscular source, as well as the effects, of spine movement dysfunction.

### **Statement of Purpose**

The purpose of this study was to determine whether the understanding of spine movement dysfunction, as indicated by abnormal displacement, velocity, and torso moment data measured by a clinical evaluation system, was augmented by a flexion-extension task and the knowledge of the EMG activity from select abdominal and back extensor muscles and/or by the knowledge of individual torso tissue forces estimated from an EMG assisted, dynamic, three dimensional spine model.

### **Research Questions**

Specifically, the following research questions were addressed:

1. Could the dynamic spine model be used for people in pain?

**RATIONALE:** The model required that maximum voluntary contractions be performed to normalize the EMG. These contractions were problematic for individuals with low back pain. Was it possible for asymptomatic individuals to perform sustained, submaximal contractions of sufficient intensity that would induce maximal electrical activation of the flexor and extensor musculature? If so, this would avoid the need to

use the typical maximal voluntary contractions to determine the maximal electrical activation.

2. Was movement dysfunction identification, as documented by abnormal peak displacement, velocity and torso moment data output from a commercial dynamometer (i.e. the Isostation B-200) confirmed and/or augmented by the assessment of the entire moment-time history?

**RATIONALE:** Displacement, velocity, and moments may be obtained from a variety of dynamometers. The Isostation B-200 is a specific commercial dynamometer that utilized this information to produce a "Back Dysfunction" rating. Individuals with low back pain have been found to produce abnormally low magnitudes of peak torque production in primary and secondary axes during isometric and dynamic testing. But this comparison was made using absolute values, which did not account for strength differences, instead of relative values. Did a comparison of the relative moment magnitudes over the whole time history enhance the assessment?

3. Did EMG obtained from select abdominal and back extensor muscles help specify a more precise location of spine movement dysfunction?

**RATIONALE:** The B-200 quantified a level of dysfunction, but it oversimplified the lumbar spine and the tests were not representative of tasks typically encountered in the activities of daily living. EMG has been utilized to assess muscular function. Did EMG profiles constructed from asymptomatic individuals performing a flexion-

extension task provide insight into normal muscle function and serve as a reference for symptomatic individuals, assisting in providing a more specific identification?

4. Was a more precise identification of the region of the back involved in the movement dysfunction, if not which tissue, possible, using the information provided by the spine model?

**RATIONALE:** The spine model provided force/time histories for 50 muscle fascicles, 12 ligaments and compression and shear forces acting on the L4/L5 motion unit. Did this level of knowledge provide more specific identification of dysfunctional structures?

5. What were the effects of recovery from spine movement dysfunction on the phase and magnitude of the lumbar spine model tissue force/time histories during isometric and dynamic contractions and did these force-time profiles provide more information than either the EMG or B-200 alone?

**RATIONALE:** The EMG assisted, dynamic, three dimensional spine model partitioned the reaction moments produced by a linked segment model into the restorative moments generated by the 50 muscle fascicles, 12 ligamentous components, and the non-linear elastic intervertebral disc. Muscle fascicles that were functionally similar were assigned activation patterns from common surface EMG electrodes. Therefore, if changes in muscle function occurred with recovery, the changes should be observed in the model muscle force outputs. Also, if changes in muscle function did occur, these may have been observed in the EMG profiles and the B-200 profiles.

### **Subproblem**

- I. What were the specific resources necessary for each method?

RATIONALE: this allowed a cost/benefit analysis to be performed so that for a specific scenario (e.g. clinical practice, research laboratory), the most appropriate method could be selected.

To answer these questions and address these subproblems, individuals asymptomatic and symptomatic for low back pain performed a series of static and dynamic tests. The Oswestry Low Back Disability questionnaire and a visual analog pain scale were given to all participants. Isometric, maximal voluntary contractions and sustained, submaximal isometric contractions were used in an attempt to elicit maximal EMG electrical activation levels. The dynamic testing incorporated a standing, flexion/extension task with and without loads. A standard clinical evaluation testing protocol was performed by all participants. The EMG from six bilateral muscle groups (i.e. 12 channels), trunk kinematics, the tissue force/time histories from a spine model and the position, velocity and moment data from the B-200 were analyzed to determine differences between the asymptomatic and symptomatic individuals. Each asymptomatic person was tested twice. The asymptomatic individuals' data provided both normative values for each day of testing and measures of variability between test days. The symptomatic individuals were tested at two points of time during the course of their recovery from dysfunction in order to determine the effects of recovery.

### **Assumptions**

1. Not all of the muscles for which forces were estimated could be monitored by surface EMG (e.g. psoas). It was assumed that the EMG-time history from agonist musculature (e.g. internal oblique for psoas) satisfactorily represented these muscles. This assumption is supported by data from McGill et al. (1996) who found that well selected surface electrode locations did provide a representation of deeper muscle EMG activity, with RMS differences of 2-15% MVC RMS difference found during the performance of clinical tasks.
2. It was assumed that each lumbar joint accounts for a constant proportion of flexion. Therefore, external measures of spine kinematics were used to measure the rotations at individual lumbar levels.
3. It was assumed that muscle forces may be approximated by using estimates of their length, velocity and linear envelope electromyogram in conjunction with appropriate low pass filtering of the electromyogram.
4. During the performance of the isometric and dynamic flexion-extension tasks, kinematic symmetry was assumed between the left and right sides of the body.
5. As a result of sufficient rest periods between test sessions, the subjects were not fatigued.

### **Limitations**

1. Conclusions were limited by the number of, age of, and to the type of subjects recruited for this study.
2. The Isostation B-200 was a novel testing device for the lumbar spine. This limited the test results to this specific type of lumbar spine dynamometer.
3. The dynamic contractions were of a small duration and intensity. It was not anticipated that fatigue would result. However, if it did, it was not be possible to correct the EMG signals for the effects of fatigue.

## Chapter II

### Review of Literature

#### Diagnosis

The process of diagnosis for individuals with low back pain has become a categorization process that should fully consider the aetiogenesis and prognosis of the disorder (Troup and Videman, 1989). However, the diagnostic process has become overwhelmed by the sheer number of "diagnoses" and their inconsistent application. Lumbar strain, lumbar sprain, lumbago, sciatica, facet syndrome, ligamentitis, myofasciitis, sacroiliac joint dysfunction, degenerative disk disease, segmental instability and low back pain of idiopathic origin, to name but a few, are very common diagnoses. These terms are not well defined and the specificity of some "diagnostic" tests is questionable (Nachemson, 1992). A patient may present with two or three different diagnoses for the same symptoms, simply by having consulted multiple health care practitioners (Spitzer et al., 1987). The application of strict diagnostic criteria does not improve matters. Bigos et al., (1986) reported that only 12 - 15% of back problems had physical findings that indicated the exact cause of symptoms. The other 85% were classified as "idiopathic" or "nonspecific". Triano et al., (1993) utilized the forced descriptive categories of entrapment, mechanical or muscular back pain, to avoid the dilemma of determining the exact pathoanatomical basis of a patient's complaint.

The assessment of higher order trunk motion characteristics has been found to facilitate the assessment of individuals with low back disorders. Marras et al. (1993, 1995) have used the parameters of trunk velocity and acceleration in conjunction with a specific test protocol as a means to quantify and classify spine movement dysfunction. Individuals performed flexion

and extension efforts in five transverse plane trunk postures. Ignoring the initial flexion and extension cycle and averaging the subsequent cycles allowed 14 trunk motion characteristics to be measured for each posture. An eight variable motion component model was developed that incorporated trunk motion characteristics from each of the movement planes. To account for the interaction of the variables, four different evaluation techniques were used to measure the success of the model's classification of the study participants. Stage one of the two stage model correctly classified more than 94% of 510 individuals as either being healthy or having a low back disorder. The second stage of the model was found to reasonably classify (30% error rate) the individuals suffering from a low back disorder into one of ten low back disorder classification groups. This method may eventually be used as a tool to help diagnose low back disorders.

The "stage" of low back injury is often described as acute, sub-acute, or chronic. Until recently, there has been no consistent definition of these terms. They are now defined based on the duration of absence from work: acute (fewer than seven days); sub-acute (seven days to seven weeks) and chronic (more than seven weeks) (Spitzer et al., 1987). Recurrent low back pain has been added to these stages and is defined as a four to six week symptom free period, prior to the current episode, with more than six episodes of pain within the last year (Triano et al., 1993).

### **Functional Anatomy**

A functional spinal unit (FSU) consists of the superior and inferior vertebrae, the connecting intervertebral disc and ligaments (White and Panjabi, 1990). Each of these



structures, and the surrounding musculature, are innervated by nerves capable of relaying sensations of discomfort when stimulated by a mechanical and/or chemical stimulus (White and Panjabi, 1990). Understanding of the functional anatomy of the spine is imperative for the interpretation of spinal model output and the understanding of low back injury mechanisms.

### **Vertebrae**

A vertebra consists of an anterior block, the vertebral body, and a posterior bony ring, the neural arch. The vertebral body of the lumbar spine is cylindrical in shape, wider in the coronal plane than the sagittal and the vertebral body increases in size from L1 to L5. The neural arch contains the oval shaped pedicle of the lumbar spine, which arises from the superior and posterior lateral border of the vertebra. The spinous process projects almost directly posterior from the vertebral body. The two superior facets are positioned laterally of the two inferior facets, so that the inferior facets from the superior vertebra articulate inside the superior facets of the inferior vertebra (Miely et al., 1990).

This articulation is a facet or zygapophyseal joint. These synovial joints permit vertebral articulation and serve as stabilizing structures to protect the spine against torsional damage. Depending on the posture and loading rate, the facets also resist anterior shear and share a percentage of spinal loading, (White and Panjabi, 1990).

### **Intervertebral Disc**

The intervertebral disc consists of a gelatinous nucleus pulposus encompassed by a laminated, annulus fibrosus and is situated between the cartilaginous endplates of the superior and inferior vertebrae. The nucleus pulposus fills 30-50% of the disc volume and its water

content (70-90%), decreases with age (White and Panjabi, 1990). The annulus fibrosus fibers are arranged in concentric layers. The fibers are angled approximately 30° from the horizontal and successive layers slant in opposite directions. The disc is avascular, relying on diffusion due to vertebral loading and unloading for nutrition.

### **Ligament**

The seven ligaments associated with the intervertebral joint are the anterior longitudinal, posterior longitudinal, ligamentum flavum, capsular, intertransverse, interspinous and supraspinous. The role of the ligaments is to protect the spinal cord by restricting motion segment displacement within an adequate physiological range, providing stability to the spine, transferring tensile loads and absorbing large amounts of energy during traumatic situations (White and Panjabi, 1990).

**Anterior Longitudinal (ALL):** This ligament runs the entire length of the spinal column, and is attached firmly to the anterior edge of the vertebral body and loosely to the annular fibers. It consists of three layers. The deep, intermediate and superficial layers connect one, two or three, and three or four vertebral layers, respectively (White and Panjabi, 1990, Miely et al., 1990).

**Posterior Longitudinal (PLL):** This ligament runs the entire length of the spinal column. It has an interwoven attachment with the intervertebral disc and is wider at the disc level than at the intervertebral body (White and Panjabi, 1990, Miely et al., 1990).

**Ligamentum Flavum (LF):** This structure has a paired appearance due to a midline cleavage, and runs from one vertebral lamina to the next. It is yellow in appearance due to the large content of elastin fibers (White and Panjabi, 1990, Miely et al., 1990).

**Capsular (CL):** These ligaments attach on the articular regions of the superior and inferior facets and blend medially with the ligament of flavum. The fibers are generally oriented perpendicular to the plane of the facet joints (White and Panjabi, 1990, Miely et al., 1990).

**Intertransverse (ITL):** These ligaments span the transverse processes. In the lumbar region these ligaments are thin and membranous (White and Panjabi, 1990, Miely et al., 1990).

**Interspinous (ISL):** The interspinous ligament is bilateral and consists of a ventral, middle and dorsal part. Its fibers transverse the interspinous space in a posterocranial direction (Heylings, 1978).

**Supraspinous (SSL):** The supraspinous ligament attaches to the tips of the spinous processes and is thick and well developed in the lumbar region (White and Panjabi, 1990, Miely et al., 1990). Heylings (1978), found this ligament not to extend caudally beyond L5. Caudal to this, its position is taken by the most medial tendon of the erector spinae.

## **Muscle**

The musculature in the region of the lumbar spinae is commonly referred to as the erector spinae. The work of Macintosh and Bogduk, (1987) and Macintosh et al., (1986) reveals that the erector spinae really consists of three distinct muscle groups, the multifidus (M), longissimus thoracis (LT), and iliocostalis lumborum (IL). The IL and LT can each be subdivided into two distinct sections, the pars thoracis (Pt) and pars lumborum (Pl). This produces five distinct muscle groups, the M, LTpT, LTpL, ILpT and ILpL.

**Multifidus (M):** The multifidus can be divided into five distinct bands. Each band has fascicles that are contiguous rostrally and arise from the tip of a spinous process, its lateral surface and the vertebral lamina. At L1, the deepest and shortest fibers insert into the vertebral mammillary process of L3. The next layer of fibers insert at L4, the next layer of fibers at L5 and the last layer of fibers attaches to the sacrum. This origin/insertion pattern is repeated for each of the remaining lumbar vertebra and the number of layers inserting on the sacrum increases for each successive vertebra (Macintosh et al., 1986). The caudal attachment point for each band of fascicles is almost directly beneath their origin on the spinous processes, making extension of the lumbar spine the primary action of multifidus (Macintosh and Bogduk, 1986).

**Longissimus Thoracis pars Thoracis (LTpT):** The LTpT arises from the thoracic transverse processes (T1 - T3,4) and ribs (T3,4 - T12). The fascicles arising from T1 - T6 attach caudally to the lumbar spinous processes. The fascicles arising from T7 - T12 attach caudally to the

sacral spinous processes, the dorsal aspect of the fourth sacral segment (Macintosh and Bogduk, 1987). Lumbar spine extension is produced by bilateral activation while unilateral activation produces lateral flexion.

**Longissimus Thoracis pars Lumborum (LTpL):** The LTpL consists of five fascicles that arise from the accessory and transverse processes of the lumbar vertebra (L1 - L5) and converge onto the posterior-superior iliac spine (L1 - L4). The L5 fascicle attachment point is the ventromedial surface of the ilium (Macintosh and Bogduk, 1987). Bilateral activation produces extension of the lumbar spine. Unilateral activation induces a small ipsilateral lateral flexion, and rotation.

**Iliocostalis Lumborum pars Thoracis (ILpT):** The ILpT arises from the ribs (T5 - T12) and attaches to the iliac crest in a medial to lateral order (T5 - T12) (Macintosh and Bogduk, 1987). Lumbar spine extension is produced by bilateral activation while unilateral activation produces lateral flexion.

**Iliocostalis Lumborum pars Lumborum (ILpL):** The ILpL consists of four fascicles that arise from the lateral one quarter of the transverse process of the lumbar vertebra (L1 - L4) and the adjacent thoracolumbar fascia. Each fascicle attaches caudally to the iliac crest (Macintosh and Bogduk, 1987). Bilateral activation produces extension of the lumbar spine. Unilateral activation induces a small ipsilateral lateral flexion, and rotation.

## **Mechanisms of Injury**

Tissue injury results when the magnitude of the tissue load is greater than the tissue tolerance. This can occur via the application of a single excessive load, cyclical loading with subcritical loads or subcritical loads sustained over a period of time. For the subcritical loads, the rate of damage may exceed the rate of repair, resulting in tissue failure under mildly abnormal loads (Goel et al., 1988, McGill, 1995). Due to the viscoelastic nature of bone, ligament, tendons and passive muscle, the rate of loading must also be considered as an injury mechanism (White and Panjabi, 1990).

## **Vertebra**

The compression strength of the vertebrae increases from C1 to L5. A sharp decrease in strength occurs after 40 years of age, primarily due to the decrease in osseous content. Every unit of decrease in osseous tissue content, produces a two fold decrease in compressive strength. The resultant central or peripheral endplate fracture of a compressed FSU depends on the nucleus pulposus' undegenerated, or degenerated, state. In either case, the annulus fibrosis is not damaged (White and Panjabi, 1990).

Yang and King (1984) found the facets transmitted between 3 - 47% of the applied load, depending on the posture and the functional integrity of the FSU. They also found that excess facet loading caused the inferior facet to pivot about the pars, stretching the joint capsule. The facets provide approximately 45% of the torsional strength (White and Panjabi, 1990) and cyclic torsional loads produce both disc and facet joint damage (Goel et al., 1988). Posterolateral disc injury has been proposed to alter facet joint asymmetry, leading to facet

cartilage degeneration, osteoarthritis, facet atrophy and intervertebral foramen narrowing (Panjabi et al., 1984).

### **Intervertebral Disc**

Simple disc compression, thought to be the cause of disc herniation and its associated pain, is more likely to result in end plate fracture. Torsion has been shown to be the mechanism required to injure the annulus fibers (Farfan et al., 1970). Disc herniation was found to occur by sudden compression of a fully flexed, laterally bent, FSU (Adams and Hutton, 1982). The compressive strength of the lumbar spine is affected by gender and age. At 40 years of age, the strength is approximately 6700 N for males, and 4700 N for females. Compressive strength decreases 1000 N and 600 N per decade, for men and women respectively (Jager and Luttmann, 1992).

Clinically, the posterior or posteriolateral portions of the disc are the most common sites of disc herniation, which is thought to be preceded by micro-tearing of the annulus fibers. Fissures in the annulus are present by 30 years of age (Holm, 1990), decreasing their capacity to contain the nucleus pulposus (Wiesel et al., 1985).

Panjabi et al., (1984) found that annulus injury and nucleus removal significantly altered the main motions, the coupled motions and the creep response of the FSU. Right posterolateral injury resulted in asymmetrical FSU motion and increased the range of spinal movement but did not affect the rate of creep. McGill and Brown (1992), measured the 50% recovery time for creep, induced in normals by prolonged flexion, to be two minutes.

Increased spinal movement due to injury and increased joint laxity due to creep effects, could combine to further increase the risk of hyperflexion trauma.

Very little is known about the effects of shear. Load shear represents the shear force produced by the weight of the load and body before the offsetting shear forces produced by the extensor musculature are considered. Jager and Luttmann (1992) report small displacement, but no damage occurring for 200 N of shear, but total rupture for 7400 N. However, biomechanical models provide insight into the magnitude of shear produced by lifting loads. In 1988, McGill and Norman studied the effects of a detailed anatomical model on the shear forces produced in response to a L4/L5 moment of 227 N·m. Improved anatomical detail was capable of reducing the magnitude of shear from 565 N with L4 shearing anteriorly on L5, to 200 N with L4 shearing posteriorly on L5, as a result of support by muscle activity. The 35% reduction illustrates the substantial contribution made by the musculature. Potvin et al., (1991), found shear forces ranged from a squat lift maximum of  $194 \pm 136.3$  N at 22 kg, to  $483 \pm 279.1$  N at 22 kg for stoop lifts (L4 shearing posteriorly on L5). Cholewicki et al., (1991), used a static, two dimensional computerized model to study female and male powerlifters during a national championship. The mean loads of 145.8 kg for females and 256.7 kg for males, produced load and joint shears of 1666 N and 1107 N respectively, for females, and 2832 N and 1739 N for males.

### **Ligament**

In the neutral position, the ligaments provide only minimal stability. It is near the physiological end ranges of motion that the ligaments play a major role.



**Posterior Longitudinal (PLL):** The PLL is strained equally by flexion and lateral bending (Panjabi et al., 1982).

**Ligamentum Flavum (LF):** The LF's high elastin content and prestress allows it to resist extreme flexion of the spine (Hukins et al., 1990). The paired structure of the LF means that both sides are strained in flexion. For lateral bending to the right, the left LF is strained more than the right LF. This pattern reverses for right lateral flexion. Minimal strain is induced by left or right rotation (Panjabi et al., 1982).

**Capsular (CL):** The coupled motion of the FSU produces strain in both the left and right CL during flexion and extension. Right rotation produces maximal strain in the right CL but no strain in the left CL. However, a right lateral bend produces minimal strain in the right CL and a strain in the left CL greater than extension (Panjabi et al., 1982). Anderson et al., (1985) predicted strains at 100% of L5/S1 flexion for the CL of 101.3%. Inaccurate modeling of the CL was cited by the authors to produce its unrealistically high strain.

**Supraspinous (SSL) and Interspinous (ISL):** The SSL and ISL are the most strained ligaments during flexion. Simulated flexion of excised spinous processes found SSL and ISL load transmission only towards the end range of motion (Hindle et al., 1990). SSL removal showed the ISL capable of handling 75% of the load. Although the entire FSU was not tested, these observations are consistent with the model predictions of McGill (1988). Adams and Hutton (1982) found the SSL and ISL the first structures damaged with hyperflexion of the

joint. Hukins et al., (1990) dismissed the SSL and ISL role of resisting flexion. Based on the SSL's collagen structure and lack of tensile stiffness, its described role is to simply act as an anchorage site for the erector spinae tendons. The ISL collagen fibre orientation, determined from x-ray diffraction, was "roughly parallel to the spinous process", "so that they do not stiffen the ligament as it is stretched during flexion of the spine". Their function for the ISL is to anchor the thoracolumbar fascia. Closer examination of "roughly parallel" shows that the arrangement is fanlike "about an axis parallel to the spinous process". This description actually describes the function of ISL perfectly. Flexion of a superior vertebrae would increase the angle of the axis, resulting in an increase in both the ISL's ability to resist flexion and the amount of anterior shear produced.

Anderson et al., (1985) predicted strains at 100% L5/S1 flexion for the SSL/ISL of 8.8%. Modeling the SSL/ISL as one structure is likely the source for its low strain prediction. McGill, (1988), found the L4/L5 ISL to contribute the greatest flexion resisting forces and to undergo the largest stress and strain. At full flexion, the ISL strain was within reported failure limits. Although the location selected for the ISL may have been responsible for these predictions, the large strains were cited to match well with the clinical observations regarding the incidence of ISL failure.

**Anterior Longitudinal (ALL):** The ALL is strained in extension and lateral bending (Panjabi et al., 1982).

## Muscle

Many individuals with low back discomfort are suffering from a non-radiating type of low back pain. The cause of the discomfort is thought to be muscular strain or a ligamentous sprain, secondary to a specific traumatic stress, continuous mechanical stress or micro-tearing of the annulus fibrosis (Wiesel et al., 1985). Large moments may be produced about the lumbar spine and modeling output would suggest that the forces required to produce the sufficient moments are primarily muscular. For example, Troup and Chapman (1969) report isometric back extensor strength of  $391 \pm 76$  N·m for men and  $244 \pm 53$  N·m for women. The body has the capacity to both withstand and produce high levels of muscle force, as shown by Cholewicki et al., (1991). How then is muscle injured?

Muscle injuries induced in laboratories have not been achieved by isometric or concentric contractions, but rather passive stretch or *eccentric* actions are required (Frymoyer and Gordon, 1989). Injury induced by an eccentric action would indicate that too much tension has been developed in some section of the myotendinous or osseotendinous junction (Frymoyer and Gordon, 1989).

Muscle injury may be maintained or aggravated by muscle spasm. Spasm is a shortening of a muscle due to nonvoluntary motor nerve activity (Gatterman, 1990). High precontraction metabolite levels due to persistent muscle spasm and prolonged tension may be associated with excessive back muscle fatigue (Armstrong, 1984). Current treatment objectives for low back injury include rest for the affected anatomical structures, and decreasing muscular spasm (Spitzer et al., 1987). Diminishing these protective spasms while eliminating the underlying cause is thought to be beneficial so that pain-spasm-pain cycles are

prevented (Spitzer et al., 1987). The pain-spasm-pain cycle may be self sustaining following injury to the muscle (Gatterman and Goe, 1990). Roland (1986) reviewed the evidence for a pain-spasm-pain cycle in spinal disorders. Experimental evidence could not prove the existence of a pain-spasm-pain cycle, however, a large body of evidence was consistent with a pain-spasm-pain cycle. Pain and spasm did not occur independently. Pathways exist whereby pain causes muscle spasm and muscle spasm causes pain.

### **Pain**

Regardless of the mechanism, tissue injury in the lumbar spine produces low back pain. The anterior and posterior longitudinal ligaments, posterior annular fibers, ligamentum flavum, interspinous ligaments, intervertebral joint capsules, periosteum of vertebrae, fascia of vertebrae, blood vessels of vertebrae, walls of epidural and paravertebral veins and paravertebral musculature are all capable of producing a pain stimulus as a result of either mechanical and/or chemical irritation (White and Panjabi, 1990).

Treatment programs often use the resolution of pain and an increase in reported capability by the patient as outcome measures. Recently, the Oswestry Low Back Pain Disability Questionnaire and the Visual Analogue scale were reported as appropriate, useful, and responsive outcome measures for use with back pain patients (Triano et al., 1993, Von Korff et al., 1992). Although these techniques provide information about the limits of a person's daily activity due to pain, they do not address whether the tissue originally injured has recovered and is capable of generating and/or transmitting force.

## Measurement of Back Function

The range, symmetry and rhythm of spinal motion are of major diagnostic significance (Wiesel et al., 1985). The clinical methods utilized to measure spinal motion include simple observation, inclinometers, electromagnetic sensors, and even sophisticated commercial dynamometers.

The Isostation B-200 (Figure 1) is a tri-axial dynamometer used for the assessment and rehabilitation of individuals with low back pain. It provides resistance and monitors torque, angular displacement and angular velocity for tri-axial low back motion. Isometric contractions may be performed with the three axes locked into a neutral position. Dynamic testing is isoinertial, that is the resistance selected to oppose an individual's effort is kept constant. Only *concentric* contractions are utilized as a testing modality.

The most common clinical evaluation protocol utilizes an individual's isometric torque production in each axis, to select resistance settings for dynamic testing in each axis. A software-driven evaluation system then compares the individual's performance parameters against a database and, based on the number of "abnormal indicators", assigns a level of back dysfunction (Deutsch, 1991). The report also graphically compares the individuals performance against the database standards so that rehabilitation decisions may be made. Although this approach "quantifies" the individual's back dysfunction, it provides no insight into the structures involved.

Implicit to this testing protocol is that an individual *always works maximally*, within their limits (i.e. as hard, as fast and through as large a range of motion as possible). Maximum efforts are assumed to provide a more consistent test result and also highlight or amplify problematic movements. Because subjects are tested at percentages of their "demonstrated capacity", it is assumed that they can produce velocities and relative torques within the

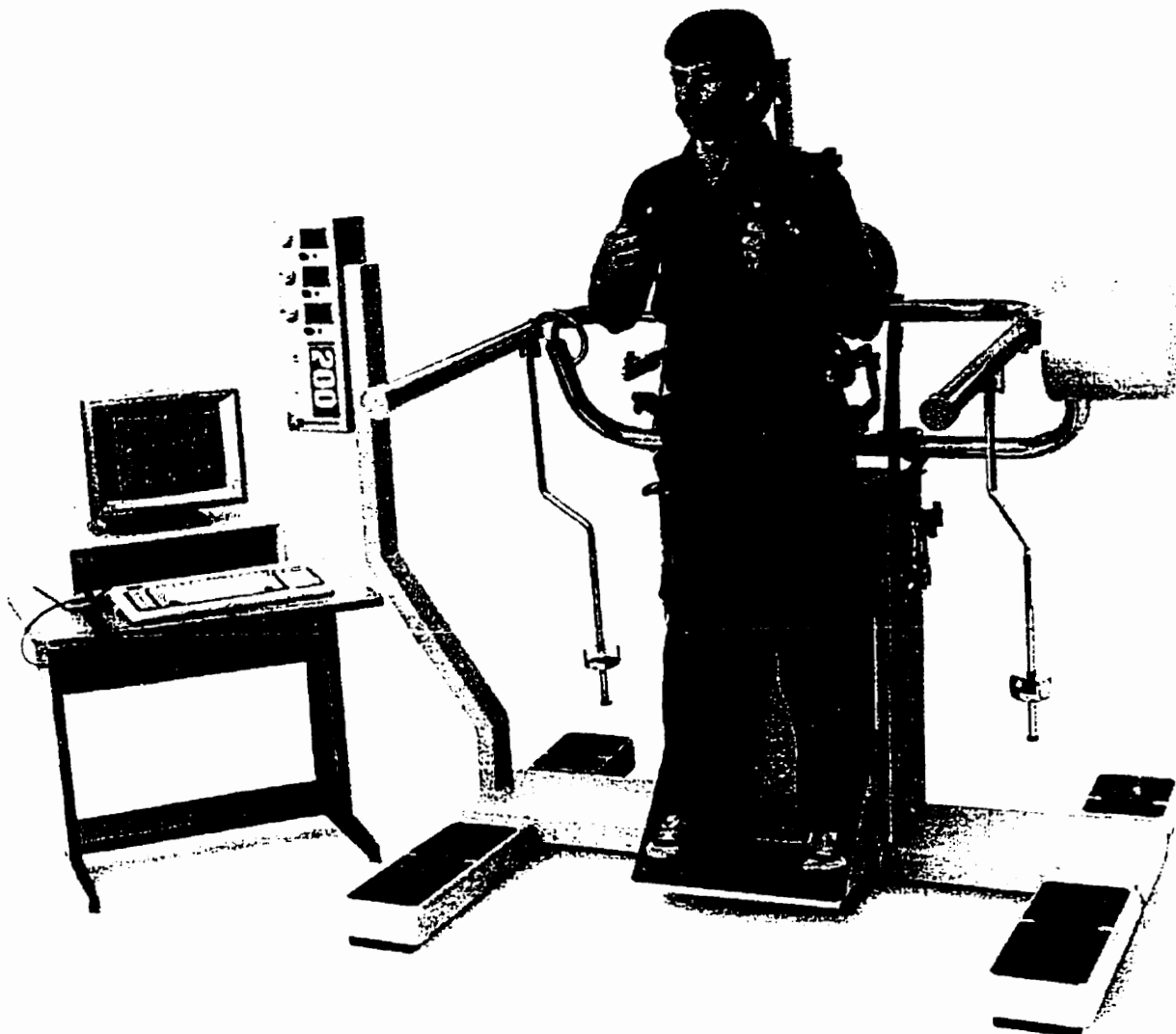


Figure 1: The Isostation B-200 is a tri-axial, lumbar spine dynamometer. Individuals may be tested using isometric or isoinertial resistances. The hydraulic resistance may be set using standard or custom protocols.

"normal" range. Consistent deficiencies are evaluated as low back dysfunctions. The maximal effort protocol masks the subtleties of movement by using peaks and averages determined from the middle three of five repetitions.

This protocol is very lengthy (25 - 40 minutes) to complete. Because a discriminant analysis approach is utilized, all of the tests must be completed before a level of back dysfunction may be assigned. Another limitation of this protocol is that the "secondary" testing axes (those that are not the principle direction of movement) are always set to their maximum resistance level. Although this allows the secondary axes torques generated by the subject to be compared for left versus right side asymmetries, it eliminates any kinematic data from being produced in the secondary axes.

A major design limitation of the B-200 is that the axes that the individual rotates about are not aligned with the anatomical axis of the spine. The measurement is designed to take place about the L5-S1 intervertebral disc, yet the intersection of the three mechanical axes is posterior to the individual, as they stand in the dynamometer. Also, mechanical stops are set in each direction of movement so that individuals may not move to their extreme ranges of motion. This inadvertently restricts ligament loading from occurring.

Due to the restraint system used, the B-200 does not load the spine in a "natural" fashion. Frazer and Norman (1993) found large levels of co-contraction in isometric activities, a required testing mode in the B-200. They questioned if the machine constraints were not increasing forces in other structures of the low back (e.g. intervertebral disc and ligaments). Preliminary data indicates large levels of co-contraction in isometric activities (a common testing mode in the B-200).

User designed "custom" protocols can be utilized within the B-200 software. This restricts the amount of normative data available for clinical assessment, but it does provide a great deal of research flexibility. Parnianpour et al. (1988) used the a custom B-200 protocol to quantify low back fatigue and concluded that fatigue caused the spinal structures to be loaded in more injury prone configurations. This conclusion was based on activity occurring in the "secondary" testing axes.

Patients with low back pain have been found to produce less secondary axis activity than normals (Deutsch, 1991, McIntyre and Glover, 1993). It was thought that these individuals may be "guarding" their movements.

These research findings and the clinical evaluation protocol have all focused on the peak moments and velocities. By concentrating solely on the magnitude of the resultant peak and/or average moments and velocities, the moment time histories that produced the movements are ignored. Evaluating this aspect of the performance may enhance the assessment protocol even further.

### **Spinal Electromyography**

The musculature of the spine provides stability of the spinal column and controls intervertebral spinal motion. The analysis of myoelectric activity is one of the primary methods for understanding the function of the spine (Frymoyer and Gordon, 1989).



### **Flexion - Relaxation Phenomenon**

The flexion - relaxation (FR) phenomenon is the absence of electrical activity in the back musculature while in a fully flexed posture. This implies that the structural loads are being carried by the ligamentous and articular passive tissues.

Floyd and Silver (1955) investigated the FR response of 45 normals and 105 patients with backache in both fully flexed standing and sitting postures. All of the normals and 71 of the patients exhibited electrical silence in both postures. In standing, the FR response was shown by 15 patients and the remaining 19 patients failed to exhibit the response in either posture. The flexion range of motion was not measured so it is not possible to determine if there was a difference in the range of spinal flexion between the three groups.

Triano and Schultz (1987) found that all 7 controls and 23 of 41 patients were able to exhibit the FR response. The 18 patients unable to produce electrical silence had significantly decreased ranges of flexion and extension motion compared to the other two groups. It is not known whether the muscle activity is present to prevent an individual from flexing into a posture which may load painful passive tissues or if the muscle activity initiates a pain-spasm-pain cycle, also preventing full flexion from being reached. It is also possible that a misalignment of the vertebrae has occurred, limiting the range of motion. This would load some of the passive structures on one side of the vertebrae, but muscle force would also be required to provide joint stability.

### **Electromyography and Low Back Discomfort**

Electromyography has been utilized extensively in the evaluation of low back discomfort. As a non-invasive technique, it may be used to distinguish between healthy and

dysfunctional backs. The technique is based on power spectral shifts that the EMG signal undergoes with sustained muscle contraction.

Moritani et al. (1992) found that people with a history of low back pain had greater rates of mean power frequency decline than controls and that there was an asymmetry in the fatigue rates between an individual's left and right lower erector spinae. Unfortunately, they did not explore the relationship between the asymmetry and the site or side of the low back pain.

De Luca and colleagues, utilizing changes in the median frequency of the power spectrum, have been able to distinguish individuals with low back pain from those without, with a minimal accuracy of 84% (Roy et al., 1989, Roy et al., 1990, De Luca, 1993). Kondraske et al. (1987) utilized a similar protocol but were not nearly as successful. Possible reasons for the success of De Luca and colleagues are the utilization of a fixed, consistent posture, monitoring of six electrode sites and careful monitoring of the isometric contractions so that they are very consistent (De Luca, 1993).

These methodologies show that EMG can be used to classify individuals with and without low back pain. EMG also assists in localizing the problem area and augments the understanding of injury effects on muscle. However, these techniques are still one step removed from estimating the force distribution in spinal tissues of individuals with spine movement dysfunction due to low back pain.

### **Electromyography Normalization**

To compare EMG activation patterns between individuals and/or different muscles, or to use EMG as an input for a biomechanical model, it is necessary to normalize the EMG

signal. This is typically achieved by scaling the EMG activation pattern of interest to the magnitude of activation produced by a maximal voluntary contraction (MVC). These normalization contractions are problematic for individuals suffering from a low back disorder. Fear of re-injury, pain, decreased motivation and inexperience in performing these types of contractions are possible explanations for an individual's inability to perform MVCs. The quantification of the maximal EMG activation level for a specific muscle group utilizing contraction intensities other than MVC is an attractive alternative for the low back injury population.

Submaximal, continuous isometric contractions in well motivated subjects, have been found to produce maximal EMG activation. Moritani et al. (1986) found the RMS EMG amplitude of the biceps brachii to increase from 50% to 100%, for a one-minute sustained 50% MVC isometric contraction (subjects were practiced and received visual feedback on the force level). Petrofsky et al. (1982) present data on the handgrip and biceps brachii muscle that illustrates constant, 70% MVC isometric contractions producing RMS EMG amplitudes of 100% MVC amplitude by the end of the testing session (subjects were practiced). The mean ( $\pm$  SD) duration for the handgrip contractions was 48 ( $\pm$  11) seconds and 73( $\pm$ 13) seconds for the biceps contractions. The durations in these two studies match well with the endurance times of Rohmert (1960) summarized by Bigland-Ritchie and Woods (1984). Although De Luca and colleagues utilize sustained, high level contractions as a part of their protocol, to the author's knowledge, they have not published the effects of these contractions on the EMG amplitude detected from the erector spinae.

As discussed previously, individuals with low back pain have difficulty in performing isometric MVC's. If submaximal, sustained isometric contractions could produce maximal electrical activation in the lumbar musculature, it would be an attractive alternative method for obtaining maximal electrical activation.

### **Biomechanical Models of the Spine**

The lumbar spine has been the focus of biomechanical modeling for over three decades. Model outputs directly affect the assessment of lumbar spine function and the estimate of tissue loads and injury risk. Therefore, the model assumptions, inputs, and operational parameters must faithfully represent the *in vivo* characteristics of the body. The most significant challenge to modeling is the development of a method to overcome the indeterminacy produced by the anatomical redundancy of structures capable of generating or supporting moments of force (Norman, 1992).

### **Reduction Models**

Early modelers used a reductionist approach for their sagittal plane analysis by simply representing the spinal extensor musculature as a single equivalent muscle with a 5 cm moment arm. Morris et al., (1961) determined the compressive force on the lumbar spine by counteracting the external load moment with an erector spinae extensor force and an intraabdominal pressure stabilizing force. Chaffin (1969) utilized a digital computer and incorporated a seven link, rigid body model to analyze maximal static strength. Regardless of their potential hip and knee extensor strengths, subjects appeared to limit their compressive

forces to a constant magnitude (males  $\approx$  6000 N, females  $\approx$  3500 N) and load shear forces never exceeded 500 N. Freivalds et al., (1984) utilized a dynamic version of the same model using actual segment motion. Estimated L5/S1 compressive force-time histories reached 7000 N for maximal volitional lifts and revealed the effects of box size, load magnitude and lifting style. Anderson et al., (1985) produced a biomechanical model of the lumbosacral joint to analyze the effects of posture and lifting loads (0 to 500 N). The calculation of the restorative moment due to abdominal pressure, disc, ligaments, and muscle showed that the muscular moment predominated. The muscular moment was distributed between the multifidi and erector spinae based on a ratio of cross sectional areas. They concluded that typical lifting tasks can produce excessive disc compression because of the large muscle moment requirements.

These models all share the same problem in that the assessment of loads routinely handled in industry results in the output of compression forces that are larger than those required to produce micro-fractures of the cartilage endplates in cadaver spinal segments (Chaffin and Andersson, 1984, White and Panjabi, 1990). This anomaly indicates that the anatomy and/or tissue tolerance data requires reassessment. Two different modeling strategies have been developed for the partitioning of forces in response to the indeterminacy produced by increased anatomical fidelity.

### **Optimization Models**

Schultz et al., (1983) investigated four optimization strategies, for an L3 level model of the trunk that consisted of 22, 14 or 10 muscles, in order to predict the muscle forces required

to perform a variety of complex tasks. The optimization functions incorporated were: 1) approximately minimize the maximal muscle contraction intensity (by setting a muscle intensity of 10 kPa (1 N/cm<sup>2</sup>), solving for minimum compression using linear programming, and then increasing the muscle intensity in 10 kPa increments as required), 2) same as 1) but with 1000 kPa (100 N/cm<sup>2</sup>) as the maximum muscle intensity, 3) minimized sum of the square of the muscle contraction forces, and 4) minimized sum of the cube of the muscle contraction forces. The 10 muscle model and the first optimization strategy produced sufficiently satisfactory results based on the validation method of correlating the mean predicted muscle force to the mean EMG amplitude. Although, the selected objective function did not permit antagonistic muscle activation, the authors found it consistent with the subjects behavior, who had "relatively little unnecessary antagonistic activity".

Schultz et al., (1982) directly measured the intervertebral disc pressure and EMG amplitudes produced during static symmetric and asymmetric postures, in an effort to validate a dual cost linear program. The program would search for the internal forces necessary to produce the net reaction moment required for equilibrium and minimize the compressive load on the third lumbar vertebrae. The previously described 10 muscle model was used and the predicted disc compression was well correlated with the mean disc pressure ( $r=0.94$ ). Correlations between predicted muscle force and mean EMG activity ranged from 0.2 for the external obliques to greater than 0.9 for the erector spinae. The authors correctly noted that the cost function utilized would produce the non-physiological responses of no antagonistic muscle activity and that the synergists with the largest moment arms would be recruited first.

Gracovetsky (1986, 1988) selected an objective function of stress minimization (compression and shear) and equalization (at all joint levels) in order to partition the reaction moment. The biomechanical model incorporated improved anatomical detail of the lumbodorsal fascia (LDF) and a method by which the transverse abdominis acts on the LDF to produce an anti-flexion moment through the posterior ligaments. However, the extensor musculature moment arm was smaller than the moment arm of the ligaments, contrary to the reports of others (Hutton and Adams, 1982, McGill and Norman, 1988). This anatomical limitation dictates that in the objective function of minimization of compression, the ligaments must be recruited first. Therefore, the model outputs and resulting conclusions all reflect the posterior ligament strategy. Further research has found the moment contribution of the LDF to be minimal (Macintosh et al., 1987, McGill and Norman, 1988), illustrating the need for an accurate anatomical representation of the entire trunk.

Bean et al., (1988) applied a double linear programming method to the model of Schultz et al., (1982). The objective functions were to minimize the muscle intensity and then minimize the joint compression force. Double linear programming is advantageous because it permits an assessment of the effects of the constraints or solution "costs". However, the resulting joint compression and predicted muscle forces are still a product of the objective function and, therefore, suffer directly from any limitations in the cost function.

### **Limitations of Optimization Models**

The selection of an appropriate cost function and the validation of the resulting optimization model are the two major issues encountered by optimization modelers. It is

simply impossible to know which objective function(s) the body incorporates at any time. The production of "true" antagonistic activity is also an issue for simple cost functions (e.g. minimize compression), but this drawback does not necessarily apply to all optimization models.

Herzog and Binding (1992) have analytically demonstrated that cocontraction is predicted in a non-linear optimization (minimized sum of the cube of the muscle contraction forces) and that cocontraction may also enhance the muscular mechanical efficiency. A planar, three link system consisting of: 1) three, single joint and two, two joint agonist muscles and 2) three, single joint and two, two joint antagonist muscles, was used. The authors identified that the approach was only analytical and that the application of physiological data would certainly influence the output.

Involved with the selection of an objective function is the application of specific boundary conditions (e.g. magnitude of maximal muscle intensity). Lavender et al. (1992) recommended that the minimum levels of muscle activation for a given moment in a specific direction be incorporated as boundary conditions for optimization models.

EMG has also been used to indirectly validate optimization models (Schultz et al., 1982, Gracovetsky, 1988, Bean et al., 1988). Using EMG as a method to control these models, or even indirectly validate them, produces an interesting situation, for a model that utilizes this as an input measure must be inherently valid (Norman, 1992).



### **EMG Assisted Models**

These models use the EMG activation patterns as one of several inputs for partitioning the resultant moment between different muscle fascicles. One of the earlier, detailed descriptions of using EMG to predict tissue forces in dynamic activities comes from the work of Hof and Van Den Berg (1981). Linear envelope EMG from the gastrocnemius and soleus, were combined with the ankle joint angle as inputs to an electrical analogue of the Hill muscle model. The analogue model values for torque, work and integrated torque were compared to those values measured by a custom torque plate. The moment calculated by the model reflected the measured moment very faithfully. The relative error for 657 positive work, negative work and integrated torque data points was 6.2% ( $\pm 14\%$ ).

Electromechanical delay, eccentric contractions and cocontractions were the areas that posed the most significant problems for the model. Determining the correct gain for the EMG signals and EMG crosstalk were signal processing complications that also affected the model output. However, the success in predicting the moment output based on the biological signal from the two muscles producing the moments, indicated that EMG should also be capable of determining the relative contribution of each muscle to the moment. This is the approach that EMG assisted modelers have expanded upon.

Marras and Sommerich (1991a, 1991b) have incorporated the 10 muscle model of Schultz et al., (1983) as the basis of their EMG assisted model which they have applied to symmetric, and asymmetric isokinetic, constant torque contractions. Each subject's L5 torso depth and breadth is used to predict the cross sectional area and moment arms. For three of the muscles, the effects of length - strength (L-S factor) are incorporated by normalizing the EMG

to maximal EMG activation ( $EMG_{max}$ ) obtained at three different flexion angles. A data base of isometric EMG to isokinetic EMG ratios is used to modulate three of the muscles for force/velocity. The gain factor incorporates the muscle force per cross sectional area, which begins at  $10 \text{ N/cm}^2$  and increases by this step amount as much as required. The EMG signal for each muscle is simplified to be represented by four time points and straight line approximation. This simplifies the calculation of forces to a maximum of 40 time points. At each time point the tension level for each muscle is therefore determined by;

$$\text{Force} = \text{Gain} * (\text{EMG}/\text{EMG}_{max}) * \text{V ratio} * \text{L-S factor} * \text{area.}$$

Output from the model showed compression increased with velocity ( $100 \text{ N}$  per  $10^\circ/\text{s}$  increase) and external load, and decreased slightly with asymmetry. Posterior shear increased with external load, while the right and left shear was found to be highly variable. Indirect model validation was attempted by a comparison of the measured torque to predicted torque. An  $r^2 \geq 0.7$  was found for more than 85% of the torque pairs. However, the average gain per subject ranged between 80 and  $250+ \text{ N/cm}^2$  and nine of eleven subjects had gains greater than  $100 \text{ N/cm}^2$ . These muscle force producing potentials are much larger than the 35 to  $100 \text{ N/cm}^2$  cited from the physiological literature by McGill and Norman (1987).

This issue was addressed in a fully dynamic version of the model which was tested under controlled isometric, isokinetic and isoinertial exertions for 20 subjects that performed sagittally symmetric and asymmetric flexion-extension tasks (Granata and Marras, 1993). Using a series of exertions and averaging the values from a range of calibration test conditions

produced an average gain of  $42 (\pm 11) \text{ N}\cdot\text{cm}^{-2}$ . Comparison of the predicted and measured lifting moments for over 2100 trials produced an  $R^2$  of greater than 0.8.

McGill and Norman (1986) developed an EMG assisted model that partitioned the L4/L5 dynamic moment into disc, ligamentous (7) and muscular (48) components using a three-dimensional skeleton, based on the anthropometrics of a 50th percentile male and the linear envelope from six electrode sites. A link segment model was used to determine the L4/L5 reaction moments and the passive tissues were assigned force and moments based on their strain. The remaining restorative moment was portioned among muscles based on the EMG activation patterns from six electrode sites. The force at any time (t) is scaled by the ratio of  $\text{EMG}(t)/\text{EMG}_{\text{max}}$ . The maximum force producing potential,  $F_{\text{max}}$ , was varied from 35 - 55  $\text{N}/\text{cm}^2$ . Muscle forces were scaled by force/velocity (V Fac) and force/length (L Fac) modulating factors. Any force due to passive elasticity was then added. The Gain factor was used to increase the relative contribution of *all* muscles to force the instantaneous predicted external L4/L5 moment to match the measured moment. This approach also ensured that cocontraction is considered. Thus, the muscle force at any time was determined by the following equation;

$$F_m(t) = \text{Gain} * [(\text{EMG}(t)/\text{EMG}_{\text{max}}) * (F_{\text{max}}) * (\text{V Fac}) * (\text{L Fac}) + F_{\text{pec}}]$$

Improved anatomical modeling decreased shear and compression estimates by 42.5% and 16.2% respectively, compared to values calculated from a model with a simple 5 cm erector tissue moment arm length. The ligaments were revealed to play a minor role in the squat lifts studied.

Potvin et al., (1991) utilized a revised version of this model to study lifts with varying degrees of trunk flexion. A total of 50 functional muscle fascicles were developed by dividing eleven muscles bilaterally and seven ligaments were represented by eleven force vectors. Increased trunk flexion significantly increased anterior shear forces of the superior on the inferior vertebrae, while compression was insensitive to this muscle-ligament interplay. This apparent anomaly occurred because the combined musculature moment arm is greater than that of the ligaments, so that the increased ligament recruitment with increased trunk flexion, offset the decreased compression due to decreased muscle activation.

This study highlighted the sensitivity of this model in determining ligament recruitment as some of the data from six of the original fifteen subjects were unable to be utilized. During some of the trials for three subjects, the ligament contributions were predicted to be greater than the total extensor moment, even though the muscles were active. This over prediction is caused by the steep slope of the ligament stress - strain curve at the end range of motion. Ligament recruitment was modeled to occur at  $6^\circ$  less than the subject's fully flexed position. For another three subjects, some of their trials had almost zero ligament recruitment, while their EMG activation levels were less than the group mean. These examples highlight the fact that, just as the regression equations used by Marras and Sommerich work better for some individuals, some people do not "fit" these models.

The previous versions of the model incorporated three dimensional anatomy but studied sagittal plane movement. McGill (1992) modified the model even further, incorporating a three-dimensional linked segment model to produce the reaction forces and moments about three orthogonal axes corresponding to the L4/L5 joint, while examining lateral bending. The

reaction moments were then partitioned into the substantive moment components using a three-dimensional representation of trunk tissues. Inputs to the model were three-dimensional joint coordinates, dynamic hand loads and 12 channels of trunk EMG. The model was found to be very sensitive to how subjects recruit their musculature to satisfy moment constraints. A reassessment of the role of the abdominal musculature in generating a flexor moment was required so that the flexion moment could be increased in order to balance the moments.

The model outputs showed very little ligament contribution. Lateral flexion towards standing decreased compression, anterior shear and lateral shear. The results showed that a 3 to 4 cm moment arm would be appropriate for a single equivalent lateral flexion muscle model to be used in industry. A large compressive penalty was observed, due to 8% coactivation in lateral bending, and was interpreted as a strategy to increase mechanical stiffness by increasing bending stiffness.

### **EMG Assisted Optimization**

Cholewicki et al., (1995) have compared an EMG assisted model (EMG), optimization approach (approximate minimization of muscle stress and then spine compression) (OPT) and a method that combines both EMG and optimization termed EMG Assisted Optimization (EMGAO). The last method used a minimization of the EMG model variable gain while satisfying the moment requirements about the L4/L5 joint as an objective function.

Mathematically this can be expressed as:

$$\sum M_i(1-g_i)^2 = \min$$

$$M_i = (M_{xi}^2 + M_{yi}^2 + M_{zi}^2)^{1/2}$$

Subjects performed isometric ramp contractions up to maximal effort in the directions of flexion, extension, lateral flexion left and lateral flexion right. The three techniques were compared using RMS difference between muscle force estimates from each technique with the EMG assisted method chosen as the reference. Average absolute errors between measured external moment and predicted moments were not found for OPT and EMGAO. The EMG method had a mean average error ranging from 5.8% in extension to 17.3% in right lateral bending. The muscle force predictions between EMG and EMGAO had RMS differences of only 17%, 31% and 42% for extension, flexion and lateral bending. EMG versus OPT produced RMS errors of 123%, 123% and 218%, respectively. Although the OPT predicted lower joint compression by 32%, 43% and 23%, it was due to the inability of the optimization method to identify "pure" antagonistic muscle activation. Activation of an "antagonist" could be produced if a moment about another axis was required.

The EMGAO approach combined the major advantages of the EMG method, by producing similar force predictions with physiologically based recruitment patterns, with that of the OPT method by fulfilling the moment constraints. The disadvantage to the optimization model was that it did not allow for cocontraction and was not sensitive to individual differences in muscle synergy.

## Neural Networks

Nussbaum et al. (1995) utilized an artificial neural network model to predict spinal muscle activity. Subjects resisted static moment loads of 10 to 50 N·m applied in 30° increments from flexion (0°), through lateral bending (90°), to extension (180°) while in an upright posture. EMG was measured bilaterally from the erector spinae, rectus abdominis, external oblique and internal oblique and were normalized using maximal and resting values. A multilayer, fully connected, feed-forward artificial neural network was trained using subsets of the moment-EMG data set. Using the normalized EMG as the criterion measure, the output of the network model was compared to two optimization-based muscle force prediction models. The optimization models were a double linear programming method that minimized maximal muscle intensity and then minimized joint compression and a nonlinear program that minimized the sum of the cubes of muscle force intensities.

The neural network model was found to predict muscle activities that were better correlated with the experimental data than either optimization method. It was also capable of predicting cocontraction. Although not performed in this study, this model would also allow spinal muscle force estimates to be made for novel loading situations. However, the utility of the model in its current form may be limited because it estimates muscle forces for only static postures. It was also designed to estimate muscle forces for a particular posture and not necessarily a particular individual.

### Summary

The mechanisms of low back injury are not well understood. This is due in part, to the number of possible injury sites, but it is also complicated by the volume of diagnoses utilized in the assessment of low back injury. Electromyography, commercial dynamometers and EMG assisted models of the spine have been utilized for the measurement and analysis of spine movement dysfunction, but the techniques have never been integrated.

1. The Isostation B-200 can be used to quantify an individual's level of back dysfunction. However, because this approach focuses on peak parameters and does not quantify the entire movement time history, limited information is learned regarding where in the movement cycle the abnormal parameters are produced. Also, some comparisons would be more appropriate if relative rather than absolute measures were used. The comparison of the relative moment magnitudes over the whole time history would enhance the assessment of back dysfunction.
2. The EMG input signals for the spine model require normalization to maximal electrical activation. This level of activity is typically produced by performing isometric, maximal voluntary contractions. These types of contractions are problematic for individuals with spine movement dysfunction. The performance of sustained, submaximal isometric contractions should elicit maximal muscle electrical activation.



3. The EMG assisted model of McGill (1992) produces force/time histories for 50 muscle fascicles and 12 ligament vectors, but it has never been applied to a population with spine movement dysfunction. It is not known if this level of knowledge would provide a more specific identification of the dysfunctional structures.
4. The EMG assisted model of McGill (1992) has never been applied to a spine movement dysfunction population, so the effects of recovery on the lumbar spine tissue force/time histories during dynamic and isometric contractions is unknown.
5. The EMG, combined with estimates of the muscle and force time histories will provide a range of tissue responses for a particular diagnosis. This information could eventually be used to document similarities and differences among the numerous diagnoses made of individuals with spine movement dysfunction.

## **Chapter III**

### **EMG Amplitude Changes in The Lumbar Spine Extensor And Flexor Musculature During Maximal And Submaximal Constant Force Contractions**

#### **Introduction**

Electromyography (EMG) has become a common tool in the analysis of human movement. To facilitate comparisons between different muscles and individuals the EMG amplitudes are typically transformed or normalized to levels of a relative contraction force, typically a maximum voluntary contraction (MVC) (Basmajian and De Luca (1985). This allows differences in amplitudes to be attributed to the phenomenon of interest rather than technical factors such as the precise location of electrodes upon re-application or differences in skin impedance. This method of normalization has been successfully used in computer models designed to estimate muscle forces in the limb (e.g. Hof and Van Den Berg, 1981; Olney and Winter, 1985). However, for the muscles of the back, EMG normalization using MVC's is more problematic. Due to the challenging nature of performing MVCs, skilled performers have been found to require several attempts using different postures in order to produce a maximal amplitude (McGill, 1991). Also, due to fear of re-injury, pain, decreased motivation and/or inexperience in performing MVCs, individuals with low back pain have difficulty producing "true" MVCs. This has prevented EMG assisted models of the lumbar spine from being utilized with individuals symptomatic for low back disorders because the performance of MVCs is required for normalization purposes (Marras and Sommerich, 1991a, 1991b, McGill and Norman, 1986; McGill, 1992, Granata and Marras, 1993). The application of these models

to this population is particularly important if injury mechanisms and rehabilitation processes are to be comprehended.

An attractive alternative would be the quantification of maximal EMG activation levels for a specific muscle group utilizing a contraction intensity other than MVC. It may be possible to obtain maximal EMG amplitude via prolonged isometric contractions. For the biceps brachii, Petrofsky et al. (1982), and Moritani et al. (1986), observed that at the termination of sustained, submaximal isometric contractions of 70% and 50% MVC, respectively, the EMG amplitudes were equivalent to those produced during MVC efforts. Another approach would be the construction of the upper portion of the EMG-moment relationship by performing repeated submaximal contractions of varying intensity. Linear regression could then be used to predict the maximal EMG amplitude.

The purpose of this study was to determine if EMG amplitudes equivalent to those observed during MVC efforts could be predicted/elicited for the extensor and flexor musculature of the spine via either: (i) sustained, 70% MVC isometric contractions, or (ii) the use of submaximal, isometric contractions, of varying intensity and linear regression. A secondary purpose was the quantification of the changes in magnitude in the mean power frequencies for these muscle groups during the sustained isometric efforts.

## **Methods**

### *Subjects*

Eight males (mean height = 1.78 m, SD  $\pm$  0.08, mean mass = 82.0 kg, SD  $\pm$  9.5, mean age = 32.4 yr, SD  $\pm$  13.0) from a university population volunteered for the study. Each

participant was asymptomatic for low back pain within the last year. Prior to participation, each individual reviewed and signed a consent form approved by the Office of Human Research.

### *Instrumentation*

EMG was recorded bilaterally from the rectus abdominis (3 cm lateral to the umbilicus, aligned straight upward), external oblique (approximately 15 cm lateral to the umbilicus, oriented diagonally down and inward), internal oblique (below the external oblique electrodes and just superior to the inguinal ligament, aligned diagonally up and outward), upper erector spinae (5 cm lateral to T9 spinous process, oriented up and slightly outward) and lower erector spinae (3 cm lateral to L3 spinous process, directed up and outward), (McGill, 1992, Sutarno, 1993), using disposable Ag-AgCl electrodes (Medi-Trace, ECE 1801) with a center-to-center distance of 2.5 cm. Prior to electrode application, the skin at each site was prepared by shaving the skin and abrading the area with tissues soaked in alcohol. The raw myoelectric signals were input to a differential amplifier (CMRR of 80 dB at 60 Hz), prefiltered (bandwidth of 20 to 500 Hz) and then amplified.

### *Tasks*

Subjects were required to perform the following tasks for extension and flexion respectively: two, ten second MVCs, a series (3 or 4) of submaximal, ten second, isometric efforts and a sustained 70% MVC held until volitional termination.

The connecting fastener of a shoulder harness was aligned with the torso (head, arms, and trunk) center of mass, measured from the greater trochanter (Winter, 1990). The subjects then lay prone (or supine) over a two-tiered bench, with the greater trochanter aligned with the edge of the higher tier and their upper body weight supported on the lower tier. To measure the moment-time history for the MVC trials a linear variable differential transformer (LVDT) was connected to the harness and secured to the floor. The length of the connecting cable was adjusted until the subject's torso was horizontal, but clear of the lower tier during isometric exertions. For the MVC trials, subjects were instructed to slowly raise themselves, to build up to their maximum effort over two seconds and then to hold that effort until they were instructed to relax. The MVC was followed by a minimum of one minute rest, during which time the maximum moment was recorded and signal quality checked. A second MVC was then performed.

These MVC efforts were followed by a series of 10 s, constant moment isometric extension/flexion efforts, ranging from the weight of the upper torso, to 85% MVC. Typically, participants performed four trials for flexion (range = 2 to 4) and extension (range = 3 to 5). For one individual, their torso mass represented 80% of their maximum flexor strength. This person was tested using only their torso mass and one other flexion load. The load (including torso mass) required to produce a specific percentage of MVC was calculated and attached to the harness cable, replacing the LVDT. Subjects were required to raise their torso to a horizontal position, resulting in the loads being raised just clear of the floor. Each 10 second collection period was preceded by a practice effort, so that subjects could become accustomed to the loading.

Upon completion of the highest submaximal level, the 70% loads were attached to the cable. Subjects were instructed to raise their torso to the same position as the previous submaximal efforts and to hold that position, with the load just clear of the floor, for as long as possible. Verbal encouragement was provided throughout the test.

#### *Data Reduction*

The LVDT and EMG signals were A/D converted (AT-MIO-16, 12 bit ADC, National Instrument, Inc.) at 1024 Hz and stored on magnetic-optical disk. For EMG normalization the raw EMG signals were full wave rectified and low pass filtered (2nd order, single-pass, Butterworth digital filter) at a cutoff frequency of 2.5 Hz to produce a linear-envelope (LE). A 2.5 Hz cutoff frequency was selected because it reaches peak response to an impulse in 63 ms, which is in the middle of the 30 - 90 ms twitch response to peak tension found by Buchthal and Schmalbruch (1970). Olney and Winter (1985) found cutoff frequencies to range from 1.8 to 2.8 Hz for the rectus femoris. Potvin (1992) found 2.7 Hz to be the best frequency for the lower and upper erector spinae musculature.

*Amplitude Analysis:* The maximum EMG amplitude observed for each muscle was determined from all MVC trials by displaying the LE EMG for a muscle against the force curve and selecting the largest amplitude in the region of approximately constant amplitude excluding the region during which force was being developed or reduced. The largest amplitude (i.e single point) for each muscle was termed MVC. For the submaximal constant moment and sustained trials, each channel was normalized to the 100% MVC amplitude. The peak amplitude and

time of occurrence were then recorded. Care was taken to avoid periods during the start of the contraction when adjustment to the load was occurring. For the sustained contractions, the peak amplitude and time of occurrence were recorded for both the first 10 second (INITIAL) portion and the remaining (TERMINAL) portion of the contraction.

*Frequency Analysis:* To determine the mean power frequency (MnPF), a 1024 point Fast Fourier Transform, using a Hanning window, was performed for each one second period of raw EMG immediately preceding and inclusive of the time point of maximal EMG occurrence. This point was selected on the basis that it would incorporate all of the EMG interference pattern that was associated with the LE envelope peak. Thus, the FFT would encompass the muscle force occurring during that contraction of that specific muscle. The MnPF of the extensor and flexor muscles was determined for each of the submaximal loads and the sustained INITIAL and TERMINAL time periods. Each MnPF was normalized to the MnPF produced during the first second of stable force production. Again, care was taken to avoid the periods in which force was being developed or reduced.

## Results

*Maximal Voluntary Contractions:* The mean peak extension and flexion moments were  $328.7 \pm 50.2$  N.m and  $307.2 \pm 43.3$  N.m, respectively (Table 1)

**Table 1** The mean moment (SD) and EMG amplitudes for all flexor (n=6) and extensor (n=4) muscles for the maximal and sustained contraction tests. The \* indicates a significant increase in EMG amplitude,  $p < .01$ .

	Maximal Contraction	70% Sustained Contraction		
	Moment (N.m)	Peak EMG Amplitude (% MVC)		Duration (s)
		INITIAL	TERMINAL	
Extension	328.7 (50.2)	67.0 (14.8)	87.8 (13.1) *	42.6 (13.1)
Flexion	307.2 (43.4)	81.6 (27.5)	115.6 (31.3) *	30.4 (10.2)

### Amplitude Analysis

*Sustained:* The sustained 70% MVC flexion and extension contractions produced a significant increase,  $p < .01$ , in the group mean EMG amplitudes for all of the extensor (n = 4) and flexor (n = 6) muscles (Table 1). The load had been selected for each person as 70% of their isometric maximum moment. The initial mean extensor amplitude was 67% MVC and increased to 88% by the end of the contraction, a 30% increase with respect to the initial contraction intensity (Table 1). The initial mean flexor EMG amplitude was 80% MVC and increased to 115% MVC by the end of the sustained contraction. This represents a 40% increase with respect to the initial contraction intensity of 82% MVC (Table 1).



*Loads of Varying Intensity:* Figure 2 illustrates representative data for the trials of the loads of varying intensity. The mean peak activation levels were calculated for all of the muscles, for all of the subjects, for each of the loads. Linear regression of the peak extensor EMG amplitudes and the extensor load moment revealed that on average, the extension musculature response was 21.5% MVC below the applied load moment ( $\text{EMG Amplitude (\% MVC)} = 1.15 \times \text{Extensor Load Moment} - 21.57$ ) (see Figure 3a). Linear regression of the peak flexor EMG

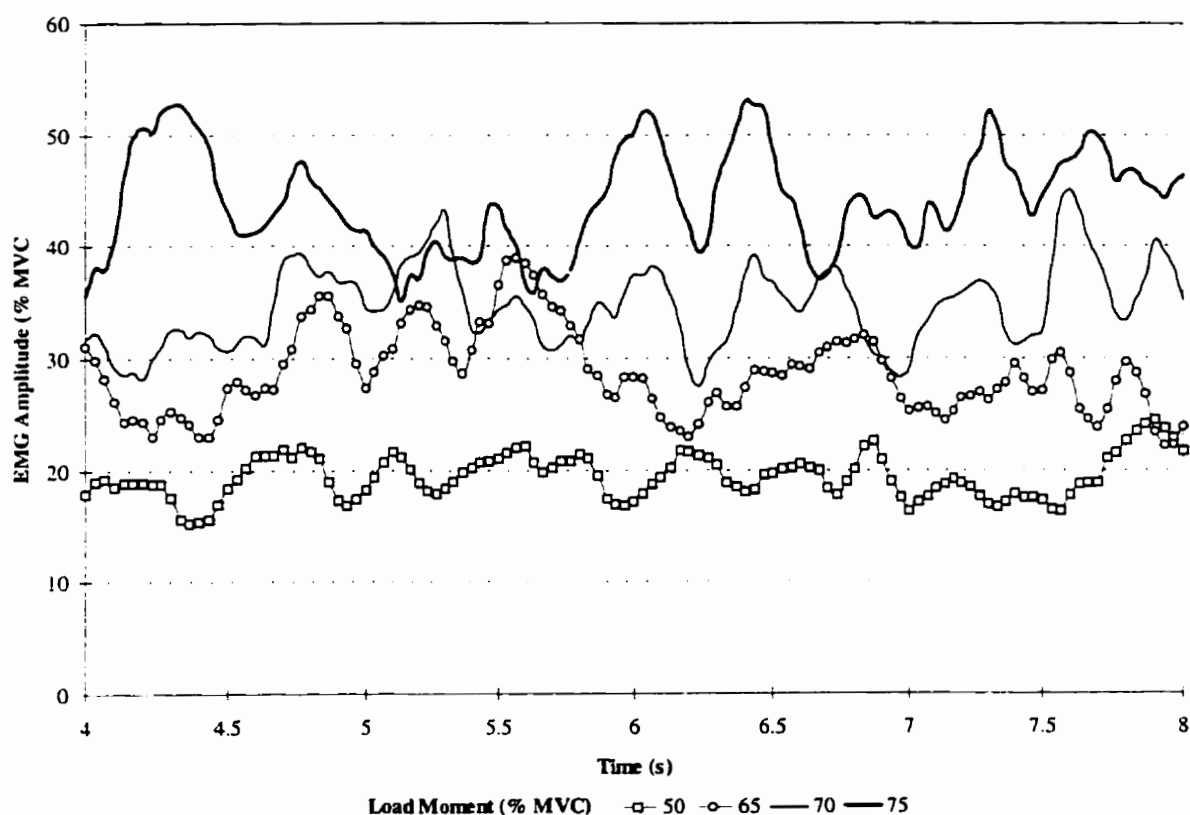


Figure 2: An example of the linear envelope EMG produced for the RUES during the 10 s submaximal isometric contractions.

amplitudes and the flexor load moments found the flexion musculature was 22.5% MVC above the applied load moment ( $\text{EMG Amplitude (\% MVC)} = 0.89 \times \text{Flexor Load Moment} + 22.54$ ) (see Figure 3b).

To further investigate this response, regression analysis of the flexor and extensor EMG amplitude versus the flexor and extensor load moment, respectively, was performed using each of the submaximal loads, of each muscle, of each subject. Surprisingly, of the 32 possible regressions for the extensor muscles (8 subjects X 4 muscles), only 13 produced significant correlations and 4 of these came from subject #6 (Table 2). The lower erector spinae accounted for 9 of the 13 significant relationships. Of the 48 possible regressions for the flexor muscles (8 subjects X 6 muscles), only 4 correlations were significant (Table 2). The rectus abdominis produced 3 of the 4 significant flexor muscle equations. The significant regressions are summarized in Table 2.

Using only the significant regression equations, the average predicted maximum extensor EMG activity, normalized to MVC, was 99.2%. However the range between the upper and lower boundaries of the 95% confidence interval was almost 50% MVC (Table 2). The average predicted maximum for the flexors was 88% MVC, with the 95% confidence interval spanning 78 to 98 % MVC (Table 2).

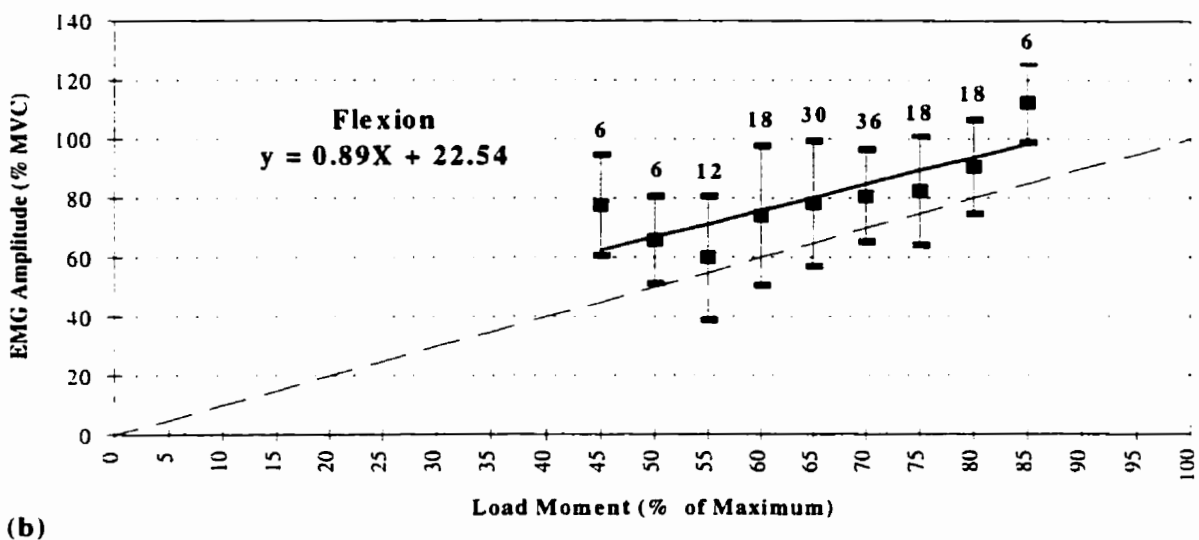
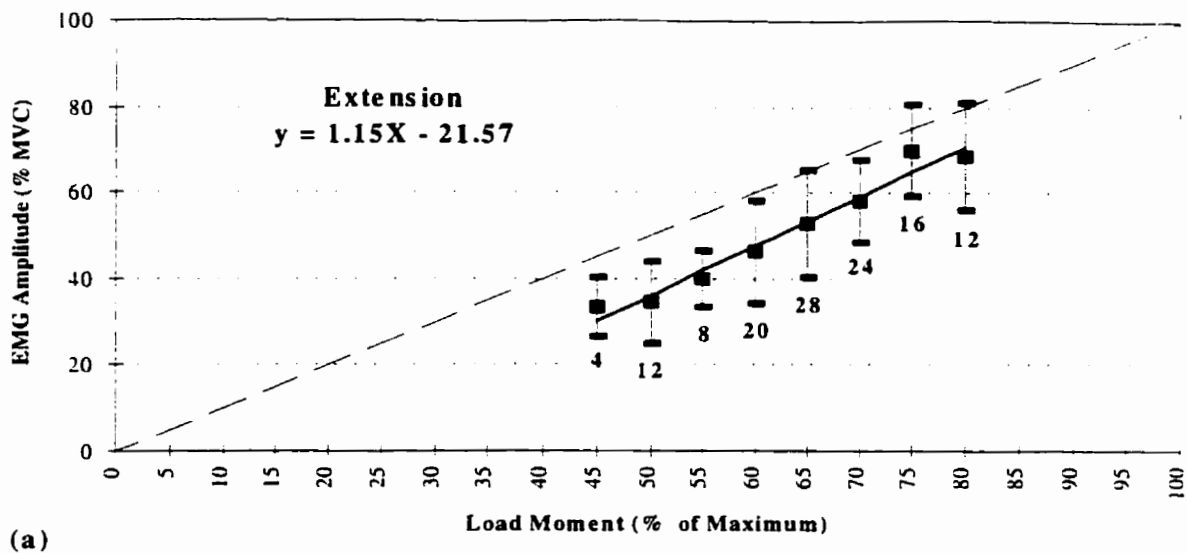


Figure 3: Mean ( $\pm 1$  SD) peak activation levels for all muscles, for all subjects during the sustained, 70% MVC extension and flexion efforts. Figure 3 (a) illustrates that the average extension activation was 20% less than anticipated. Figure 3 (b) shows that the average flexion activation was 22% greater than anticipated. In each figure, the dashed line represents the identity line. The filled squares represent the mean EMG response of all muscles and subjects at that load. The error bars represent 1 SD. The solid line is the line of best fit produced by linear regression. The number below each data point indicates the number of muscles used to calculate the mean for that data point.

**Table 2** Significant regression relationships from the 10 s, submaximal isometric contractions.

Subject	Muscle Group	Predicted Maximum EMG	95% Confidence Interval		Probability
			Lower	Upper	
Extension					
1	RLES	103.4	67.9	138.9	.021
1	LLES	94.4	74.0	119.0	.014
2	RLES	110.2	100.6	119.8	.001
2	LUES	96.2	40.7	151.8	.047
2	LLES	114.4	87.2	141.5	.012
3	LLES	61.2	31.4	91.0	.045
5	LUES	104.3	92.5	116.1	.003
6	RUES	96.1	93.0	99.1	.001
6	RLES	98.6	69.6	127.7	.020
6	LUES	103.0	60.8	145.2	.030
6	LLES	125.2	105.1	145.2	.005
7	LLES	100.9	82.6	119.1	.007
8	RLES	81.5	76.2	86.9	.015
	Average (SD)	99.2 (15.5)	75.5 (21.9)	123.2 (21.3)	
Flexion					
1	LRA	95.8	81.1	110.5	.040
4	RRA	71.9	67.1	76.7	.003
7	RIO	115.8	102.2	129.4	.018
8	RRA	69.8	62.6	77.1	.028
	Average (SD)	88.3 (21.8)	78.3 (17.8)	98.4 (26.0)	

*Comparison of the two techniques.*

The MVC amplitudes for the UES, LES and RA were determined for two subjects to illustrate the use of the techniques. Subject 2 was selected because he had 3 significant regressions for the extensors and 0 for the flexors. Subject 4 was selected because he had 0 significant regressions for the extensors and 1 for the flexors. As shown in Table 3, neither method results in a consistent and satisfactory value for the maximum EMG amplitude.

**Table 3** The EMG amplitudes (% MVC) as determined by the sustained contraction and regression analysis prediction techniques. The \* indicates the muscles that had significant regressions for each subject.

Muscle	Subject 2		Subject 4	
	Sustained	Regression	Sustained	Regression
RUES	88	98	95	102
LUES	122	96*	87	104
RLES	78	110*	106	91
LLES	100	114*	92	120
RRA	185	116	107	71*
LRA	112	66	129	63

Frequency Analysis

*10 s and Sustained Contractions:* For each of the muscles there was no significant difference between the average MnPF for the 70% MVC 10 s contractions and the INITIAL period of the sustained contractions. The sustained isometric contractions produced significant decreases in

**Table 4** The average mean power frequency (%) (Mean ( $\pm$  SD)) obtained at the time of peak EMG activity, normalized to the first value for each muscle, for each contraction during the 70% MVC, 10 s, and sustained isometric exertions. L = left, R= right. The † and ¶ indicate significant differences between the INITIAL and TERMINAL normalized MnPFs for  $p < .05$ , and  $p < .01$ , respectively. The \* and § indicate significant difference between the 10 s and TERMINAL normalized MnPFs for  $p < .05$  and  $p < .01$ , respectively.

70% MVC Contraction Duration:		10 s	Sustained		Decrease (%)
<u>Muscle</u>	<u>Side</u>		<u>INITIAL</u>	<u>TERMINAL</u>	
External Oblique	L	101.2 (21.4)	105.1 (18.3)	86.6 (21.5)	18.5
	R	102.2 (8.4)	102.5 (9.2)	88.5 (13.3) <sup>†¶</sup>	14.0
Internal Oblique	L	99.5 (15.2)	94.6 (13.7)	78.8 (15.1) <sup>§¶</sup>	15.8
	R	101.2 (2.9)	101.0 (7.3)	82.3 (5.0) <sup>§¶</sup>	18.7
Rectus Abdominis	L	90.0 (15.8)	97.4 (16.6)	76.4 (13.8) <sup>¶</sup>	21.0
	R	97.5 (5.1)	99.1 (9.4)	81.5 (7.1) <sup>§¶</sup>	17.6
Average					17.6
Upper Erector Spinae	L	108.8 (8.45)	108.8 (10.3)	92.5 (8.9) <sup>*†</sup>	16.3
	R	109.4 (3.8)	107.4 (7.6)	92.7 (4.6) <sup>*†</sup>	14.7
Lower Erector Spinae	L	114.2 (12.7)	113.3 (16.7)	87.4 (15.8) <sup>§¶</sup>	25.9
	R	113.03 (14.9)	110.7 (17.4)	82.5 (11.7) <sup>§¶</sup>	28.2
Average					21.2

each of the muscles except the left external oblique (Table 4). On average, the extensor and flexor musculature MnPF decreased 21% and 17%, respectively.

## Discussion

As expected, the extensor musculature produced a larger MVC moment than the flexor musculature. The flexion and extension moments are 2.0 and 1.3 times larger, respectively, than those produced in standing postures in our lab (Frazer and Norman, 1993). Troup and Chapman (1969) measured flexor and extensor moments that were 1.2 and 1.5 times larger than those produced in this study. Part of the differences are due to the restraining devices and measurement techniques. For example, Troup and Chapman (1969) had their subjects hold their arms horizontal and in an extended position and measured the flexion and extension forces at the hands. The differences also reflect the large variability that exists in human strength.

The sustained, submaximal isometric contractions did produce an increase in the EMG amplitude of the flexor and extensor musculature. For the extensors, the final mean amplitude increased by about 20% MVC, from 67 % MVC to 88% MVC. For the flexors, the increase was even more dramatic as the mean amplitude increased 35% MVC, from 80% MVC to 116% MVC. Even if the technique had produced increases to the 100% MVC amplitude, there would be difficulty in applying the technique to individuals unable to produce true MVCs. For these individuals, any measure of the “maximum” moment producing ability would likely be an underestimate. This would result in the 70% load calculation also being an underestimate. The challenge to the musculature would be decreased, making it unlikely that the final amplitude would match the 100% MVC amplitude.

The repeated submaximal loading technique was also successful in producing a relationship between the EMG amplitude and the load moment for both the flexors and extensors. The resulting regression equations allow the prediction of the EMG amplitude based upon the load moment. Unfortunately, the peak extensor EMG amplitudes were 20% less than anticipated while the flexor EMG amplitudes were 20% greater than expected.

On a case by case basis, the regression results became even more varied. For the extensors, the average maximal predicted EMG was 100% MVC, but the upper confidence interval was 25% MVC greater than the predicted value. If this regression method were to be used to predict the maximal EMG amplitude, it would definitely be possible to incorporate an erroneously low maximum EMG amplitude. If the predicted value was 100 % MVC, but the correct value was actually 125 %MVC, then the result would be an underestimate, by 20% of the relative magnitude for that particular EMG signal.

One of the difficulties in applying the repeated submaximal loading technique in the prone and supine positions is that the upper body weight represents a large percentage of a person's maximum moment. This minimizes both the number and range of data points available to use. For example, the torso moment for one of the subjects represented 80% of their flexion strength. They were able to comfortably maintain only one greater load, 85% MVC, limiting their regression analysis to only two data points.

Figure 3 also provides further insight into the difficulty of this technique. By adjusting the subjects position and altering the load attached to their torso, specific torso load moments could be produced. However, despite being in a very similar posture as the MVC trial, there was apparently a difference in how the subjects supported the induced load moment. The



depressed peak extensor amplitude during the submaximal ten second and sustained exertions indicates that musculature not monitored (e.g. multifidus) was contributing to the production of the required support moment. Also, subtle shifts in posture may have had significant effects on the EMG activation levels. Elevation of the peak flexor amplitude during the submaximal ten second and sustained exertions was very surprising. With the LVDT no longer affixed to the floor, subjects may have adopted a slightly more flexed posture than in the MVC condition. The resulting shorter muscle length would require increased EMG activation levels for each unit of muscular force produced. Inbar et al. (1987), found that Median Power Frequency increased as muscle length decreased. Analysis of the MnPF for the flexion trials showed no significant increase in any of the monitored musculature, although the mean value was typically greater than the 100% value obtained during the MVC trials.

The significant decreases in the mean power frequencies for the extensor muscles is similar in magnitude to those found by Roy et al. (1989) and Mayer et al. (1989). Roy et al. (1989) used an upright standing posture with an 80% MVC load sustained for up to 1 minute. Mayer et al. (1989) utilized the individuals upper torso mass as the load in conjunction with a horizontal posture similar to this study. However, the subjects were required to perform 10, 15 second trials, each trial separated by 10 s of rest. To the author's knowledge, this is the first study to investigate the flexor musculature changes in MnPF.

This study found that submaximal isometric contractions, either sustained or of varying intensity, were capable of producing increases in both flexor and extensor EMG amplitude. However, the increases were either not large enough and /or consistent enough to be used as a means for predicting maximal EMG amplitudes.

### **Conclusions**

Maximal EMG amplitude of the flexor and extensor musculature of the spine may not be reliably determined:

- 1) using sustained, submaximal exertions, or,
- 2) using the maximal amplitude observed from submaximal contractions of varying intensity.

## Chapter IV

### A Technique for the Calculation of EMG to Muscle Force Scaling Factors for an EMG Assisted Lumbar Spine Model.

#### Nomenclature

a	joint axis of the dominant moment
e	EMG electrode site
m	muscle fascicle
r	moment arm
$\delta$	velocity factor
$\Omega$	length factor
AMF	average muscle force (N)
AEMG	average EMG (a/d unit)
EMG	linear envelope EMG amplitude (a/d unit)
EMG <sub>max</sub>	maximum linear envelope EMG amplitude (a/d unit)
EMG <sub>Force</sub>	EMG to force scaling factor (N/a/d unit/cm <sup>2</sup> )
EMG <sub>peak</sub>	within trial maximum linear envelope EMG amplitude (a/d unit)
F	muscle force (N)
F <sub>pec</sub>	passive elastic force (N)
G	common gain factor
HI	highest submaximal isometric trial
LO	lowest submaximal isometric trial
M <sub>e</sub>	external moment at L4/L5
M	muscle moment (N·m)
M <sub>r</sub>	reaction moment at L4/L5 (N·m)
M <sub>l</sub>	moment of ligament l (N·m)
M <sub>d</sub>	moment of L4/L5 intervertebral disc (N·m)
P <sub>0</sub>	muscle force per cross sectional area (N/cm <sup>2</sup> )
XS <sub>area</sub>	physiological cross-sectional area (cm <sup>2</sup> )

#### Introduction

Lumbar spine tissue force time histories have been estimated via electromyography (EMG) assisted models (McGill, 1992; Granata and Marras, 1993). In order to calculate tissue forces, these models require scaling factors for the EMG. The scaling factors are obtained by

the performance of maximal voluntary contraction (MVCs) efforts in trunk flexion and extension.

However, eliciting maximal contractions from an individual is not a trivial task and there is no single “best” method for all subjects (McGill, 1991). Injury during the performance of trunk extension MVCs has also been reported (Zeh et al., 1986). There are also populations (e.g. individuals with low back pain, workers inexperienced with maximal contractions) who cannot produce “true” MVCs. Yet it is exactly these populations that would benefit greatly if these sophisticated models were applied to their specific situations.

This chapter describes a technique for the calculation of scaling factors that does not require MVCs. The outputs of an EMG assisted lumbar spine model were compared, using both this new method and the standard MVC procedure, in the application of calibrated EMG to a healthy and an injured population.

## **Methods**

### *Model Overview:*

The structural biomechanical model used to estimate tissue loads consisted of two parts. The first is a dynamic, three dimensional, fifteen link segment representation of the body which utilized the externally applied dynamic forces and individual anthropometrics as inputs. The reaction forces and moments were calculated about three orthopaedic axes corresponding to the L4/L5 joint using inverse dynamics and working through the hands, arms, head and trunk linkages [see McGill and Norman (1986) for a detailed two-dimensional description]. The second part, an anatomically detailed model of a three-dimensional pelvis, ribcage and intervening lumbar vertebrae, was then used to partition the three reaction forces into their

tissue components. This model incorporates fifty muscle fascicles, thirty-eight of which are capable of producing a restorative moment at the L4/L5 joint, thirteen ligamentous elements spanning the joint, a non-linear elastic intervertebral disc and an-equivalent torsional spring that represented the gut, skin viscera etc. Moment partitioning is accomplished by using EMG as an indicator for the neural activation level for each muscle. This neural input combined with modulators for velocity, length and the passive elastic component produces a muscle moment, which can then be adjusted to ensure that a sufficient restorative moment is produced [see McGill (1992) for a detailed description].

The model was "tuned" for each subject by having the subject "hang from their ligaments" in a fully flexed position. Subtracting the passive elastic muscular component from the external moment allowed the ligamentous contribution to be calibrated for the angular displacements of the torso with respect to the pelvis.  $EMG_{max}$  scaling factors, obtained from a standard set of static flexion and extension contractions and combined with physiologic muscle fascicle cross sectional area, allow each muscle fascicle's force ( $F_m(t)$ ) to be calculated (Equation 1). A common gain factor ( $G(t)$ ) was obtained by dividing the external reaction moment ( $M_e$ ) by the sum of the muscle moments (Equation 2). Multiplication of the muscle forces by the gain factor (Equation 3) amplified or attenuated the muscle forces, so that the summation of all of the tissue moments equaled the measured external moment, thereby preserving the *relative* contribution of the muscular components to the muscle moments (Equation 4).

$$F_m(t) = \left[ \left( \frac{EMG(t)}{EMG_{\max}} \right) * P_0 * XS_{aera} * \delta(t) * \Omega(t) + F_{pec}(t) \right]_m \quad (1)$$

$$G(t) = \frac{M_e(t)}{\sum_{m=1}^{38} (F_m(t) * r_m(t))} \quad (2)$$

$$M_m(t) = G(t) * F_m(t) * r_m(t) \quad (3)$$

$$M_r(t) = \left[ \sum_{m=1}^{38} M_m(t) + \sum_{l=1}^{13} M_l(t) + M_d(t) \right]_a \quad (4)$$

Representative model output is shown in Figure 4a. Muscle forces were calculated for the L1 - L4 pars lumborum muscle fascicles of longissimus thoracis and iliocostalis for the performance of a single trunk flexion and extension. To illustrate the effect of the MVC scaling factor, the magnitudes of the  $EMG_{\max}$  values were respectively doubled and halved (i.e. subject had twice, and then one-half of their original strength) and the muscle forces

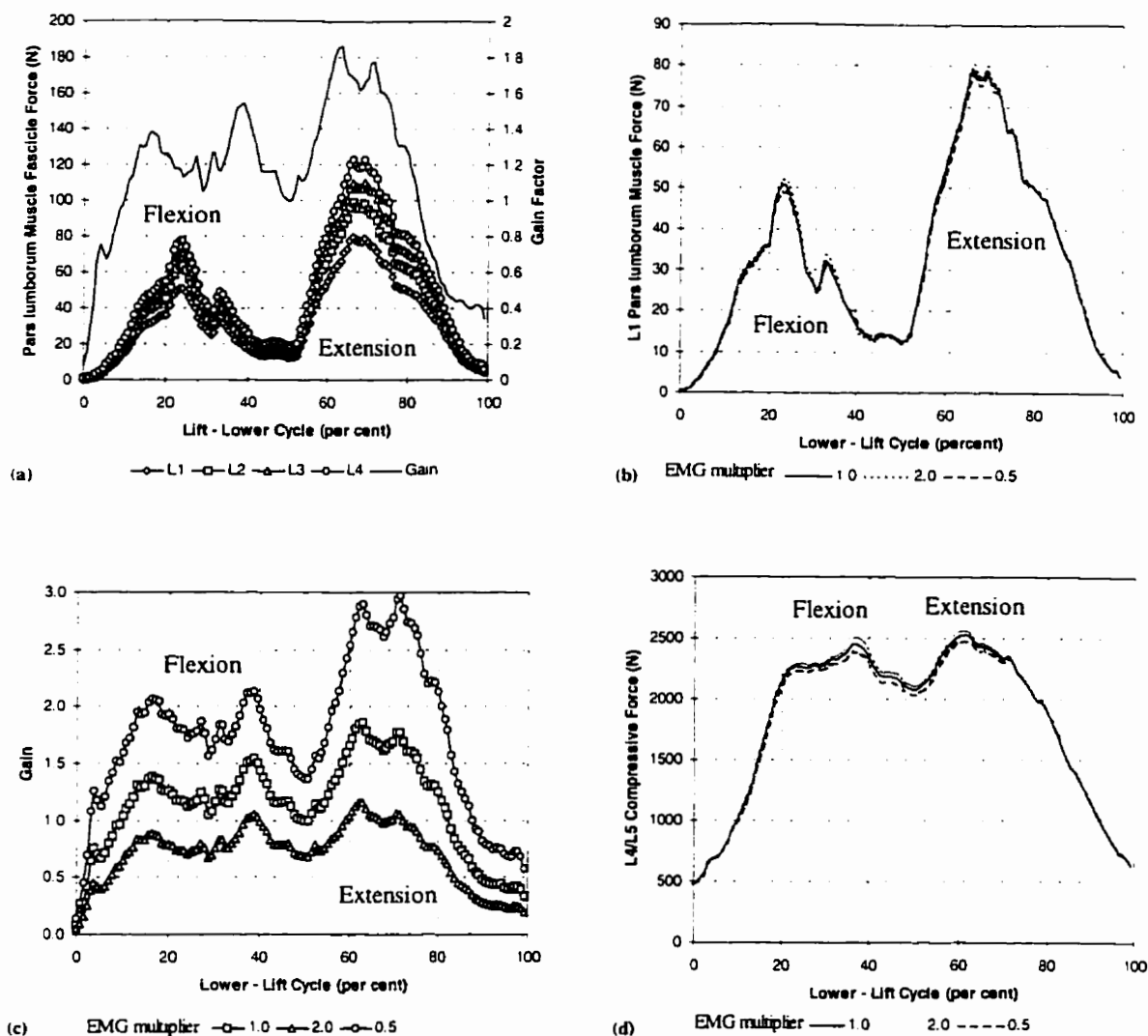


Figure 4: Representative output of the EMG assisted lumbar spine model during the performance of a single trunk flexion and extension with no load in the hands. (a) Muscle forces and gain factor, for the L1-L4 pars lumborum muscle fascicles of longissimus thoracis and iliocostalis. Altering the maximum EMG amplitudes used for scaling the EMG signal by a factor of 0.5 and 2.0 preserved the muscle forces calculated by the model (b), due to modulations in the gain factor (c). The L4/L5 compressive force was also unaffected (d).

recalculated. Altering the  $EMG_{max}$  values produced an RMS difference of only 0.8 N for the L1 pars lumborum muscle fascicle, which had an average force of 32.8 N (Figure 4b). The right lower erector spinae electrode, which supplied the EMG time history for eight muscle

fascicles, had an average force of 300 N for the flexion - extension trial. The altered EMG scaling factors produced an RMS difference of 8.3 N. Figure 4c illustrates the gain factor compensations produced by the altered  $EMG_{max}$  values. The effect of altering the MVC scaling factors on L4/L5 compression is shown in Figure 4d. The average compression was 1800 N with an RMS difference of 32 N. Regardless of the EMG level used as the MVC scaling factor, the model predicts the same net dynamic muscle forces and compressions.

This occurs because of the underlying assumption that the muscles are all recruited to the same level of activation (e.g. 50 or 100% MVC), which keeps the partitioning of the forces biologically consistent. If it were possible to have individuals recruit their musculature to a specific submaximal level, then this would be an alternative method for scaling the EMG. However, if there are differences in the levels of recruitment, then the model will incorrectly calculate muscle forces. For example, if the right lower erector spinae muscle was only activated to 50% of its MVC level, but 100% activation was assumed, then it would effectively be credited with force production two times what it was really producing. This would not only produce an error for this muscle, but it would also alter the scaling factor, resulting in incorrect forces for the other muscles.

However, the model's ability to predict the same muscle force for a specific level of EMG which is not truly "maximal" is still a very important observation. If, for each electrode site, the largest single EMG amplitude observed within a trial (i.e.  $EMG_{peak}$ ) is utilized in place of the  $EMG_{max}$  term in Equation 1, then muscle forces may be determined for each electrode site. Performing a trial of a different muscular contraction intensity and obtaining the  $EMG_{peak}$  for each of the electrode sites would allow the muscle forces associated with each electrode site



to be determined. For each electrode site this would result in a pair of  $EMG_{peak}$  values and associated muscle forces. This allows for an EMG-to-Force scaling factor to be calculated for each electrode site. This chapter discusses how this approach was utilized for a series of submaximal, isometric, flexion and extension efforts which then allowed the calculation of an EMG electrode site specific EMG-to-Force scaling factor.

Each submaximal flexion and extension trial was treated as if it were a Maximal Voluntary Contraction, with the  $EMG_{peak}$  term determined from the largest EMG amplitude produced in each channel for that specific *submaximal* trial. This  $EMG_{peak}$  term was used in place of the  $EMG_{max}$  term in Equation 1. Analysis of the lowest submaximal isometric effort (e.g. 50% MVC) via the spine model produced isometric muscle forces for that specific flexion or extension effort. The highest submaximal isometric trial (e.g. 75% MVC), which required a larger isometric moment, was then analyzed and the  $EMG_{peak}$  terms determined and substituted into Equation 1 in place of the  $EMG_{max}$  term. Analysis of that trial by the spine model produced a second set of isometric muscle forces for that specific trial. For each flexion and extension trial, the muscle forces were summed for each EMG electrode site. Then for each trial, the specific flexion or extension muscle forces and EMG amplitudes were averaged across the portion of the contraction where the L4/L5 moment was constant. This produced average muscle forces and EMG amplitudes for two different load (moment) situations, allowing the construction of a specific EMG-to-Force ( $EMG_{Force}$ ) scaling factor for each EMG channel (Equation 5). To facilitate calculation of the EMG-to-Force scaling factors, it was assumed that co-contraction did not occur during the submaximal isometric efforts. This

assumption was also verified for each subject prior to data analysis. Therefore, the respective flexor/extensor EMG was set to zero during performance of the isometric extension/flexion.

$$EMG_{Force} = \frac{\left[ \frac{AMF_{HI} - AMF_{LO}}{AEMG_{HI} - AEMG_{LO}} \right]_e}{XS_{area_e}} \quad (5)$$

The  $EMG_{max}$  and  $P_0$  terms in Equation 1 were replaced by the  $EMG_{Force}$  term, producing Equation 6.

$$F_m(t) = \left[ EMG(t) * EMG_{Force} * XS_{area} * \delta(t) * \Omega(t) + \right. \quad (6)$$

To evaluate the effect of incorporating the EMG-to-Force technique into the model, both the MVC (Equation 1) and EMG-to-Force (Equation 6) techniques were applied to dynamic, sagittal plane lifts.

### *Subjects*

Ten participants asymptomatic for low back pain, and four participants symptomatic for recurrent low back pain, were recruited for this study (Table 5). Each participant was tested on two separate occasions, with the low back pain population identifying a “good” and a “bad” day. For two of the asymptomatic participants, the first test session was their bad day. Test sessions averaged six and eight weeks apart for the symptomatic and asymptomatic groups, respectively. Each subject signed a consent form, approved by the Office of Human Research,

after reading an information letter that described the experimental procedures and associated risks.

**Table 5** Characteristics of study participants (mean (SD)).

	<u>Age (years)</u>	<u>Height (m)</u>	<u>Mass (kg)</u>
Normals (n = 10)	27 (2)	1.77 (0.05)	78.4 (7.4)
Patients (n = 4)	30 (11)	1.81 (0.03)	90.0 (10.7)

#### *Instrumentation*

Surface EMG was recorded bilaterally, using Ag-AgCl disposable electrodes (Medi-Trace, ECE 1801) with a 2.5 cm center-to-center distance, from the following muscles: rectus abdominis (3 cm lateral to the umbilicus, aligned straight upward), external oblique (approximately 15 cm lateral to the umbilicus, oriented diagonally down and inward), internal oblique (below the external oblique electrodes and just superior to the inguinal ligament, aligned diagonally up and outward), latissimus dorsi (lateral to T9 over the muscle belly, oriented up and outward), upper erector spinae (5 cm lateral to T9 spinous process, oriented up and slightly outward) and lower erector spinae (3 cm lateral to L3 spinous process, directed up and outward) (McGill, 1992, Sutarno, 1993). The raw myoelectric signals were prefiltered (bandwidth of 20 to 500 Hz) and amplified with a differential amplifier (CMRR of 80 db at 60 Hz). Reflective markers, representing the fifth metatarsal, heel, knee, hip, L4/L5, ear canal, shoulder, elbow, wrist and hand were attached to measure body joint displacements. Trunk kinematics were measured using the 3Space IsoTrak (Polhemus Navigation Sciences, McDonnell Douglas Electronics Company), which consisted of a magnetic source, placed over

the sacrum, and a sensor, placed over the 12<sup>th</sup> thoracic vertebrae spinous process. The three dimensional position and orientation of the sensor relative to the source, were calculated by the 3Space electronics. The 3Space signal was collected at 20.5 Hz.

### *Tasks*

Subjects were required to perform a standard set of Maximal Voluntary Contractions designed to elicit maximal EMG activation. The peak EMG amplitude observed for each channel was termed  $MVC_{max}$ , and these scaling factors were used when Equation 1 was used to calculate muscle forces (MVC method). Two, 10 second trials for each isometric effort were performed. For the abdominal musculature, the subject sat in a bent knee sit up position, hands behind the head, feet restrained, with their torso approximately 30° to the horizontal. A manual resistance was provided to the subject's shoulders while they performed a maximal sit-up and trunk twisting effort. For the extensor musculature, the subject lay prone over the edge of a test plinth, hands behind their head and their feet restrained. A maximal extensor effort was performed against manual resistance. For the latissimus dorsi, the subject sat on the edge of the plinth, with shoulders abducted to 90° and elbows flexed to 90°. Manual resistance was provided against the elbows while the subject attempted to adduct maximally. Finally, the subjects performed a series of "quasi isometric" efforts, attempting to activate each muscle group maximally while performing exertions similar to those of body builders posing in competition.

Subjects also performed isometric flexion and extension efforts, that ranged from 50% to 90% of their maximal flexion and extension moments, respectively. The 10 second

isometric efforts were performed in a prone (extension) and supine (flexion) position utilizing a two tier bench, so the postures would be similar to those used to elicit the MVCs (Figure 5). A pair of maximal effort isometric exertions were obtained by restraining the subject so that the greater trochanter was aligned with the edge of the upper tier. A chest harness was secured to an LVDT which was attached to the floor. The connecting fastener of the chest harness was aligned with the torso (head, arms and trunk) center of mass, measured from the greater trochanter (Winter, 1990). Subjects were then able to raise their torso off the lower tier in an extension, or flexion, effort and the isometric moment was measured. The single highest peak was termed maximal. Submaximal loading was induced by releasing the cable from the floor and having subjects raise their torso to a horizontal position. The external moment was increased by adding the appropriate load required to produce a pre-determined moment (e.g. 60%, 65%, 70% of maximum moment) and having subjects raise to a horizontal position. The external load was then incrementally increased until the subject reached the load that they could comfortably hold for the 10 second trial. The series of submaximal efforts produced a low (body weight) and a high moment (largest percentage) condition for analysis. The resulting low and high moment trials were used to construct the muscle specific EMG-to-Force scaling factors, as described previously, and were used when Equation 6 was used to calculate muscle forces ( $EMG_{Force}$  method).

Subjects then performed four repetitions of full range trunk flexion and extension, with loads of 0, 5 and 10 kg, which were assumed to be distributed evenly between the hands. Subjects utilized self selected style and pace, and the load originated 0.185 m in front of the

great toe. The load was lifted to knuckle height and the subjects were instructed to pause at each end point.



Figure 5: The postures used for producing the isometric extension maximal and submaximal efforts. Maximal extension efforts (a), were produced by raising the torso off of the bench and extending against the LVDT secured to the floor. Submaximal loading (b), was induced by hanging known loads to produce appropriate percentages of the maximum moment. Flexion efforts were performed in the supine posture.

#### *Data Reduction*

The LVDT and EMG signals were A/D converted (AT-MIO-16, 12 bit ADC, National Instrument, Inc.) at 1024 Hz and stored on magnetic-optical disk. The EMG signals were full wave rectified and low pass filtered (2nd order, single-pass, Butterworth) at a cutoff frequency of 2.5 Hz to produce a linear-envelope (LE). A 2.5 Hz cutoff frequency was selected because it reaches peak response to an impulse in 63 msec, which is in the middle of the 30 - 90 msec twitch response to peak tension found by Buchthal and Schmalbruch (1970). Olney and Winter (1985) found cutoff frequencies to range from 1.8 to 2.8 Hz for the rectus femoris. Potvin (1992) found 2.7 Hz to be the best frequency for the lower and upper erector spinae

musculature. The LE EMG and LVDT signals were interpolated, and the 3Space signals extrapolated, respectively, to 30 Hz, in order to match the video sampling frequency.

All trials were video taped (Panasonic AG-180U) in the sagittal plane and the joint coordinates digitized (Peak5, version 5.2, Peak Performance Technologies, Inc.) at 30 Hz to form a linked segment representation of the body. Right and left symmetry was assumed. The Z coordinate for each marker was assigned a positive or negative offset from midline equal to  $\frac{1}{2}$  of the shoulder width, as calculated from the subject's height (Winter, 1990).

The joint coordinate data, combined with the dynamic hand forces were input into the linked segment model, producing the L4/L5 reaction forces and moments. Muscle and ligament lengths were determined via the kinematic portion of the model and the lumbar spine position information (3Space). For the dynamic flexion and extension trials, the kinetic portion of the model calculated the passive tissue moments (ligament and disc) and then partitioned the remaining moment amongst the muscles, using either the MVC method, equation 1 or the  $EMG_{Force}$  method, equation 6.

### *Data Analysis*

To control for task initiation and termination in the dynamic trials, the second and third flexion and extensions were used for data analysis. Shoulder marker velocity was used to determine the start and end points for each flexion and extension movement segment. To facilitate within and between subject comparisons, model outputs for each flexion and extension segment were normalized to fifty data points. The muscle force per cross sectional area ( $P_0$ ) was set to 35 N/cm<sup>2</sup>. The individual muscle fascicle forces were summed together for

each electrode site. This allowed a direct comparison of the muscle force being produced by each electrode. To keep muscle forces within a biologically valid range, the gain factor ( $G$ ), was not allowed to get larger than 3.5. Trials in which this occurred were not included in data analysis.

To assess model behavior for the individuals asymptomatic for low back pain, the peak and average compressions, flexor muscle moments, and extensor muscle moments were analyzed using a repeated measures ANOVA of Method (2) by Day (2) by Load (3). The flexor and extensor muscle forces were analyzed in repeated measure ANOVAs of Method (2) by Day (2) by Load (3) by Electrode Muscle Force (6).

Model behavior for the individuals symptomatic for low back pain was assessed using a repeated measures design of Method (2) by Day (2) for peak and average, compression, flexor moment and extensor moment, and Method (2) by Day (2) by Muscle Force (6) for the flexor and extensor muscles.

To satisfy the assumption of similarity of variance, it was necessary to perform a log transformation of the muscle moment and muscle force data.

## **Results**

The repeated measures analysis requires that observations be available for each subject in each condition. To maximize the number of asymptomatic individuals available for comparison ( $n = 9$ ), the second flexion was used for assessment. To maximize the analysis for the number of individuals symptomatic for low back pain, the statistical analysis was restricted to the first flexion performed with the 10 kg load.



### *Effect of Day*

The day of testing produced no significant difference in any of the model parameters, for either test group. To simplify the graphical presentation of the processing method and load effects, the data were averaged across days.

### *Effect of Processing Method*

**Compression:** For the individuals asymptomatic for low back pain, the EMG<sub>Force</sub> processing method produced significantly lower peak and average compressions (Figure 5a). The differences in peak compression ranged from 245 - 410 N (Table 6). For the symptomatic individuals, the trend of the EMG<sub>Force</sub> method producing a lower peak L4/L5 compression was not statistically different for the 10 kg load, ( $p < .056$ ), although the differences ranged from 400 - 900 N (Table 6). The EMG<sub>Force</sub> method did produce a significantly lower average L4/L5 compressions in the SYMP group (Figure 5b). Interestingly, the difference in the MVC and

**Table 6** The difference (MVC - EMG<sub>Force</sub>) in peak compression (N) produced during the dynamic flexions due to the technique used to calculate the EMG scaling factors. There was no statistical difference between days for the loads in each group.

Load (kg)	Day 1 / Good Day			Day 2 / Bad Day		
	0	5	10	0	5	10
ASYMP	245	260	399	380	410	262
SYMP	---	---	396	---	---	902

EMG<sub>Force</sub> methods for the 10 kg load was similar for the SYMP group's Good Day and the ASYMP group's Day 1 and Day 2.

**Muscle Moments:** For the symptomatic and asymptomatic subjects, the EMG<sub>Force</sub> method produced significantly lower peak and average extensor and flexor muscle moments (Figure 6a - d).

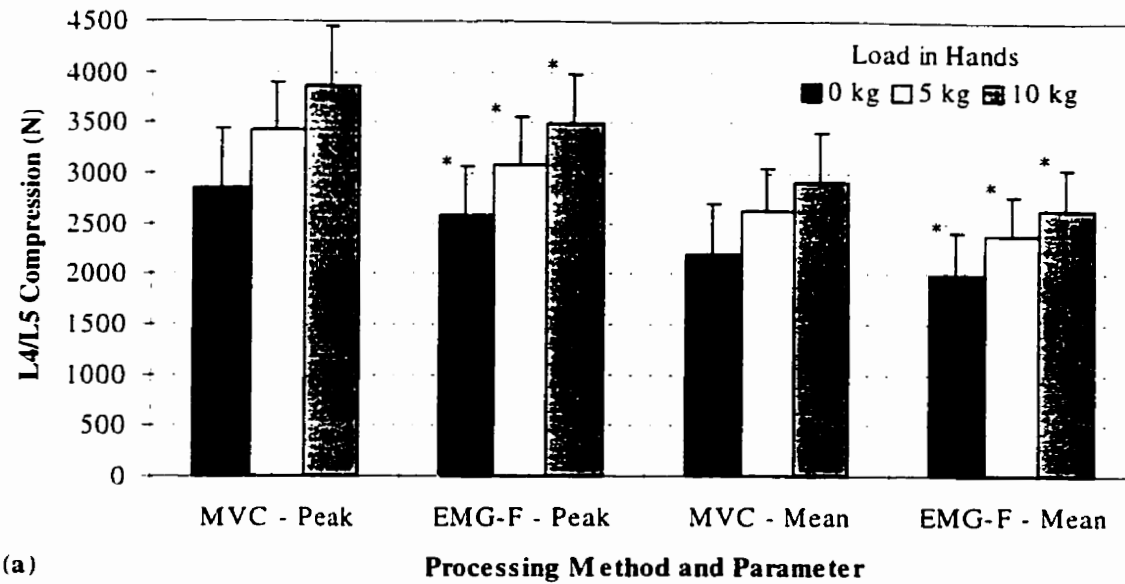
**Muscle Forces:** The average and peak extensor muscle forces were found not to be affected by the processing technique for either group (Figure 7 a, c). However, the EMG<sub>Force</sub> processing technique resulted in significantly lower peak and average flexor muscle forces for both groups of subjects (Figure 7 b, d).

#### *Effect of Load*

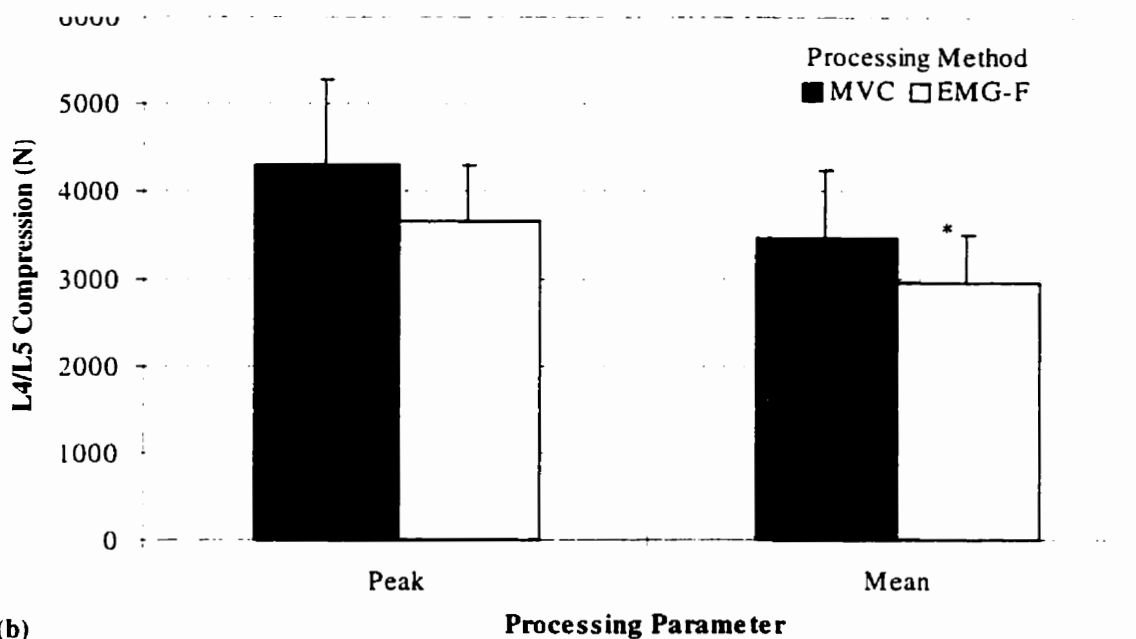
**Compression:** For the asymptomatic individuals the peak and average values of compression, were all significantly increased ( $p < .0005$ ) by the load held in the hands (Figure 5a).

**Muscle Moments:** For the asymptomatic participants, the load in the hands significantly increased the peak and average values of the extensor muscle moments (Figure 6a), but not the peak and average flexor muscle moments (Figure 6b).

**Muscle Forces:** Figure 7a shows the significant increase in the peak and average extensor muscle forces with an increase in hand load for the asymptomatic individuals. However, the peak and average values of flexor muscle forces were not affected significantly (Figure 7b).



(a)



(b)

Figure 6: The effect of processing method and load on the peak and mean L4/L5 compressive force. For the asymptomatic individuals (a), the \* indicates that during the second flexion, the peak and mean compressions calculated using the  $EMG_{Force}$  method were significantly smaller ( $p < .0005$ ) than those obtained using the MVC technique. The load held in the hands significantly increased the peak and mean compressive forces ( $p < .0005$ ). For the symptomatic individuals (b), during the first flexion with the 10 kg load in the hand, the processing method produced no difference in the peak L4/L5 compressive force ( $p > .056$ ), but the mean compression for the  $EMG_{Force}$  method was significantly lower ( $p < .039$ ), as indicated by the \*.

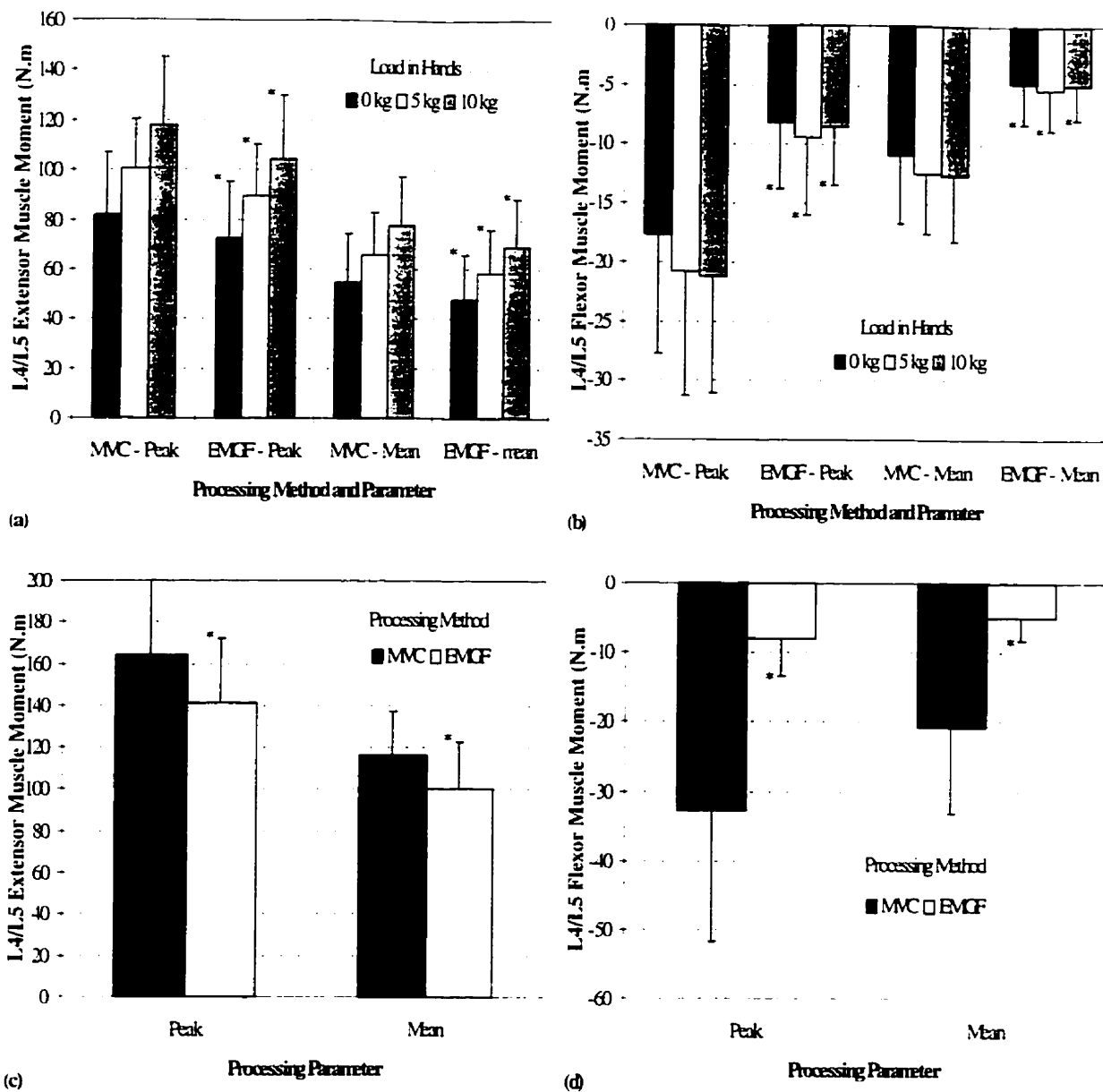


Figure 7: The effect of processing method and load on the peak and mean L4/L5 muscle moments. For the asymptomatic participants, the \* indicates that during the second flexion, the  $EMG_{Force}$  technique produced significantly smaller peak ( $p < .0005$ ) and mean ( $p < .0005$ ) extensor muscle moments (6a) and significantly smaller peak ( $p < .002$ ) and mean ( $p < .0005$ ) flexor muscle moments (6b), than those calculated using the MVC method. The load held in the hands increased the peak ( $p < .0005$ ) and mean ( $p < .02$ ) extensor muscle moment significantly (6a), but had no effect on either the peak or mean flexor muscle moment (6b). For the symptomatic individuals, performing the first flexion with the 10 kg load, the \* indicates that the  $EMG_{Force}$  technique produced significantly smaller peak ( $p < .034$ ) and mean ( $p < .021$ ) extensor muscle moments (6b) and peak ( $p < .016$ ) and mean ( $p < .014$ ) flexor muscle moments (6d).

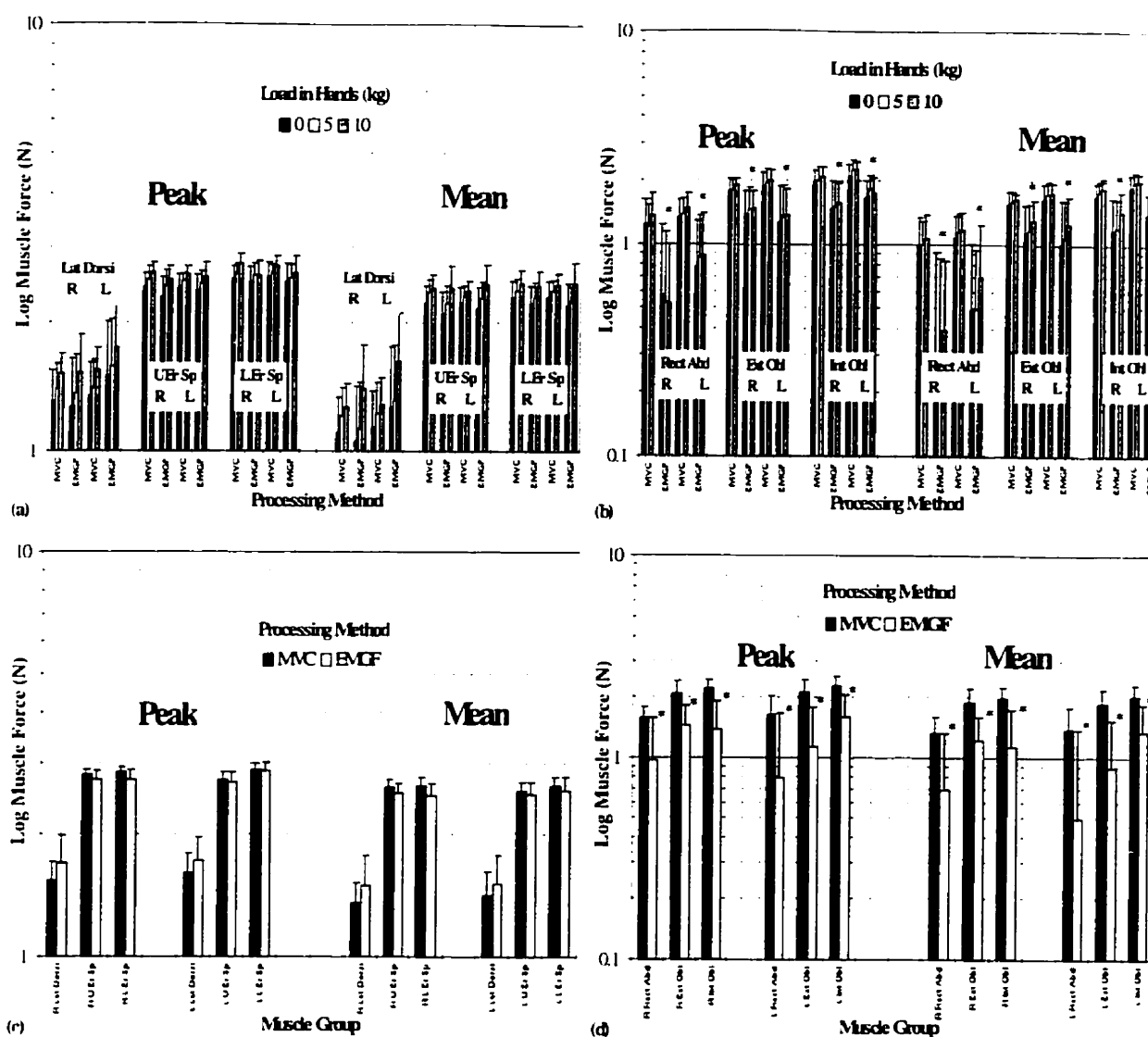


Figure 8: The effect of processing method and load on the peak and mean muscle forces. For the asymptomatic group, performing the second flexion, there was no statistical difference between the  $EMG_{Force}$  and MVC methods in the peak and mean extensor muscle forces (7a). The load held in the hands significantly increased both the peak ( $p < .0005$ ) and average ( $p < .0005$ ) extensor forces (7a). The peak and mean flexor muscle forces (7b) were both significantly lower using the  $EMG_{Force}$  processing method ( $p < .0005$ ), as indicated by the \*. The hand load had no effect on the peak flexor muscle force (7b), but did increase the mean flexor muscle force significantly ( $p < .037$ ). For the symptomatic group, during the first flexion with the 10 kg load, the processing method had no effect on peak or mean extensor muscle force (7c). The flexor muscle forces (7d) were significantly smaller using the  $EMG_{Force}$  method for both the peak ( $p < .025$ ) and mean ( $p < .022$ ) values, as indicated by the \*. R = right; L = left; Lat Dorsi = latissimus dorsi; U Er Sp = upper erector spinae; L Er Sp = lower erector spinae; Rect Abd = rectus abdominis; Ext Obl = external oblique; Int Obl = internal oblique.

## Discussion

This study compared a maximal voluntary contraction (MVC) and an EMG-to-Force ( $EMG_{Force}$ ) methodology for determining EMG scaling factors in order to calculate muscle forces of the lumbar spine, using an EMG assisted model. In flexion/extension tasks with hand loads of 0, 5 and 10 kg, the  $EMG_{Force}$  method, compared to the standard MVC method, resulted in significantly lower compressions, flexor and extensor muscle moments and flexor muscle forces in individuals asymptomatic for low back pain. The  $EMG_{Force}$  method successfully produced the same peak and average extensor muscle forces as the MVC method. Both of these findings occur as a result of the  $EMG_{Force}$  calibration procedure utilized in this study.

With the MVC approach, the model incorporates a non-linear EMG-to-Force term for the calculation of muscle forces (Figure 8). Points along this curve are also used when calculating the EMG-F scaling factors. However, the equation which calculates muscle force for the  $EMG_{Force}$  method is linear, because the 100% MVC value is unknown. This would produce only slightly different muscle forces. However, these small differences are then magnified by differences in gain factors.

Differences in gain factor occur as a function of the horizontal posture used in the calibration procedure. Each subject's torso mass represented a substantial percentage of their maximal flexion and extension moment producing ability. This resulted in the calculation of the  $EMG_{Force}$  scaling factors occurring in the upper region of the EMG-to-Force relationship. In this area, there is very little difference in the amount of force produced by each method per each unit of EMG (Figure 8). At lower levels of EMG, the large calibration moments result in

an overestimation of the amount of force produced per unit EMG for the  $EMG_{Force}$  method, resulting in a lower gain.

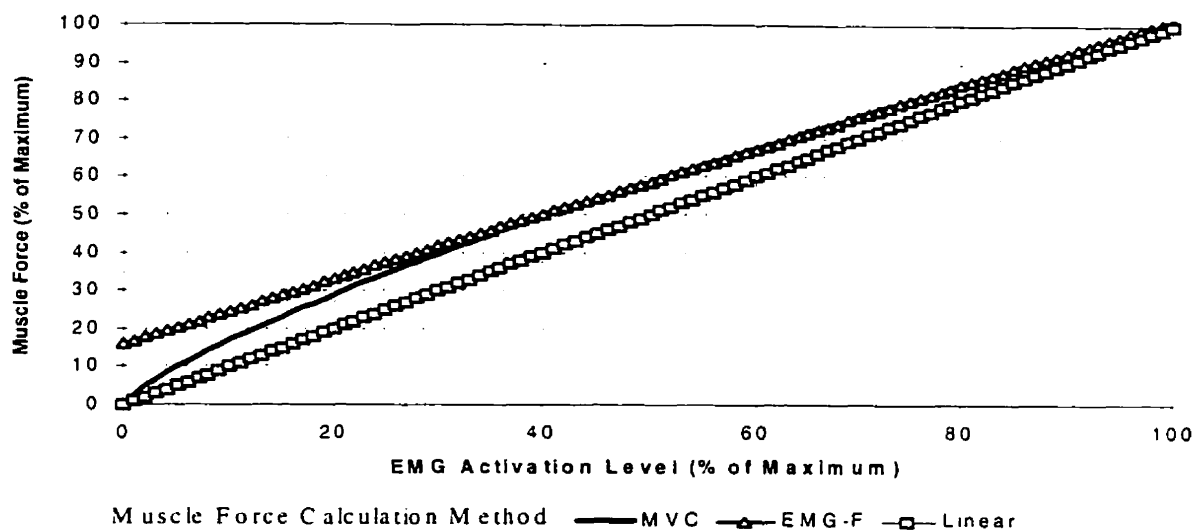


Figure 9: The muscle force produced in the lumbar spine model as a function of EMG activation level. The dark line indicates the non-linear method used with the standard, MVC approach. The open squares illustrate a linear force-EMG relationship. The  $EMG_{Force}$  method uses a combination of the two, calculating the muscle forces at two levels of EMG using the MVC method and then assuming a linear EMG-to-force relationship.

The  $EMG_{Force}$  method is also sensitive to the calibration forces calculated for each muscle. Thus, even though for both trials, the muscle force and EMG were averaged over a period of time when the moment was stable, fluctuations in the EMG signal and muscle forces calculated would allow changes in the  $EMG_{Force}$  relationship to occur (Figure 9). Overall, the  $EMG_{Force}$  calibration method calculated significantly smaller force values for the flexor musculature electrodes and similar force values for the extensor musculature electrodes. This resulted in a smaller calculated flexor muscle moment, which in turn lead to the calculation of a greater net extensor moment, and ultimately, a smaller gain factor. The combined effect of the linear  $EMG_{Force}$  muscle force calculation and differences in gain factor resulted in the

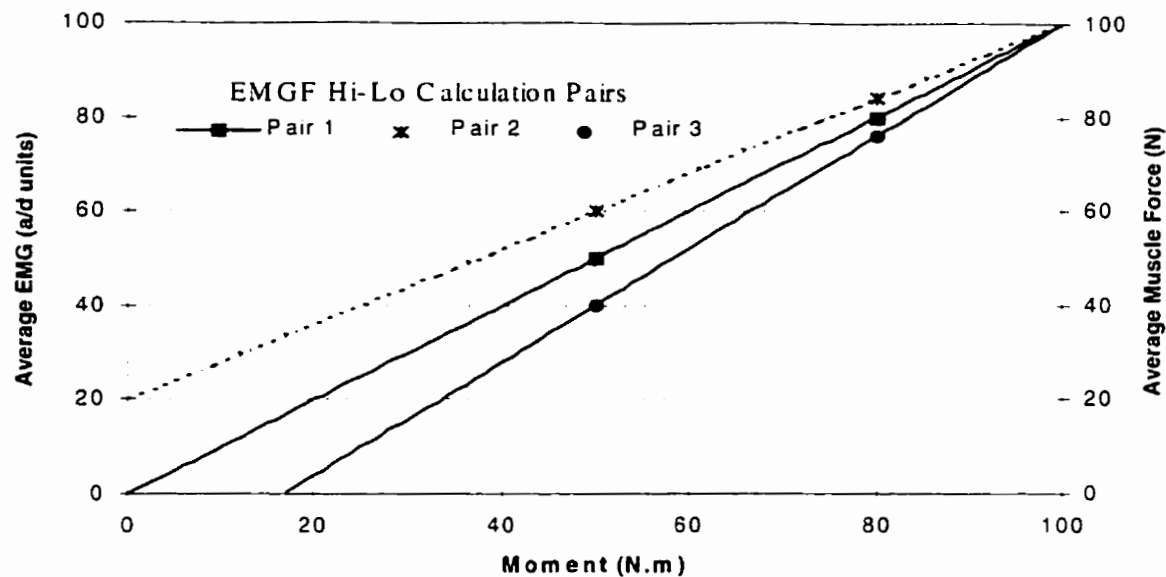


Figure 10: An illustration of the average EMG, average muscle force and moment relationship. Ideally, The solid squares show the ideal relationship. However, increases or decreases in either the average EMG value or the average muscle force over the period where the moment is stable would alter the  $EMG_{Force}$  scaling factor calculated.

calculation of less muscle force, flexor and extensor moment and L4/L5 compression.

The  $EMG_{Force}$  processing method allowed a sophisticated EMG assisted model of the lumbar spine to be successfully applied to individuals suffering from recurrent low back pain who knowingly could not provide true maximum voluntary contractions (MVCs). However, using the  $EMG_{Force}$  technique did result in peak and average spinal compressions that were significantly smaller than those calculated using the MVC method. Nevertheless, this difference should not minimize the importance of the  $EMG_{Force}$  processing method for the compressions in the ASYMP group were only underestimated by 250 - 400 N, depending on the load being handled. Also, for the SYMP group's Good Day, the underestimate was also only 400 N. Thus, the magnitudes of the spinal compressions could be corrected if desired.



Secondly, for the SYMP group, no statistical difference in peak compression was found between the two methods (Figure 5b). Yet according to the SYMP group's data a difference in the compression would be expected between the two methods (Figure 5a). It may be that the compressive forces calculated by the  $EMG_{Force}$  method were elevated. This would seem unlikely because the  $EMG_{Force}$  method incorporated moments that were easier to produce than MVCs. It is more probable that the EMG scaling factors determined using the MVC method were altered as result of not producing true MVCs. If this is the reason for the compressions not being significantly different, then the compressions would be even greater than those calculated.

If the compressions for symptomatic individuals are going to be underestimated, as occurred in this study, the  $EMG_{Force}$  method would be the preferred method for calculating the EMG scaling factors because the required muscle contractions are easier for symptomatic individuals to perform. Also, compression is only one parameter calculated by the model. Muscle forces are also calculated and there were no differences in the extensor forces for either group of individuals. Changes in muscle force distribution may be one method of monitoring recovery for symptomatic individuals. If the MVC method alters the scaling factors, then the  $EMG_{Force}$  method would appear to be a superior technique.

The  $EMG_{Force}$  method may be desirable to use on populations other than those symptomatic for low back pain. Populations in which the individuals are inexperienced in the performance of MVCs (e.g. industrial workers) would find the  $EMG_{Force}$  method easier to perform. Also, if the relative magnitudes and or distribution of muscle forces were the primary factors of interest, then the  $EMG_{Force}$  method would also appear to be advantageous.

The non-significance of test day is an important finding for EMG assisted models that use either method for calculating muscle forces. As the areas and subject populations of research that EMG assisted models are applied to increase, (e.g. assessment of treatment modalities for individuals with low back pain), then differences in outputs may be more confidently assigned to factors other than the day of the test.

The production of maximal voluntary contractions (MVCs) is a challenging task, and one in which skilled performers may require several attempts using different postures in order to obtain a maximal value (McGill, 1991). The knowledge that different postures produce different maximal amplitudes, means that a researcher may never be certain that some other posture may have elicited an even greater value. Mirka (1991) has also demonstrated that trunk angle is a factor which needs to be considered when normalizing EMGs. Zeh et al. (1986) encountered several reports of back discomfort and objective signs of injury in three employees during strength testing involving MVCs. As an alternative method for scaling EMGs, the  $EMG_{Force}$  method addresses these concerns and provides other important advantages.

The first and most significant is that MVC's are not performed. This allows populations (e.g. low back pain, industrial workers) that are not typically included in research that incorporates these models. Not having to perform MVCs also alleviates the concerns of low back injury associated with MVCs (Zeh et al., 1986). As with the MVC approach, the  $EMG_{Force}$  calibration is both subject and muscle specific. However, the  $EMG_{Force}$  method has the added advantage that a specific posture may be utilized, if desired. In this study, a horizontal posture was selected in an attempt to match the MVC postures. This particular strategy was time consuming, because of the size of the moments used and the way they were

created. Adopting other postures (e.g. standing) and incorporating visual feedback for the maintenance of a desired moment would decrease the length of time required to perform the calibration and allow the assessment of posture specific activities. The calibration procedure may also be performed using load levels much closer to those being evaluated. For example, in a lifting task the extensor muscles may be calibrated at 40% to 50% MVC, while the flexor muscles are calibrated at a much smaller level. This would minimize the errors associated with the incorporation of a linear EMG to Force relationship.

In order to facilitate the comparison of the two methods, the model gain factor was allowed to vary, even for very small moments. Both the magnitude and the variability of the model gain factor contain information which reflect the biology of the system being modeled. The magnitude of the model gain factor indicates how well an individual "fits" the model. For example, if the gain factor had an average value of 2, it would reflect that perhaps the  $35 \text{ N/cm}^2$  utilized for  $P_0$  was an underestimate for that individual. The variability about the average value provides an indication of where in the movement the model parameters required much more or less modulation. For example, during periods when very small moments are required (e.g. standing upright), the muscles have stability requirements that they must satisfy, not moment requirements (Cholewicki and McGill, 1996). This produces a situation where the gain factor becomes very large because there is a very small signal-to-noise ratio (moment-to-EMG), so that data from the model during these periods may not be valid. However, as the moment demand increases, the muscles function to meet this demand and the model output should be correct. If the gain factor becomes variable during these periods it allows the effects of other model modulators to be examined (e.g. velocity factor, ligament contribution). This

information may then be used to correct the biological representation in the model to more accurately reflect the human system.

In summary, a technique for the calculation of EMG scaling factors required for using an EMG assisted model of the lumbar spine was developed and successfully applied to symptomatic and asymptomatic low back pain populations. The results show that the EMG<sub>Force</sub> method produces significantly lower compressions and flexor muscle forces, but no difference in extensor muscle forces when compared to the standard MVC approach. The day of testing was found not to significantly affect model output for either method and the EMG<sub>Force</sub> approach has several benefits for future research.

## **Chapter V**

### **The Assessment of Spine Movement Dysfunction by a Commercial Dynamometer, EMG and an EMG Assisted Model**

#### **Introduction**

The understanding of injury mechanisms is pre-requisite to the development of effective rehabilitation and prevention methods (Norman, 1992). Yet for the low back, the mechanisms of injury are not well understood. It is estimated that in 20 - 85% of low back pain (LBP) cases, the exact etiology of injury is unknown (White and Gordon, 1982). Typically, if a low back injury is not structural or neural in nature, then abnormal muscular activity and other soft tissues are suspected. Also, even when the cause of an injury is known, it is probable that normal muscle function will be impaired secondary to pain or mechanical disorders (De Luca, 1993). It is not surprising then, that researchers have developed many methods in an effort to quantify muscle function, and that clinicians have developed numerous treatments to improve the muscle function of LBP sufferers.

A common clinical goal that conservative treatment methods have focused on is the return or improvement of an individual's "abnormal" function to "normal". Typically this is achieved through the use of modalities such as flexibility, strengthening and/or manipulation. The variable(s) used to quantify "normal" depends on the clinician, but being pain free is typically associated with normal function. However, due to the difficulty in quantifying pain, and the increased costs associated with LBP, there has been a move towards objective methods for the quantification and treatment of LBP individuals.

The quest for objectivity has led to the development of uni-axial and multi-axial computerized spinal dynamometers and they have become a routine component of the assessment and rehabilitation procedure of individuals with LBP (Spengler and Szpalski, 1990). Quantification of performance, computerized summary reports, and the relatively short testing time are all reasons for the increased utilization of these dynamometers. Incorporating objective assessments as part of the rehabilitation program has been demonstrated to be more effective than simple pain management programs (Mayer et al., 1986).

The purpose of this study was to determine whether the understanding of spine movement dysfunction, as indicated by abnormal displacement, velocity, and torso moment data, was augmented by knowledge of the EMG activity from select abdominal and back extensor muscles and/or by knowledge of the individual torso tissue forces estimated from an EMG assisted, dynamic, three dimensional spine model. This was investigated by the assessment of individuals symptomatic and asymptomatic for low back pain, on two separate test days, using:

- 1) a computerized lumbar spine dynamometer (Isostation B-200) and a clinical evaluation protocol (OOC software, Version 3.1),
- 2) a custom profile analysis of the B-200 moment-time histories,
- 3) the assessment of spinal EMG profiles (presented as mV and %MVC) produced from the performance of a dynamic flexion-extension task, and,
- 4) the assessment of spinal muscle force profiles, estimated by an EMG-assisted model of the lumbar spine, produced while performing a dynamic flexion-extension task.

### **Rationale**

The Isostation B-200 is a three dimensional lumbar spine dynamometer designed to objectively measure "back function". It compares an individual's torso displacement, velocity and moment data, produced during a specific clinical evaluation protocol (Occupational Orthopaedic Center (OOC) Version 3.1), against a data base in order to classify or quantify an individual's level of "back dysfunction". The resultant level of back dysfunction and associated performance data may then be used as a guide in the rehabilitation process and to assist in determining when an individual has regained "normal" function. Although this approach identifies normal and dysfunctional spine movements, it oversimplifies the individual's movement patterns. By concentrating on the magnitude of the resultant peak and/or average moments and velocities, it ignores the moment time history that produced the movements. Evaluating this aspect of the performance may further enhance the assessment. Also, this dynamometer oversimplifies the lumbar spine. The dynamometer's rotation, flexion extension and lateral flexion mechanical axes do not align with the mechanical axes of the lumbar spine.

Electromyography (EMG) of the lumbar spine musculature has emerged as a method through which the function of the musculature in individuals symptomatic and asymptomatic for LBP may be evaluated. The amplitude component of the EMG signal from the spinal musculature has been used to quantify the flexion-relaxation phenomenon (Floyd and Silver, 1955; Triano and Schultz, 1987; Sutarno, 1993). In 1968, de Vries found greater amplitude changes for people with LBP in the fatigue response of the spinal muscles during quiet standing. Other researchers have looked at a combination of static and dynamic tasks to try and

identify differences associated with LBP and the results have been mixed. Ahern et al. (1988) found no difference between a control and a LBP group in static tasks, but in dynamic tasks there was decreased EMG activity in the LBP group. Arena et al., (1989) found that for standing, sitting, laying prone and flexion-extension, controls had decreased EMG activity compared to those with intervertebral disk disorder and those with unspecified musculoskeletal backache. Nouwen et al., (1987) found no significant differences in bilateral paraspinal EMG between LBP patients and pain-free controls during the performance of rotation, flexion-extension or lateral bending.

One reason for the divergent results is the different tasks that are performed. Another important difference is the EMG reporting method. Each of these studies have used  $\mu V$  to express the EMG and have used a single number to represent the activity performed, either mean EMG ( $\mu V$ ), integrated EMG ( $\mu V \cdot s$ ), or rate of EMG ( $\mu V/s$ ) production. Arena et al., (1990) found that surface EMG of the paraspinal muscles was more reliable when expressed as an absolute rather than a relative measure and many researchers do use  $\mu V$ . However, Basmajian and De Luca (1985) recommend that the EMG signal amplitude should be normalized to a convenient and referable quantity, such as its maximum value. Also, by condensing an entire movement cycle or task into a single number, important differences in the task's EMG time history may be lost. Therefore, it is important the entire EMG time history of the task be utilized in the analysis.

Sutarno (1993) documented 3D kinematic and EMG time histories of 14 trunk muscles during the performance of uni-axial twist, flexion-extension and side-bend movements for 24 normals and 5 low back pain individuals. Difficulty in categorizing the LBP individuals using



the kinematic and EMG variables may have been due to the use of a small load (10 kg) and the fact that only one of the LBP individuals was in pain at the time of testing. Although the technique was applied in a laboratory setting, it could easily be adapted for use in a clinical environment.

Other researchers have used changes in the EMG power spectrum, associated with fatigue during the performance of isometric tasks, in order to differentiate between normals and LBP individuals (Kondraske et al., 1987; Roy et al., 1989; Roy et al., 1990; Biedermann et al., 1991, Moritani et al., 1992; De Luca, 1993). The power spectrum method facilitates the classification of individuals, assesses some aspects of muscle function and assists in tracking improvement during the rehabilitation process. However, this method is limited to the performance of static contractions. Therefore, it fails to identify the specific impairments that are associated with the performance of activities of daily living, such as flexion and extension.

Determining the force time histories of the lumbar musculature is another method through which normal function may be quantified. Knowledge of these force time histories for individuals asymptomatic and symptomatic for LBP may also provide insight into injury mechanisms. Forces in the lumbar spine may be estimated using sophisticated, computerized models (McGill and Norman, 1986, McGill, 1992, Marras and Sommerich, 1991a, Granata and Marras, 1993). McGill (1992), developed a three dimensional dynamic model which uses EMG as a biological input signal in order to partition the restorative moment into force/time histories for 50 muscle fascicles, 12 ligament vectors and the compression and shear forces acting on the L4/L5 motion unit. Previously, this model required the performance of maximum voluntary contractions (MVCs) in order to calculate the scaling factor for the EMGs. Pain,

decreased motivation and/or fear of re-injury are all reasons which may prevent an individual from performing a "true" MVC. This prevented this model from being used in conjunction with LBP individuals. An adaptation of the method for determining the model's EMG scaling factors, as reported in the previous chapter, now allows this model to be used in conjunction with LBP individuals.

Knowledge of the force time histories of the lumbar spine musculature would also be beneficial in assessing recovery from injury. With recovery, performance parameters such as peak isometric moment, peak velocity or endurance time may show improvements or return to normal. But they provide no information regarding changes in muscular function, nor do they necessarily relate to activities of daily living. However, the comparison of the muscle forces during a dysfunctional or painful period (i.e. a bad day), versus those during a functional or pain free period (i.e. a good day), may reveal changes in the function of the lumbar spine musculature. Another advantage to assessing muscle forces is that the EMG time histories required as model inputs, may also be used by themselves as an intermediate method of assessing muscle function. This EMG time-histories may be beneficial in improving our understanding of the relationships between muscular function and physical performance.

Arena et al. (1991) assessed the muscular function of people with LBP performing six tasks, on days with low, and high pain states. A non-significant trend of increased mean EMG activity was observed. Differences may not have been detected due to the absence of normalization and/or representation of each task by a single number. To this author's knowledge, the assessment of muscle forces in individuals symptomatic for LBP during days of different levels of functionality, has never been performed.

In summary, there is a clinical need for quantitative methods to evaluate lumbar spine function. It is possible that the assessment produced by one commonly used evaluation tool, the Isostation B-200, may be augmented by evaluation of the moment time histories produced during testing. Incorporating EMG of the spinal musculature as a component of the evaluation is an attractive technique for the quantification of muscular performance. EMG assessments of individuals symptomatic for LBP in previous research have typically ignored assessment of the EMG time history and the performance tasks have not been strongly related to activities of daily living. EMG of the spinal musculature may also be used as an input for a computer model of the spine, allowing lumbar spine muscle forces to be estimated. Finally, in previous investigations of muscular function, the testing has typically involved a single evaluation period and the focus has been on comparing the results for individuals symptomatic for LBP to an asymptomatic group. Very little research has followed individuals symptomatic for LBP longitudinally, in an effort to compare an individual's bad day results to their good day.

## **Methods**

### *Subjects*

Ten participants asymptomatic for low back pain (ASYMP Group) and four participants symptomatic for recurrent low back pain (SYMP Group) were recruited for this study (Table 7). Each participant was tested on two separate occasions, with the low back pain population identifying a "good" and a "bad" day. For two of the symptomatic participants, the first test session was their bad day. Test sessions averaged six and eight weeks apart for the symptomatic and asymptomatic groups, respectively. Each subject signed a consent form,

approved by the Office of Human Research, after reading an information letter that described the experimental procedures and associated risks.

**Table 7** Characteristics of study participants (mean (SD)).

	<u>Age (years)</u>	<u>Height (m)</u>	<u>Mass (kg)</u>
Normals	27 (2)	1.77 (0.05)	78.4 (7.4)
Patients	30 (11)	1.81 (0.03)	90.0 (10.7)

#### *Instrumentation*

During the isometric and dynamic flexion and extension trials EMG, body segment locations, trunk kinematics and torso/hand forces were recorded. Surface EMG was recorded bilaterally (R = Right, L = Left), using Ag-AgCl disposable electrodes (Medi-Trace, ECE 1801) with a 2.5 cm center-to-center distance, from the following muscles: rectus abdominis (**RA**, 3 cm lateral to the umbilicus, aligned straight upward), external oblique (**EO**, approximately 15 cm lateral to the umbilicus, oriented diagonally down and inward), internal oblique (**IO**, below the external oblique electrodes and just superior to the inguinal ligament, aligned diagonally up and outward), latissimus dorsi (**LD**, lateral to T9 over the muscle belly, oriented up and outward), upper erector spinae (**UES**, 5 cm lateral to T9 spinous process, oriented up and slightly outward) and lower erector spinae (**LES**, 3 cm lateral to L3 spinous process, directed up and outward) (McGill, 1992, Sutarno, 1993). The raw myoelectric signals were prefiltered (bandwidth of 20 to 500 Hz) and amplified with a differential amplifier (CMRR of 80 db at 60 Hz). Reflective markers, representing the fifth metatarsal, heel, knee, hip, L4/L5, ear canal,

shoulder, elbow, wrist and hand were attached to measure body segment displacements. Trunk kinematics were measured using the 3Space IsoTrak (Polhemus Navigation Sciences, McDonnell Douglas Electronics Company), which consisted of a magnetic source, placed over the sacrum, and a sensor, placed over the 12<sup>th</sup> thoracic vertebra spinous process. The three dimensional position and orientation of the sensor relative to the source, were calculated by the 3Space electronics and were sampled at 20.5 Hz. The torso and hand forces produced in the vertical direction during the isometric and dynamic flexion and extension trials were measured using a linear variable differential transformer (LVDT) and amplifier (Daytronics Transducer Amplifier, Model 3270).

### *Tasks*

#### Oswestry Questionnaire

At the start of each test sessions, subjects completed the Oswestry low back pain disability questionnaire (Fairbank et al., 1980, see Appendix A) .

#### Pain Scale

At the start of each test session, each subject was asked to indicate “How much pain do you feel at this time?” by placing a mark on a 10 cm visual analogue scale (VAS)(Appendix B). The left and right ends of the line were labeled “No Pain” and “Worst Imaginable”, respectively (Von Korff et al., 1992). Subjects also completed a new VAS following the completion of the isometric flexions and extensions, the dynamic flexions and extensions, the Isostation B-200 and 24 hours post-testing.

### Maximal Voluntary Contractions

Subjects were required to perform a standard set of Maximal Voluntary Contractions designed to elicit maximal EMG activation so that the EMGs could be presented as %MVC. The peak EMG amplitude observed for each channel was termed  $MVC_{max}$ . Two, 10 second trials for each isometric effort were performed. For the abdominal musculature, the subject sat in a bent knee sit up position, hands behind the head, feet restrained, with their torso approximately  $30^\circ$  to the horizontal. A manual resistance was provided to the subject's shoulders while they performed a maximal sit-up and trunk twisting effort. For the extensor musculature, the subject lay prone over the edge of a test plinth, hands behind their head and their feet restrained. A maximal extensor effort was performed against manual resistance. For the latissimus dorsi, the subject sat on the edge of the plinth, with shoulders abducted to  $90^\circ$  and elbows flexed to  $90^\circ$ . Manual resistance was provided against the elbows while the subject attempted to adduct maximally. Finally, the subjects performed a series of "quasi isometric" efforts, attempting to activate each muscle group maximally while performing exertions similar to those of body builders posing in competition.

### Isometric Flexion and Extensions

Subjects performed isometric flexion and extension efforts, that ranged from 50% to 90% of their maximal flexion and extension moments, respectively. The 10 second isometric efforts were performed in a prone (extension) and supine (flexion) position utilizing a two tier bench. The subject was positioned so that the greater trochanter was aligned with the edge of the upper tier and their legs were restrained with velcro straps. A chest harness was secured to

an LVDT which was attached to the floor. The connecting fastener of the chest harness was aligned with the torso (head, arms and trunk) center of mass, measured from the greater trochanter (Winter, 1990). Subjects were then able to raise their torso off of the lower tier in an extension, or flexion, effort and the isometric moment was measured. A pair of maximal effort isometric exertions were obtained and the single highest peak was termed maximal. Submaximal loading was induced by releasing the cable from the floor and having subjects raise their torso to a horizontal position. The external moment was increased by adding the appropriate load required to produce a pre-determined moment (e.g. 70% of the isometric maximal moment) and having the subject raise their torso to the horizontal position. The external load was then incrementally increased until the subject reached the maximal load that they felt they could comfortably hold for the 10 second trial. This series of submaximal efforts produced a low (body weight) and a high moment (largest percentage) condition for analysis. The resulting low and high moment trials were used to construct muscle specific EMG-to-Force scaling factors, in conjunction with an EMG assisted lumbar spine model in order to calculate muscle forces.

#### Dynamic Flexion and Extensions

Subjects performed four repetitions of full range trunk flexion (lower) and extension (lift), with loads of 0, 5 and 10 kg. The 5 and 10 kg loads were attached to an instrumented load plate with a pair of handles. An uniaxial LVDT measured the vertical forces applied to the right handle. Forces on the left handle were assumed to be the same. Subjects utilized a self selected

style and pace, and the load originated 0.185 m in front of the great toe. They were instructed to pause at the end point of each flexion and extension.

### Isostation B-200

Subjects were restrained in the Isostation B-200 (Isotechnologies, Inc.), in an up-right, neutral standing posture. The subject's pelvis was restrained firmly, the thoracic pack was adjusted to the level of the 12<sup>th</sup> thoracic vertebra and the thigh strap was securely fastened. A standard clinical protocol, the Occupational Orthopaedic Center (OOC) protocol (Version 3.1), was then performed. Two repetitions of range of motion (ROM) were performed for right (R) and left (L) rotation (ROT), flexion (FLEX) and extension (EXT) and right and left lateral flexion (LF). Two repetitions of maximum isometric effort were then performed for RROT, LROT, RLF, LLF, FLEX and EXT. Five repetitions of rotation were then performed against a resistance which was 25% of the isometric rotation maximum. The load was then increased to 50% of the isometric maximum and five more repetitions were performed. Five repetitions of 25% MVC and then 50% MVC loading were then performed in the FLEX/EXT axis. Due to strength differences in producing flexion and extension moments, the protocol selected the smaller of the flexion and extension isometric values, to ensure that the weaker muscle group would be able to complete the task. Five repetitions of 25% MVC and then 50% MVC resistance were then performed in the LF axis. During dynamic testing the resistance in the non-movement axes (e.g. FLEX/EXT & LF during ROT) were set to the machine maximum. A second, 2 repetition ROM test was then performed for each axis, followed by a second series of dynamic testing for each axis. Although the order of testing the axes was identical to the first



dynamic sequence, the loading was reversed so that the 50% MVC load was followed by the 25% MVC load. Consistent with the clinical protocol, subjects were not informed of the order change and they were encouraged in all trials to move as hard, as fast and as far as they could.

### Calibration

A calibration trial was collected using a 1 mV, peak-to-peak 100 Hz sine wave as a known input signal into the EMG bioamplifiers. The LVDT was calibrated in the vertical direction using a zero load and a 10.3 kg mass.

### Model Overview

The structural biomechanical model used to estimate tissue loads consisted of two parts. The first is a dynamic, three dimensional, fifteen link segment representation of the body which utilized the externally applied dynamic forces and individual anthropometrics as inputs. The reaction forces and moments were calculated about three orthopaedic axes corresponding to the L4/L5 joint using inverse dynamics and working through the hands, arms, head and trunk linkages [see McGill and Norman (1986) for a detailed two-dimensional description]. The second part, an anatomically detailed model of a three-dimensional pelvis, ribcage and intervening lumbar vertebrae, was then used to partition the three reaction forces into their tissue components. This model incorporates fifty muscle fascicles, thirty-eight of which are capable of producing a restorative moment at the L4/L5 joint, thirteen ligamentous elements spanning the joint, a non-linear elastic intervertebral disc and an-equivalent torsional spring that represented the gut, skin viscera etc. Moment partitioning is accomplished by using EMG as an indicator for the neural activation level for each muscle. This neural input combined with

modulators for velocity, length and the passive elastic component produces a muscle moment, which can then be adjusted to ensure that a sufficient restorative moment is produced [see McGill (1992) for a detailed description].

The model was "tuned" for each subject by having the subject "hang from their ligaments" in a fully flexed position. Subtracting the passive elastic muscular component from the external moment allowed the ligamentous contribution to be calibrated for the angular displacements of the torso with respect to the pelvis. EMG-to-force scaling factors ( $EMG_{Force}$ ) for each electrode site, for each subject, were obtained from a set of submaximal, isometric flexion and extension contractions. These factors, combined with each muscle fascicle's physiologic cross sectional area and an assumed force per cross sectional area of 35 N/cm<sup>2</sup>, allowed each muscle fascicle's force ( $F_m(t)$ ) to be calculated (Equation 7). A common gain factor ( $G(t)$ ) was obtained by dividing the external reaction moment ( $M_e$ ) by the sum of the muscle moments. Multiplication of the muscle forces by the gain factor amplified or attenuated the muscle forces, so that the summation of all of the tissue moments equaled the external moment, thereby preserving the *relative* contribution of the muscular components to the muscle moments.

$$F_m(t) = [EMG(t) * EMG_{Force} * \delta(t) * \Omega(t) + F_{pec}(t)]_m \quad (7)$$

where:

m            muscle fascicle

$\delta$             velocity factor

$\Omega$	length factor
EMG	linear envelope EMG amplitude (a/d unit)
$EMG_{Force}$	EMG-to-force scaling factor (N/a/d unit)
F	muscle force (N)
$F_{pec}$	passive elastic force (N)

### *Data Reduction*

#### Oswestry Questionnaire

Each response to the 10 questions on the Oswestry questionnaire was scored from 0 to 5. The total was then divided by 50 and multiplied by 100 to express it as a percentage. Unanswered questions were not included in the scoring and the denominator was adjusted accordingly (Fairbank et al., 1980).

#### Pain Scale

Each 10 cm VAS was divided into 20 sections and numbered from 1 (No Pain) to 20 (Worst Imaginable) (Scott and Huskinsson, 1976). The scoring value for each 0.5 cm section was equal to each section's respective number. The scale was scored by recording the "level of pain" indicated.

#### Isometric and Dynamic Flexion and Extension Trials

The LVDT and EMG signals were A/D converted (AT-MIO-16, 12 bit ADC, National Instrument, Inc.) at 1024 Hz and stored on magnetic-optical disk. The EMG signals were full

wave rectified and low pass filtered (2nd order, single-pass, Butterworth) at a cutoff frequency of 2.5 Hz to produce a linear-envelope (LE). A 2.5 Hz cutoff frequency was selected because it reaches a peak response to an impulse in 63 msec, which is in the middle of the 30 - 90 msec twitch response to peak tension found by Buchthal and Schmalbruch (1970). Olney and Winter (1985) found cutoff frequencies to range from 1.8 to 2.8 Hz for the rectus femoris. Potvin (1992) found 2.7 Hz to be the best frequency for the lower and upper erector spinae musculature. The LE EMG and LVDT signals were interpolated, and the 3Space signals extrapolated to 30 Hz, in order to match the video sampling frequency.

The isometric and dynamic flexion-extension trials were video taped (Panasonic AG-180 UR) in the sagittal plane and the joint coordinates digitized (Peak5, version 5.2, Peak Performance Technologies, Inc.) at 30 Hz to form a link segment representation of the body. Right and left symmetry was assumed. The Z coordinate for each marker was assigned a positive or negative offset from midline equal to  $\frac{1}{2}$  of the shoulder width, as calculated from the subject's height (Winter, 1990).

The joint coordinate data, combined with the dynamic hand forces were input into a linked segment model (3DYNLNK), producing the reaction forces and moments for the L4/L5 joint. Muscle and ligament lengths were determined via the kinematic portion of the model and the lumbar spine position information (3Space). For the dynamic flexion and extension trials, the kinetic portion of the model calculated the passive tissue moments (ligament and disc) and then partitioned the remaining moment amongst the muscles, using muscle specific  $EMG_{Force}$  scaling factors.

### Isostation B-200

For each trial of the OOC protocol, the position, velocity and moment data for the ROT, F/E, and LF axes were A/D converted (Labtender, 8 bits, Scientific Solutions) at 50 Hz and stored on hard disk. The OOC software utilized a set of decision rules to analyze ROM, peak isometric moment, peak velocity, average velocity and peak secondary axes moments to produce a report that indicated both the severity of the subject's back dysfunction (i.e., none, mild, moderate, or severe) and the quality of effort (physiological, non-physiological) put forth during the test (Deutsch, 1991).

### *Data Analysis*

#### Oswestry Questionnaire

The Oswestry low back pain disability questionnaire scores for the ASYMP and SYMP individuals were analyzed in a 2 X 2 (Day X Group) repeated measures ANOVA.

#### Pain Scale

The VAS data for the ASYMP and SYMP subjects were analyzed in a 2 X 2 X 5 (Day X Group X Test) repeated measures ANOVA.

#### Dynamic Trials

To control for the effects of task initiation and termination in the dynamic trials, the second and third flexion (lowers) and extension (lifts) cycles were used. These segments of the task were termed Lift A, Lower A, Lift B, Lower B, respectively. A shoulder marker velocity of

zero was used to determine the onset and termination times for each segment. For comparison purposes, the output variables associated with the dynamic trials, video, EMG, 3Space and hand force, were normalized (interpolated or extrapolated as required) in order to produce fifty data points for each segment of the dynamic test.

### Electromyography Profiles

#### EMG - units of "mV"

The time normalized EMG data (A/D units) for each muscle were converted to mV using each channels scaling factor obtained from the 1 mV calibration trial. A normal profile of the ASYMP individuals response to each movement and load was produced by ensemble averaging the ASYMP responses at each point in time for each segment, for each muscle. Summary figures of the mean ( $\pm 1$  SD) response for each of the 12 muscles, for each lift and lower, were then produced for each day and load combination (e.g. Day 1, Load 0 kg). Each single page figure then served as a comparison template for the SYMP group.

#### EMG - units of "% MVC"

The time normalized EMG data (A/D units) for each muscle were normalized to the maximum value obtained for that muscle during the MVC trials. The data for each muscle, day and load were then ensemble averaged as described in the previous section. This produced six, single page figures of the ASYMP individuals which served as comparison templates for the SYMP group.

### EMG - units of moment (N·m)

For each of the isometric extension position loads, the link segment model calculated the L4/L5 reaction moment. For the right (R) and left (L) upper erector spinae (UES) and lower erector spinae (LES) electrode sites the average level of EMG activity associated with each load was calculated for the isometric portion of the trial. This allowed the construction of an EMG to moment scaling factor for the RUES, LUES, RLES and LLES as well as the average response of the R + L UES (i.e., mathematical average  $([RUES(t) + LUES(t)] / 2)$  and R + L LES. The extensor EMG signals produced during the dynamic flexion and extension trials were then converted by the scaling factors into a representation of the L4/L5 reaction moment. The RMS differences between the link segment model L4/L5 moment and the six L4/L5 moment time histories produced using the EMG were calculated for each of the electrode sites for each of the subjects. The EMG based moment time history with the smallest RMS difference compared to the link segment model was then used as a representation of the L4/L5 moment. The EMG moment time histories for the ASYMP group were then ensemble averaged for each day, load, lift and lower condition. This produced a single page profile of the ASYMP group which served as a comparison profile for the SYMP individuals.

### Lumbar Spine Model - Muscle Forces

Each electrode site provided an EMG signal which was used to estimate muscle fascicle forces. For each electrode, their respective individual muscle fascicle forces were summed together for each point in time, producing cumulative muscle force time histories for each of the twelve electrode sites. The cumulative muscle forces for the ASYMP individuals were then

ensemble averaged producing summary muscle force profiles (mean ( $\pm 1$  SD)) for each electrode site, for each day, load and lift/lower condition. This produced a single page for each day and load combination, which were used as comparison templates for the SYMP group. The upper boundary (mean + 1 SD) for each of the ensemble averaged muscle force profiles was also used to create an Amplitude Probability Distribution Function (APDF) for each electrode site. The APDF describes the distribution of different levels of muscle force during the period over which the activity was recorded. Each point on the distribution function curve shows the probability of the muscle force being lower than or equal to the actual muscle force level (Jonsson, 1978). Plotting electrode specific APDFs for a SYMP individual's muscle force and the ASYMP upper boundary (mean +1 SD) on the same graph illustrates the if excessive muscle force has been produced. Excessive muscle force is illustrated when the APDF curve for an ASYMP individual lay to the right of the APDF criterion curve (mean + 1 SD) (e.g. Figure 38). This produced a comparison template for the symptomatic individuals muscle force data which facilitated the quantification of the amount of time that a muscle force was above the ASYMP criterion (mean + 1 SD). To keep muscle forces within a biologically valid range, the gain factor (G), was not allowed to get larger than 3.5. Trials for subjects in which this occurred, were not included in the data analysis and are summarized in Table 8.



**Table 8** The number of trials excluded due to gain factoring exceeding 3.5.

	Load (kg)	Lift A	Lower A	Lift B	Lower B
Day 1	0	2	1	2	0
	5	4	0	4	0
	10	5	1	4	0
Day 2	0	3	1	3	0
	5	3	1	4	0
	10	4	2	6	0

#### Lumbar Spine Model - Compression

The model estimates of the L4/L5 compression time history were time normalized for each subject's lifts and lowers. Ensemble averages of the ASYMP participants were produced for each load, day, lift/lower and displayed on a single page. These were then used as comparison templates for the SYMP group.

#### Isostation B-200

The OOC clinical report variables of "Abnormal Indicators" and "Non-Physiological Indicators" were tested by a 2 X 2 (Day X Group) repeated measures ANOVA.

To analyze the primary axis velocity as well as the secondary and tertiary axes moments for a specific movement, a custom data analysis system was developed. The dynamic trials for the 25% and 50% rotation, flexion-extension and lateral flexion trials were converted to ASCII

files so that a "normal" performance profile could be produced. For each dynamic trial, the start and end times for each movement (e.g. right rotation, left rotation), within each repetition were determined from the primary axis velocity and moment data. To accommodate for task initiation and termination, the first and fifth repetitions for each test were discarded. For the middle three repetitions, the primary axis moment, velocity and position data, as well as the secondary and tertiary moment data, were then normalized with respect to time, so that each half repetition (e.g. right rotation) consisted of 25 data points. The position data were then normalized to the maximum range of motion value obtained in each direction for that particular resistance. The secondary and tertiary moment data was normalized to the maximum isometric value obtained for each specific axis and direction of movement (e.g. lateral flexion left). The three repetitions were then averaged together for each direction of movement, producing an average profile, for each variable, for each of the asymptomatic individuals. An ensemble average of the asymptomatic individuals' average profiles was then constructed for primary axis velocity, primary axis position, secondary axis moment and tertiary axis moment for each of the 25% and 50% tests. A profile page of these five variables was then created for each of the eight dynamic tests sequences performed.

## **Results**

### Overview

The purpose of this study was to determine if the understanding of spine movement dysfunction was augmented by the knowledge of EMG activity and torso tissue forces. In this study the understanding of spine movement dysfunction is augmented beyond the information

provided by the kinematic and moment data from the Isostation B-200 or from the free lifts and lowers if:

a) a symptomatic individual is not identified by the clinical Isostation B-200 report or the free lifts on the Good or Bad days but emerges using the EMG, Isostation B-200 and/or muscle force profiles,

and/or,

b) a profile for a particular EMG electrode site or muscle force is located outside the normal range (mean  $\pm$  1 SD).

To facilitate the presentation of the Oswestry Low Back Disability Questionnaire, the Pain Scale and the Isostation B-200 OOC results, the ASYMP and SYMP groups are presented together. This is followed by the custom profiles developed from the ASYMP individuals for the B-200, EMG and model outputs. The use of each of the normal profiles as comparison templates is illustrated by using data from symptomatic Case #3. The data for each of the symptomatic individuals is then presented using a case study format.

In this study, the symptomatic participants identified a "Good" and a "Bad" day with respect to their back function. For inclusion in the study it was not necessary to have the Bad day occur first. For the purposes of statistical analysis, the Good day for the symptomatic individuals was assumed to have occurred on Day 1. This required an adjustment of test order for two of the SYMP group members. However, for the profile analysis, the asymptomatic data were plotted against the appropriate ASYMP profile for that test day.

### Oswestry Low Back Disability Questionnaire

The Oswestry disability questionnaire data were analyzed using a 2 X 2 (Day X Group), repeated measures analysis of variance (ANOVA). As illustrated in Figure 10 the individuals asymptomatic for low back pain reported less low back disability than the symptomatic people with a significant Group main effect,  $F(1, 12) = 22.28, p < .0005$ . The mean scores for both groups would be classified as Minimal Disability (Fairbank et al., 1980). The Day of testing main effect and the Day X Group interaction were both non-significant,  $F(1, 12) = .72, p < .412$  and  $F(1, 12) = 2.16, p < .168$ , respectively.

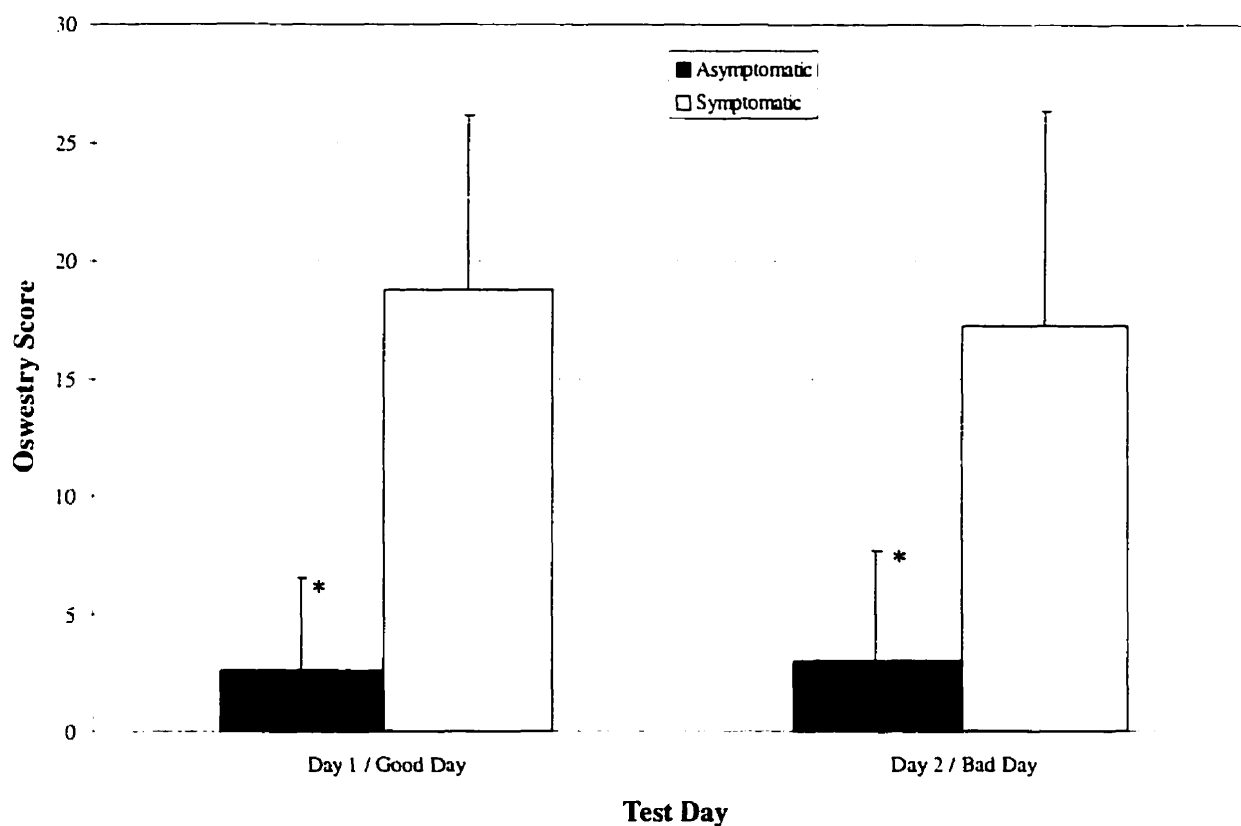


Figure 11: The mean (+ 1 SD) Oswestry pain scale scores (note: maximum score = 100). The \* indicates that the mean score for asymptomatic subjects was significantly lower than that for symptomatic subjects ( $F = 22.28, p < .0005$ ). There was not a significant difference between days ( $F = .72, p < .412$ ).

### Pain Scale

The 10 cm pain scale measures were analyzed using a 2 X 2 X 5 (Day X Group X Test) repeated measures ANOVA. The ASYMP individuals indicated significantly lower levels of pain than the SYMP participants, as the Group univariate main effect was significant,  $F(1, 12) = 23.15, p < .0005$  (see Figure 11). The univariate Day and multivariate Test main effects were non-significant,  $F(1, 12) = .42, p < .530$  and  $F(4, 9) = 3.37, p < .06$ , respectively. All of the interaction effects were non-significant.

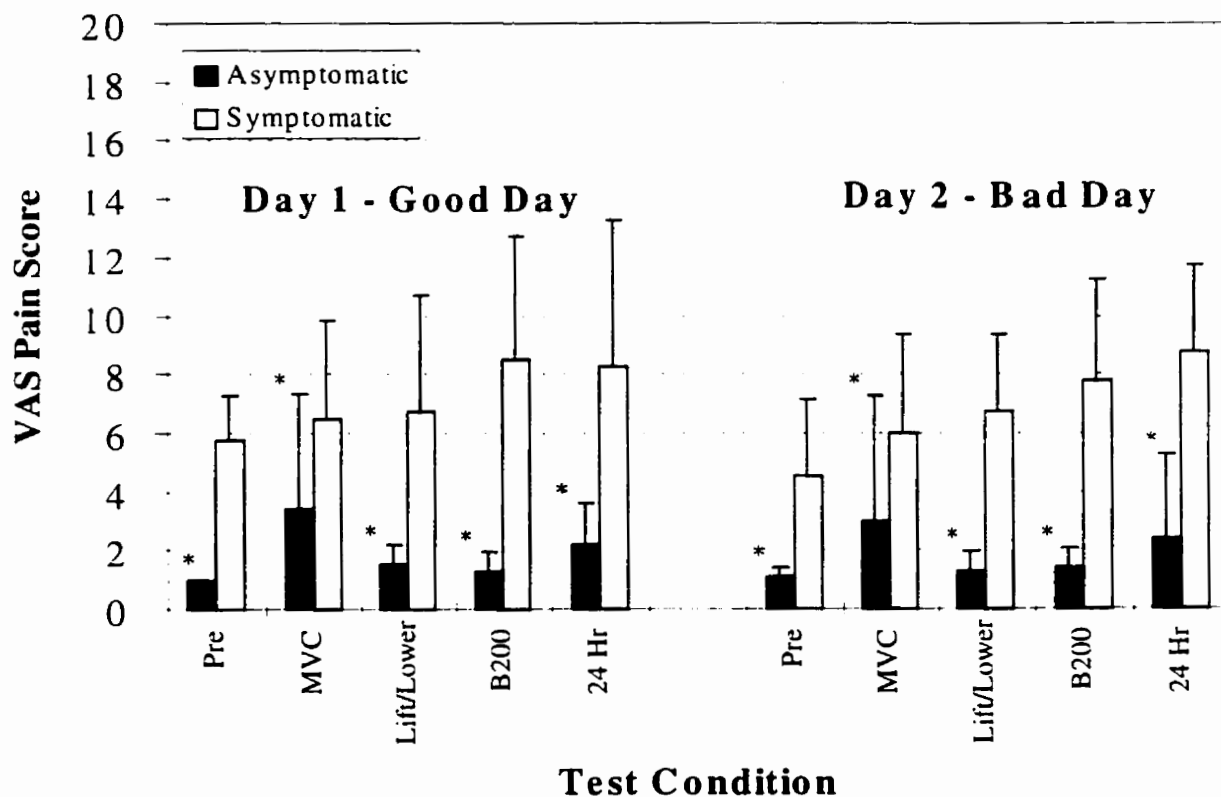


Figure 12: The mean (+ 1 SD) Visual Analog Scale pain scores (note: maximum score = 20) for both groups and days, following each specific test. The \* indicates that the asymptomatic individuals indicated significantly lower levels of pain ( $F = 23.15, p < .0005$ ) than the symptomatic people. There were no significant differences between days ( $F = .42, p < .530$ ) or between test conditions ( $F = 3.37, p < .06$ ).

Isostation B-200 - OOC

The number of abnormal and non-physiological indicators used to prepare the B-200 OOC clinical reports are summarized in Table 9. Each variable was analyzed using a 2 X 2 (Day X Group), repeated measures ANOVA. For the abnormal indicators there were no significant main effect for Day or Group, and no interaction effect for Day X Group,  $F(1, 12) = 1.86, p < .198$ ,  $F(1, 12) = 2.48, p < .141$ ,  $F(1, 12) = 3.54, p < .084$ , respectively. For the non-physiological indicators there were no main effects for Day or Group, and no interaction effect for Day X Group,  $F(1, 12) = 1.06, p < .323$ ,  $F(1, 12) = .74, p < .405$ ,  $F(1, 12) = 1.81, p < .203$ , respectively. Interestingly, 3 of the 4 cases had normal function on both their "Good" and "Bad" days.

**Table 9** A summary of the B-200 OOC protocol test results. No significant differences were found between groups or days for the abnormal indicators, the non-physiological indicators or the amount of back dysfunction.

<b>B-200 Indicator</b>	<b>Day 1 - Good Day</b>			<b>Day 2 - Bad Day</b>		
	<b>Abnormal</b>	<b>Non- physiological</b>	<b>Amount of Dysfunction</b>	<b>Abnormal</b>	<b>Non- physiological</b>	<b>Amount of Dysfunction</b>
<b>Back Pain</b>						
<b>ASYMP</b>	1	2	mild	0	0	none
	0	1	none	0	0	none
	0	0	none	0	1	none
	0	1	none	0	0	none
	0	1	none	0	0	none
	1	0	mild	0	0	none
	0	0	none	0	1	none
	0	1	none	0	1	none
	0	0	none	0	1	none
	0	0	none	0	1	none
<b>SYMP (Case #)</b>						
1	0	0	none	0	2	none
2	0	0	none	0	1	none
3	4	0	mild	9	0	moderate
4	0	0	none	0	0	none

### B-200 Profile Pages

The asymptomatic individuals' data from each of the two 50% resistance tests, for each day of testing, were used to produce four comparison profiles each for the rotation and lateral flexion axes. Figure 12 is the asymptomatic group profile for the first dynamic 50% rotation test for Day 1. To facilitate analysis of the profiles, each primary axis movement was divided

into four quadrants. For example a complete right and left rotation repetition was divided from maximal left rotation to neutral (Q1), neutral to maximal right rotation (Q2), maximal right rotation to neutral (Q3) and neutral to maximal left rotation (Q4). Overlaying an asymptomatic individuals test results graphically illustrates the regions where the velocity, secondary axis moments and/or tertiary axis moments deviate from the mean  $\pm 1$  SD region (Figure 13, for Case study #3). The consistency of the performance is illustrated by the coefficient of variation (CV) for the average velocity in each movement quadrant. For example, in Figure 12, for Quadrant 1 (full left rotation to the mid-range or neutral position) the asymptomatic group had a CV of 10%. Each B-200 profile page provides a graphical summary of the primary axis velocity and secondary axis torques for a particular axis and resistance. Displaying the movement data for a specific individual against the profile allows a comprehensive, qualitative visual assessment of primary axis velocity and secondary and tertiary axis moment to be made, facilitating the identification of “abnormal” regions (Figure 13).



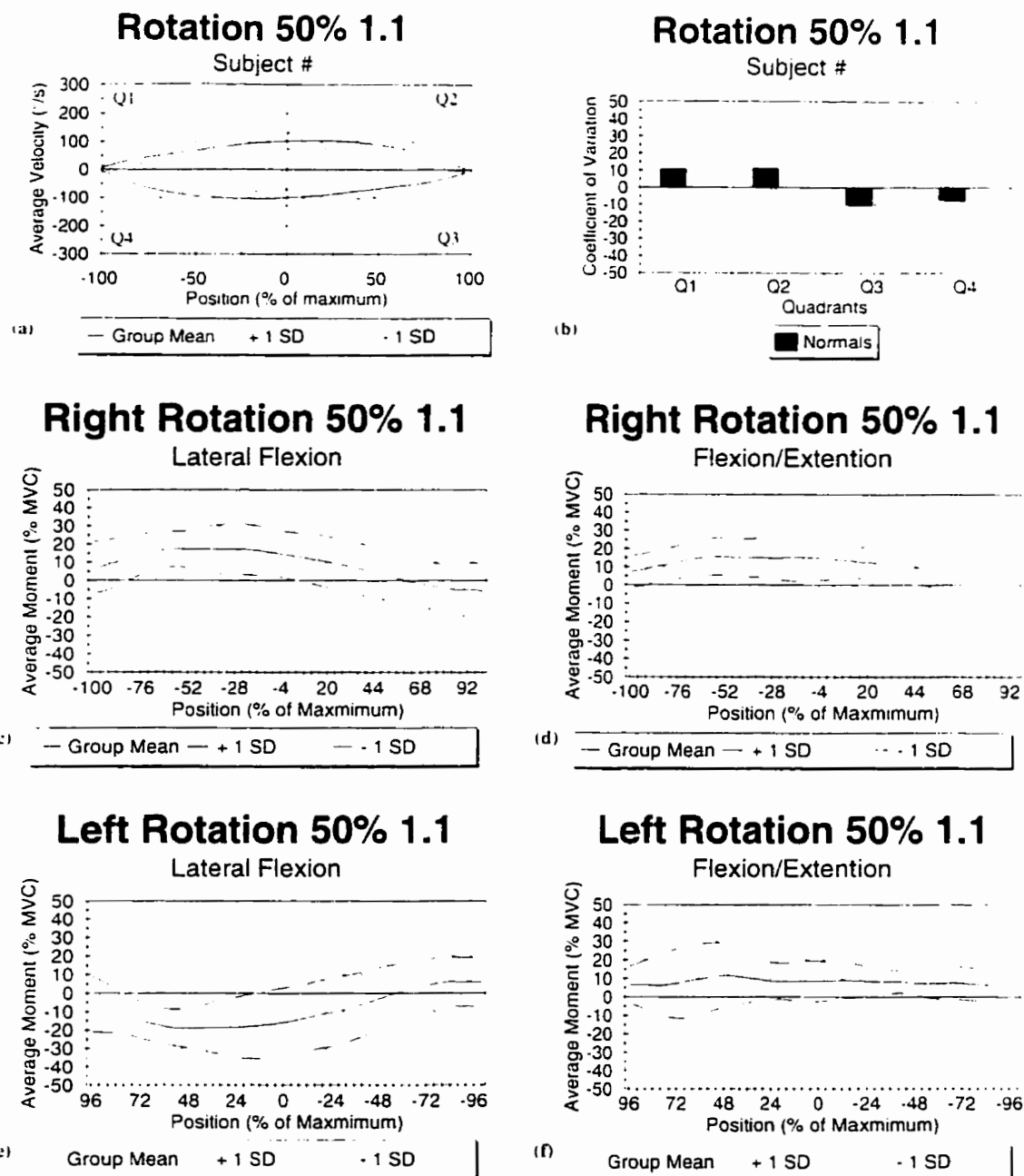


Figure 13: B-200 summary performance profile for the ASYMP group (10 subjects) for Dynamic Rotation at 50% Resistance, Day 1, Sequence 1. (a) The mean velocity ( $\pm 1$  SD) for right (+ve velocity) and left (-ve velocity) rotation. The horizontal axis is normalized from -100% of left rotation to 100% of right rotation. The rotation velocity and position axes were paired into 4 quadrants (Q1, Q2, Q3 and Q4). (b) Mean coefficient of variation for each quadrant. (c) The mean lateral flexion moment produced during right rotation. (d) The mean flexion-extension moment produced during right rotation. (e) The mean lateral flexion moment produced during left rotation. (f) The mean flexion-extension moment produced during left rotation.

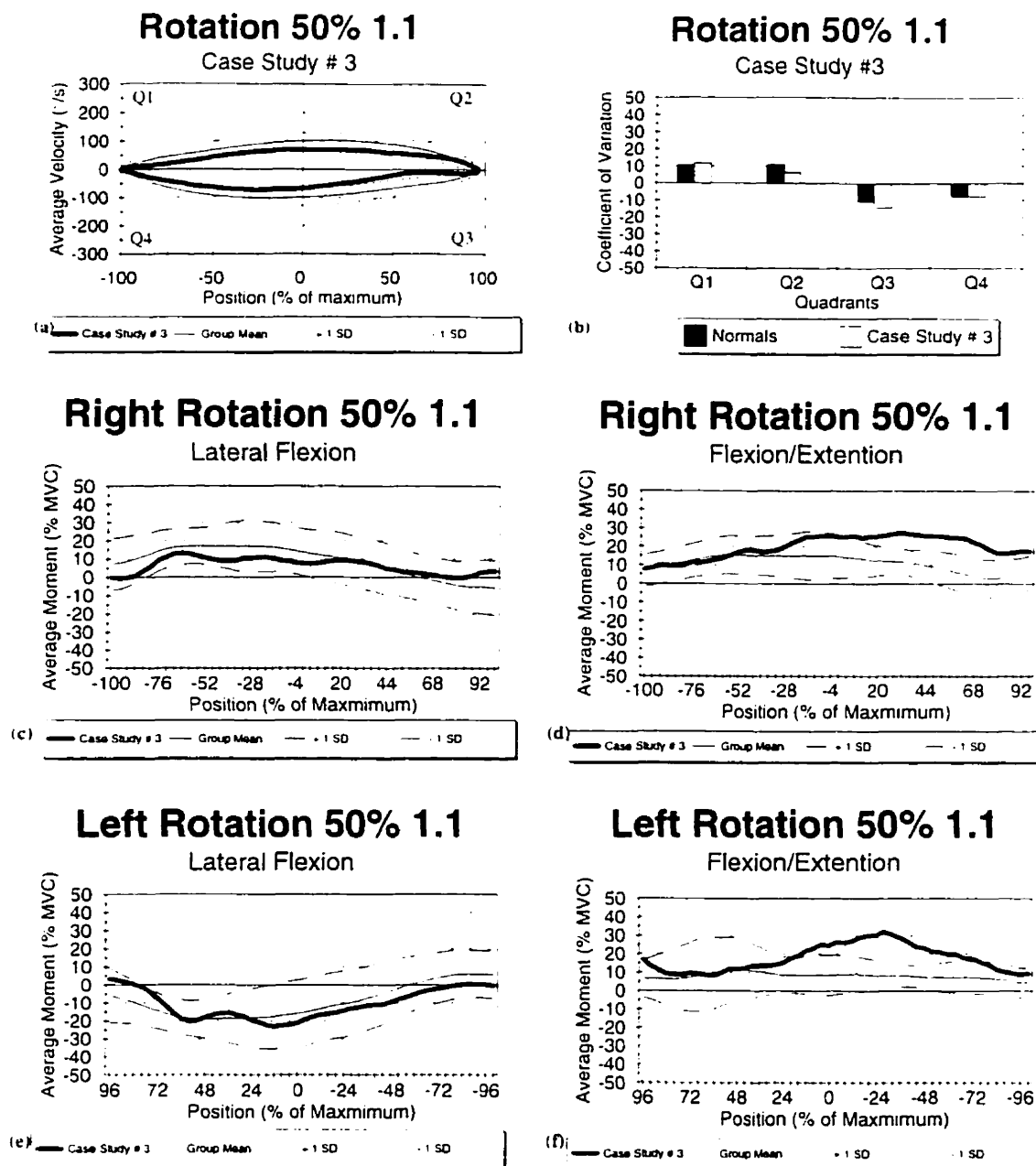


Figure 14: B-200 summary performance profile for case study #3 (Good day), for dynamic rotation at 50% resistance, day 1, test sequence #1. (a) The decreased average rotation velocity falls within the normal band for most of the test. (b) The test shows normal variability. (c) The mean lateral flexion moment during right rotation is within the normal bands for Q1 and Q2. (d) The mean flexion-extension moment during right rotation is within the normal band during the first half of the rotation (Q3), but is outside of this range for the second half (Q4). (e) The mean lateral flexion moment during left rotation is within the normal bands for Q3 and Q4. (f) The mean flexion-extension moment during left rotation is within the normal range for the first half of the movement (Q3), but is outside of this range for the second half (Q4).

To quantify the moment profiles, the secondary and tertiary axis moment time histories curves were evaluated numerically. If a symptomatic individual's data were outside the mean  $\pm 1$  SD region for more than 50 % of the quadrant, it was regarded as a positive response. An example of a case study profile is shown in Figure 13. During this 50% resistance rotation test, only two positive responses were observed. These occurred in quadrant 2 and quadrant 4 for the tertiary moment (Flexion-Extension) and this test would be scored as having two positive responses in the tertiary axis. To complete the rotation repetitions, this person required almost 30% of their flexion strength, while the asymptomatic group utilized less than 10% of their flexion strength. If an individual had excessive secondary and tertiary moments for an entire movement (e.g. increased lateral flexion and flexion-extension activity during rotation) there would be eight positive responses.

This assessment was applied to all of the OOC tests for each symptomatic participant. The dynamic 50% resistance tests for rotation and lateral flexion were found to be the most responsive tests and the summary data for Case Study #3 is shown in Table 10.

**Table 10** Summary of increased secondary and tertiary axes moment activity for OOC 50% resistance tests, dynamic sequence 1 and 2, test days 1 and 2. Excessive secondary and tertiary activity is indicated by a ✓ and ✗, respectively. For rotation tests, lateral flexion is secondary, flexion extension is tertiary. For lateral flexion tests, flexion-extension is secondary and rotation is tertiary. The shaded areas indicate the tests for the individual's "Bad" day.

Case Study # 3	Test (day - sequence #)	Movement Quadrant				Totals
		1	2	3	4	
Primary Axis Rotation	1 - 1		✗		✗	0, 2
	1 - 2					0, 0
	2 - 1	✓	✓✗	✓✗	✓✗	4, 3
	2 - 2	✓✗	✓	✓✗	✗	3, 3
Lateral Flexion	1 - 1	✓✗		✓	✓	3, 1
	1 - 2	✗	✓	✓	✓	3, 1
	2 - 1		✓	✓	✓	3, 0
	2 - 2	✗	✓	✓		2, 7
Totals	Bad Day	2, 2	4, 1	4, 2	2, 2	12, 7
	Good Day	1, 2	1, 1	2, 0	2, 1	6, 4

### Electromyography Profiles

The ensemble averaged myoelectric profiles were plotted to illustrate the amplitude and temporal components of the muscle activity patterns produced for the two extensions (lifts) and flexions (lowers).

EMG - units of "mV".

The EMG "mV" amplitude profiles (Figures ?? to ??) produced for the 0, 5 and 10 kg lifts and lowers for each test day were designed to facilitate the assessment of an individual by simply overlaying their EMG time histories. As anticipated, low levels of activation were observed for the right and left rectus abdominis, externa oblique and internal oblique, regardless of the type of activity (lift or lower).

The extensor musculature produced greater levels of activity which increased in order from the latissimus dorsi, upper erector spinae and lower erector spinae. The activity for a specific extensor electrode was always greater for the lifting phases. Increasing the load did not appear to increase the flexor activity, but did produce an increase in the extensor activity, particularly the upper and lower erector spinae. With increases in load these two electrodes showed specific patterns. The upper erector spinae peaked at the onset of the lift. The lower erector spinae produced two identifiable peaks of activity, the first located at the onset of the lift and the second during the mid-range. Similar mean activity levels and patterns were produced for Day 1 and Day 2. Figure ?? is an example of the EMG patterns for a SYMP individual (Case Study #3).

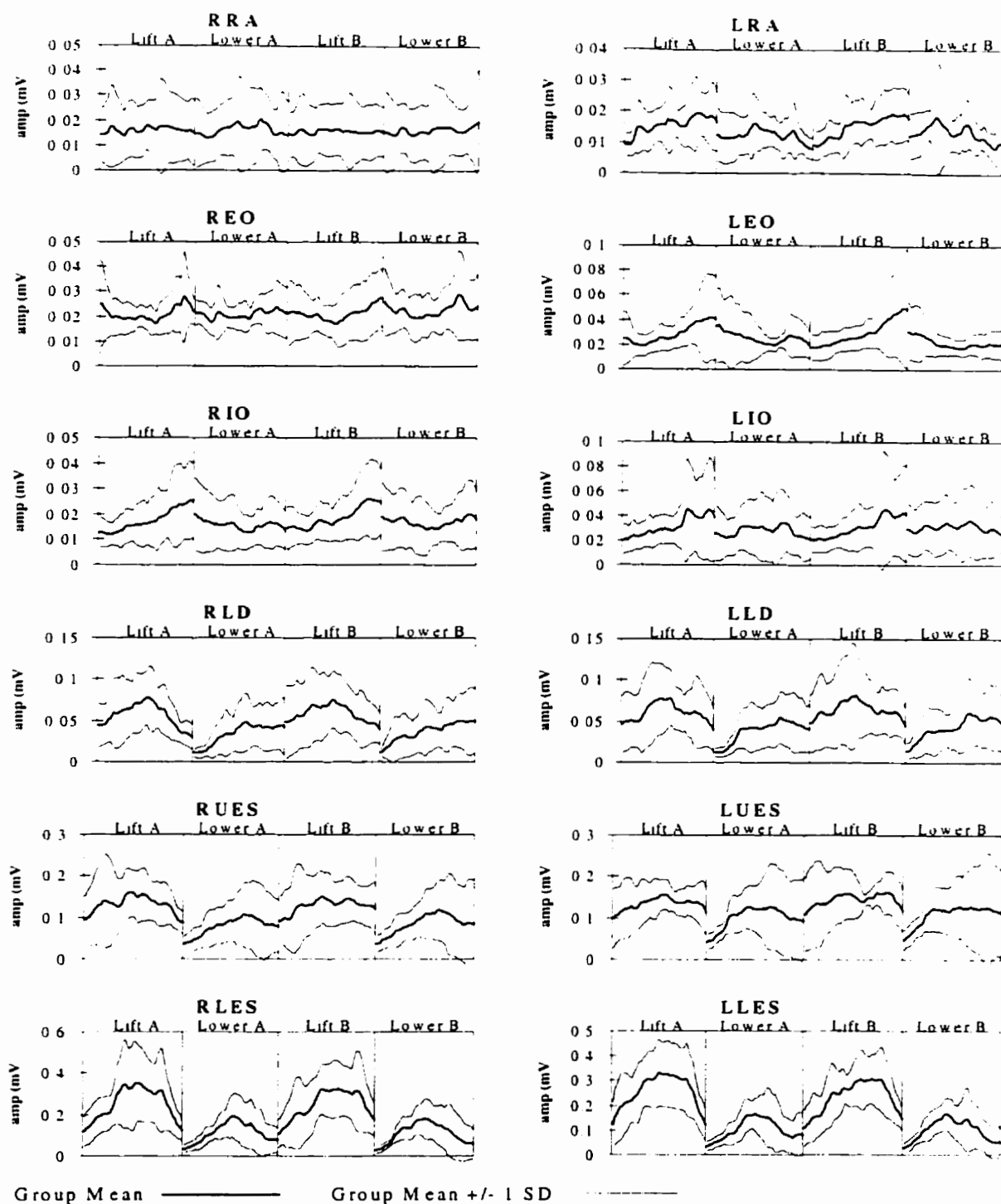


Figure 15: Summary profile of the mean ( $\pm$ 1 SD) ASYMP (9 subjects) EMG activity levels (mV) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

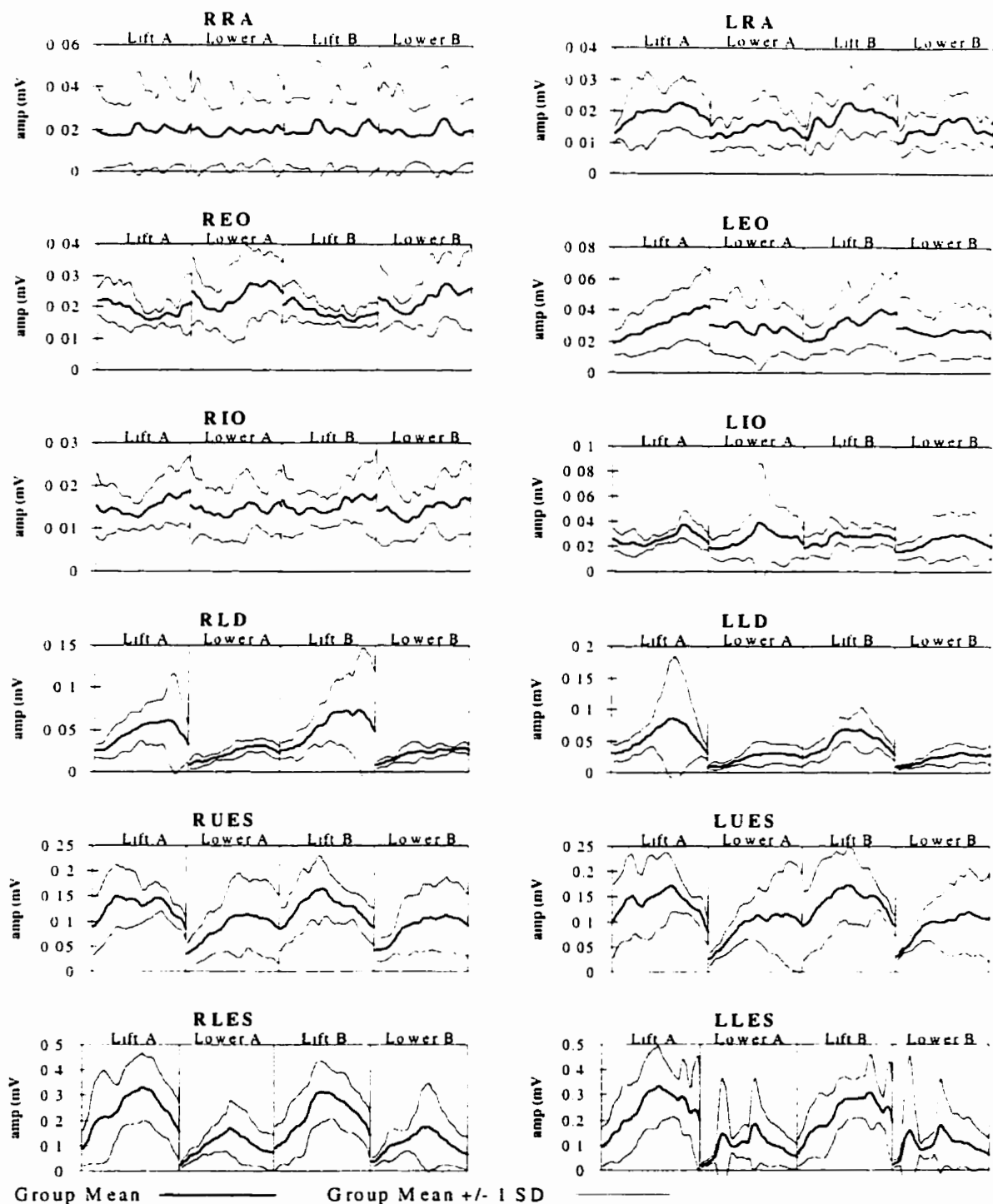


Figure 16: Summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (mV) for Day 2, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

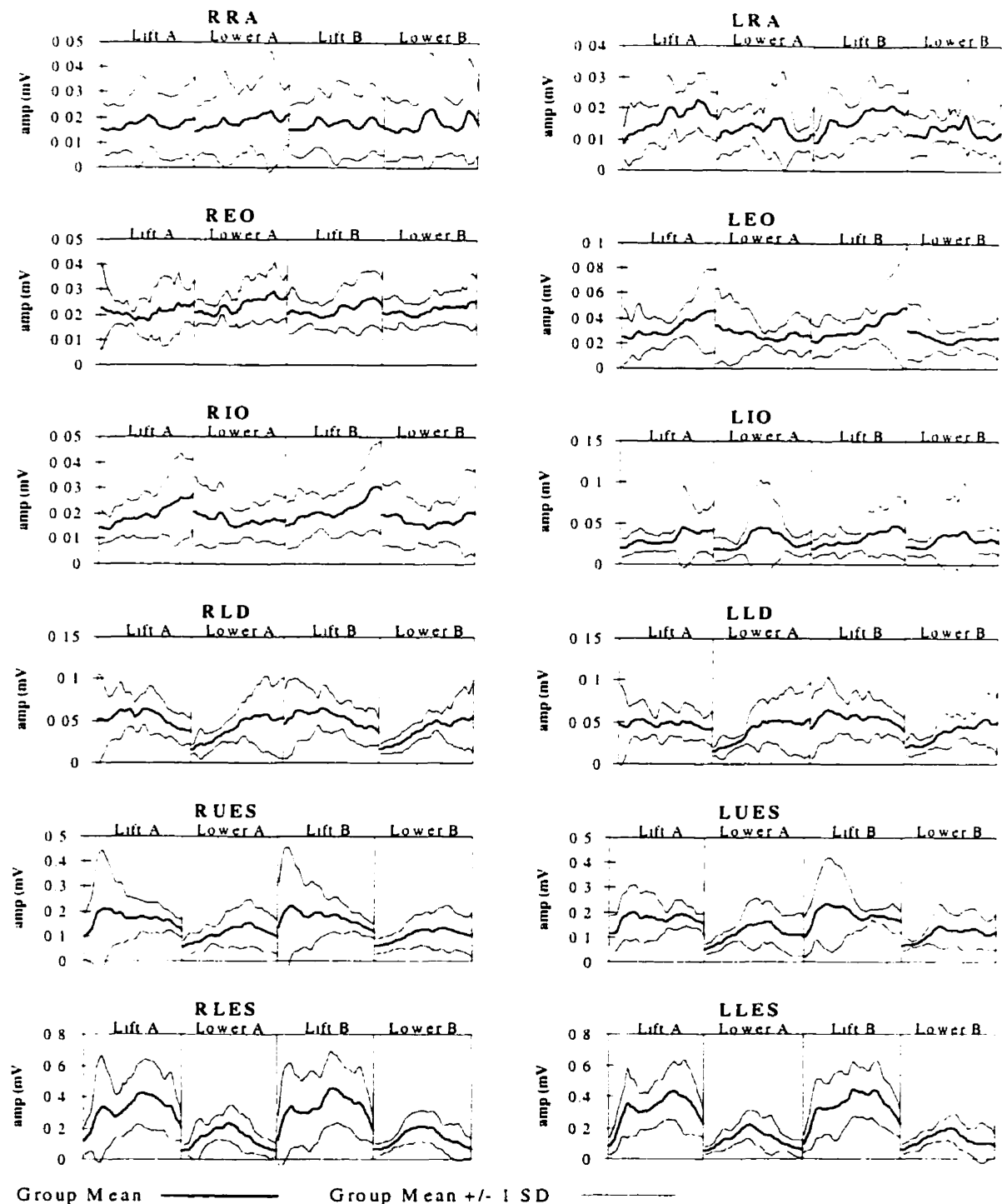


Figure 17: Summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (mV) for Day 1, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.



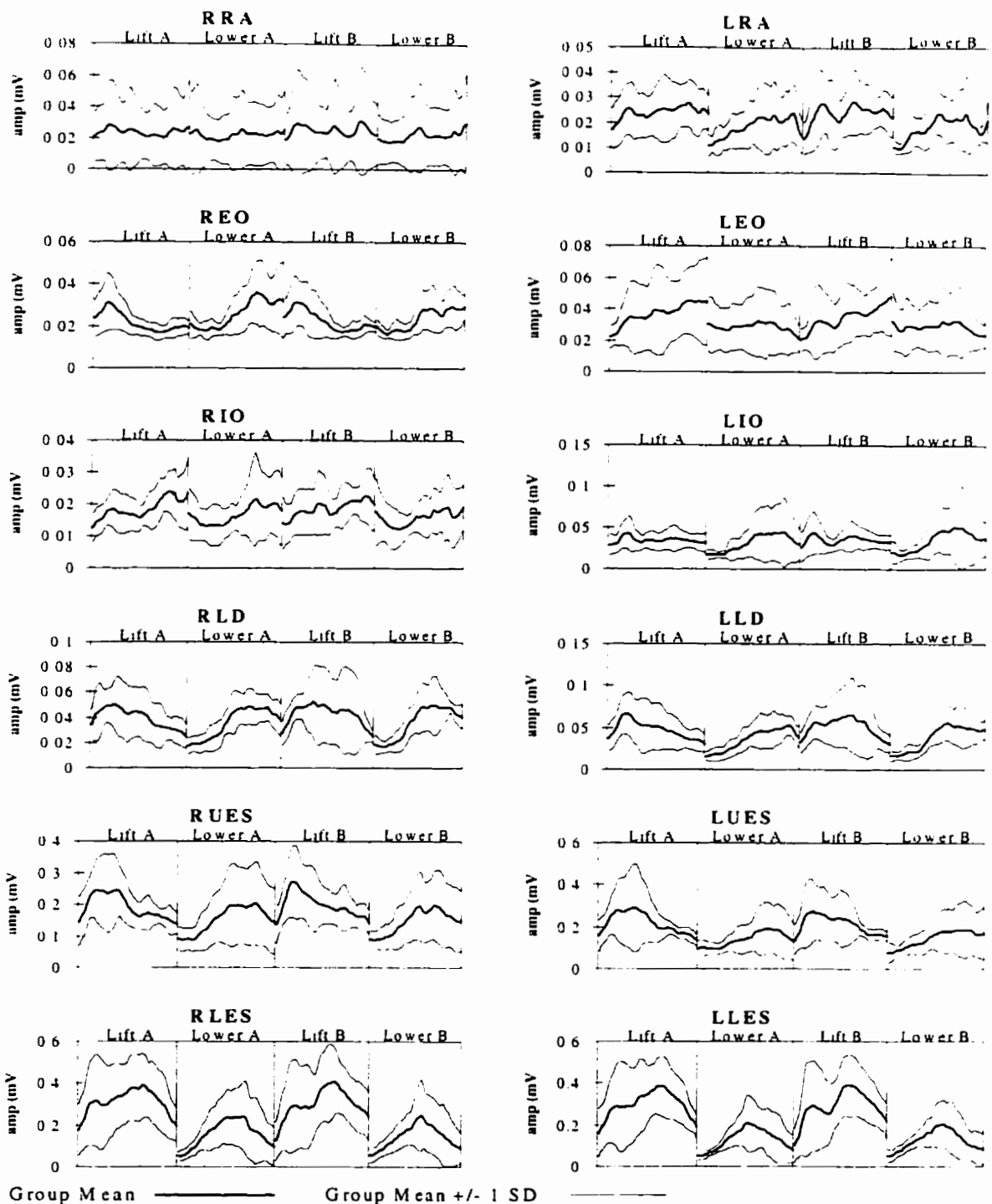


Figure 18: Summary profile of the mean ( $\pm$ 1 SD) ASYMP (10 subjects) EMG activity levels (mV) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

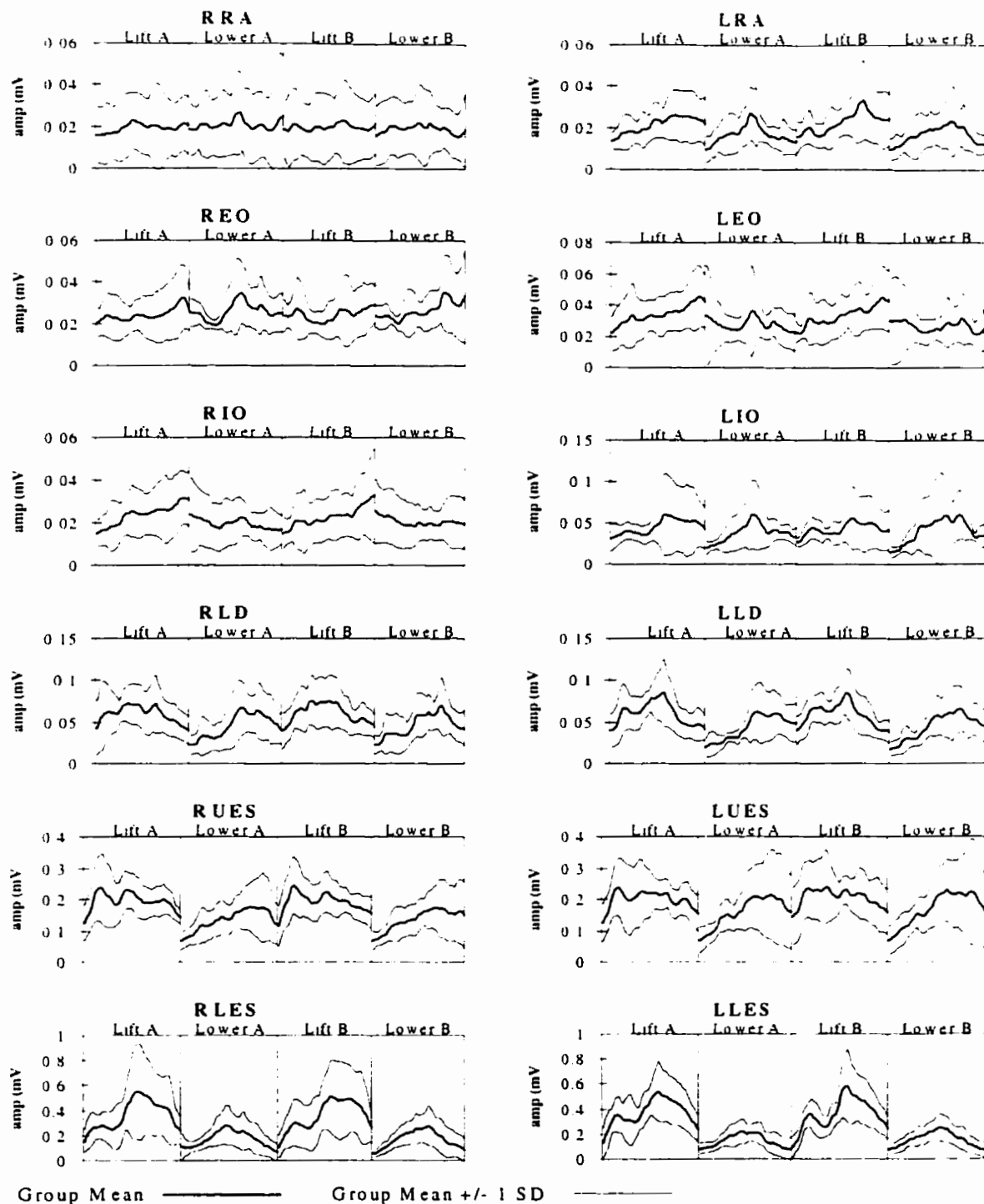


Figure 19: Summary profile of the mean ( $\pm 1$  SD) ASYMP (6 subjects) EMG activity levels (mV) for Day 1, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

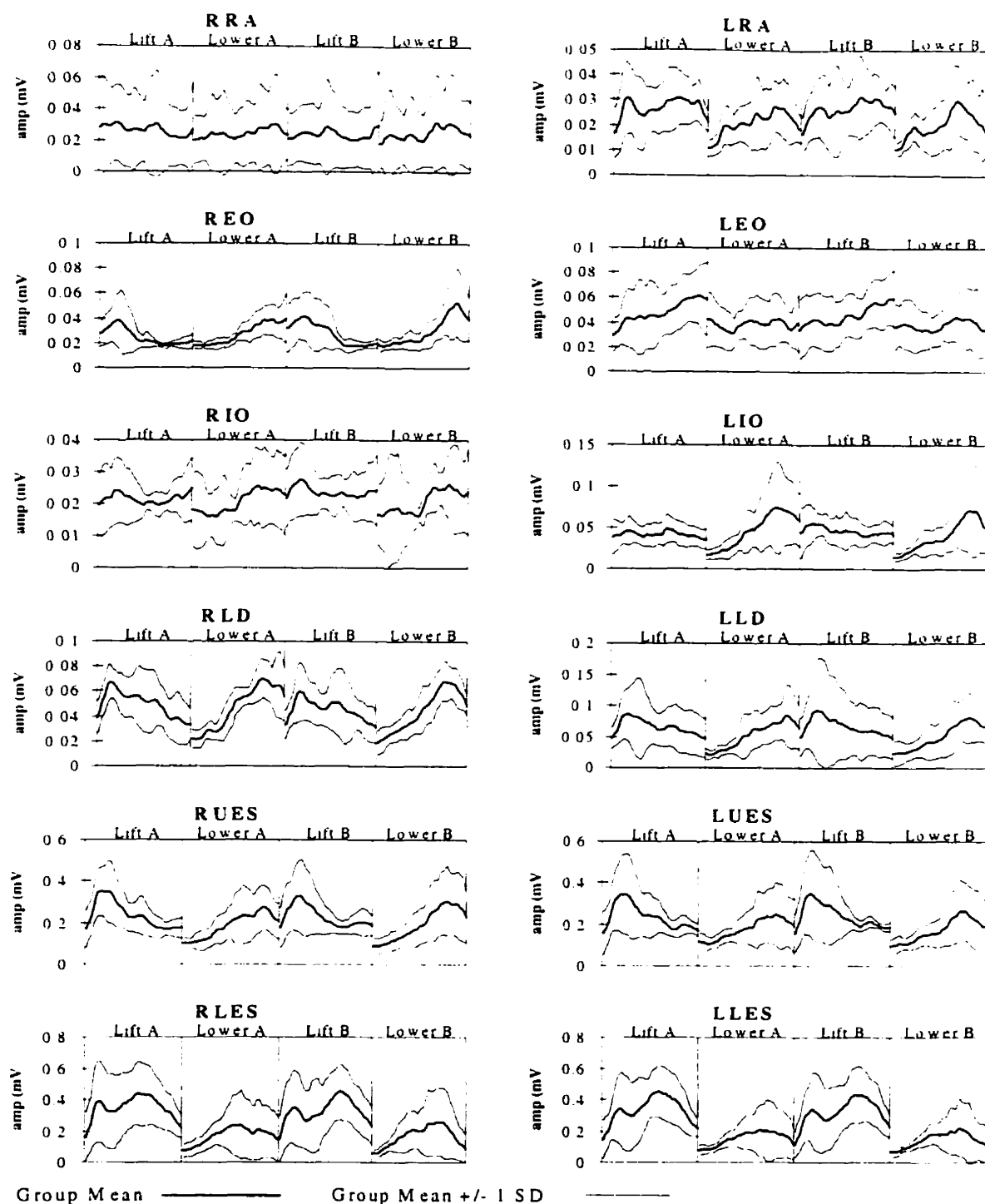


Figure 20: Summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (mV) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

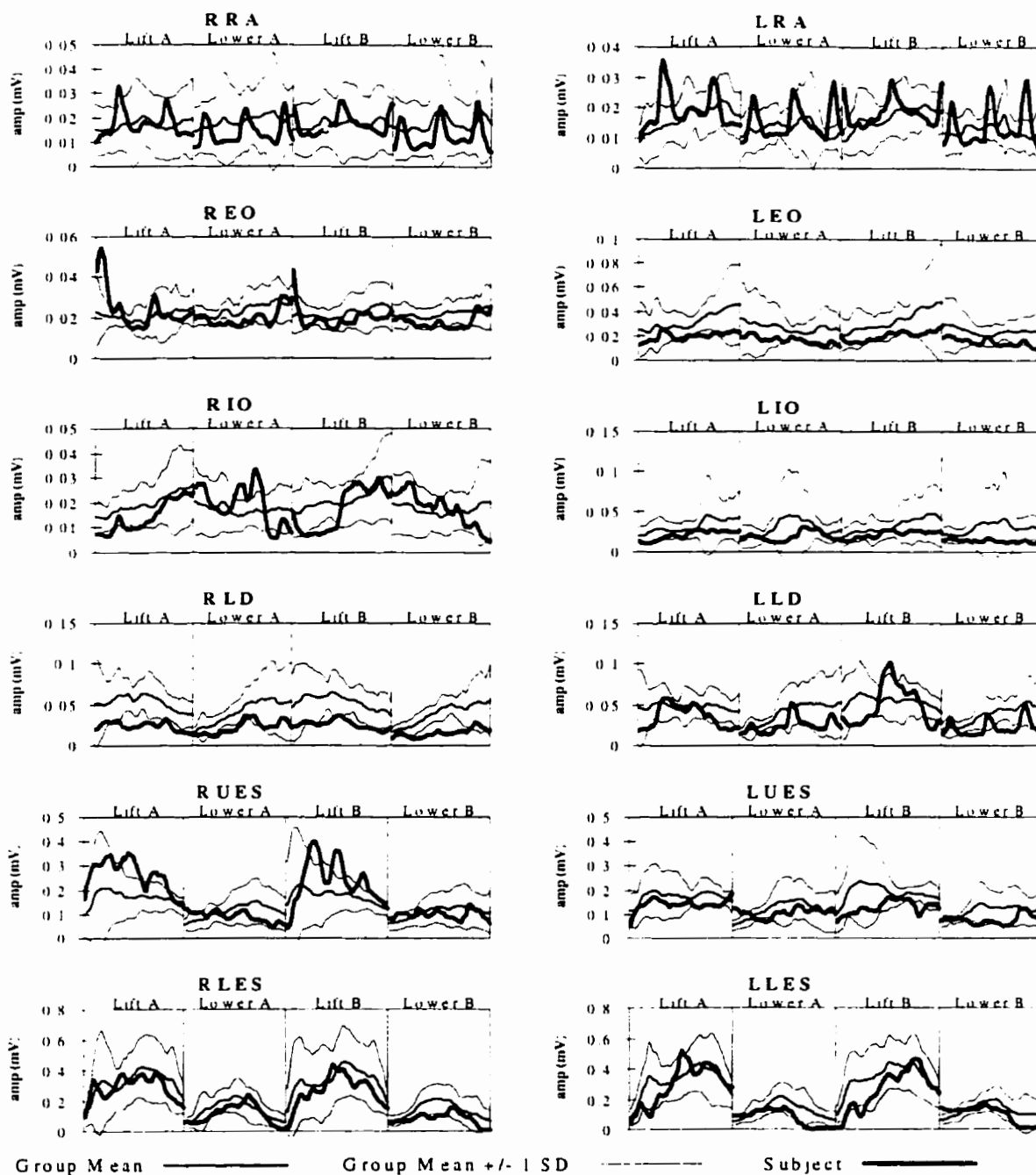


Figure 21: Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (mV) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. This figure highlights the difficulty with the profile technique. All patterns are within the normal band, yet RRA and LRA display distinct bimodal patterns not observed in group mean. Legend: R= right, L= left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

### EMG - units of "% MVC"

As anticipated, normalizing the EMG amplitude profiles to % MVC did not alter the shapes of the resulting profiles (Figures 21 to 26). For the flexor musculature, the activation levels ranged from 2 - 6 % MVC, regardless of the type of activity (lift or lower), load or day of testing.

The magnitude of activity in the extensor musculature increased from the latissimus dorsi, which had the least activity, to the upper erector spinae, to the lower erector spinae, which had the greatest amount of activity. The activity for a specific extensor electrode was always greater for the lifting phases. Increasing the load did produce an increase in the extensor activity, particularly the upper and lower erector spinae. With increases in the size of the load, the upper erector spinae showed a distinct peak at the onset of the lift. The lower erector spinae produced two identifiable peaks of activity, the first located at the onset of the lift and the second during the mid-range. Similar mean activity levels and patterns were produced for Day 1 and Day 2. Figure 27 is an example of the EMG patterns for a SYMP individual (Case Study #3).

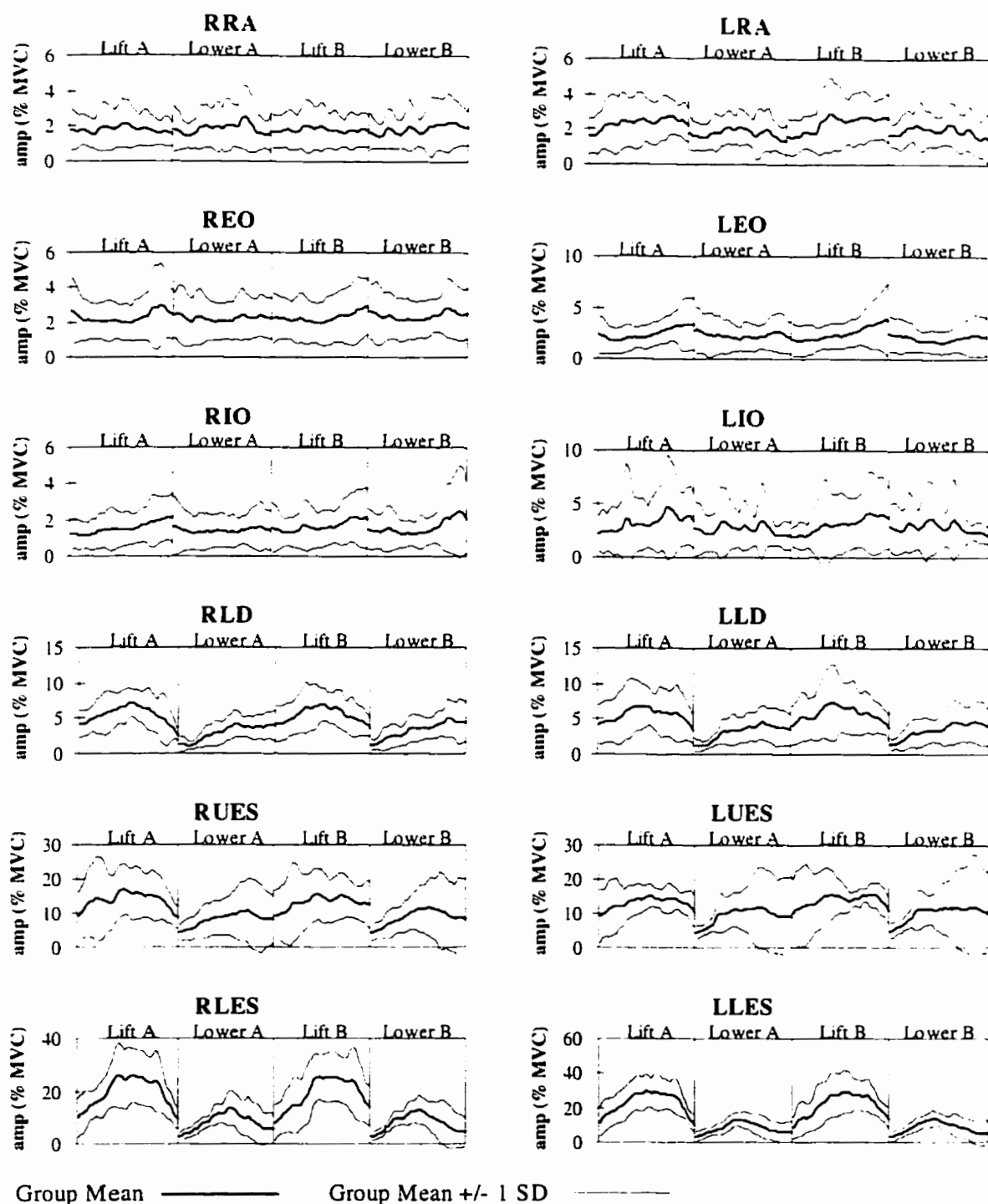


Figure 22: Summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (% MVC) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae. LES = lower erector spinae.

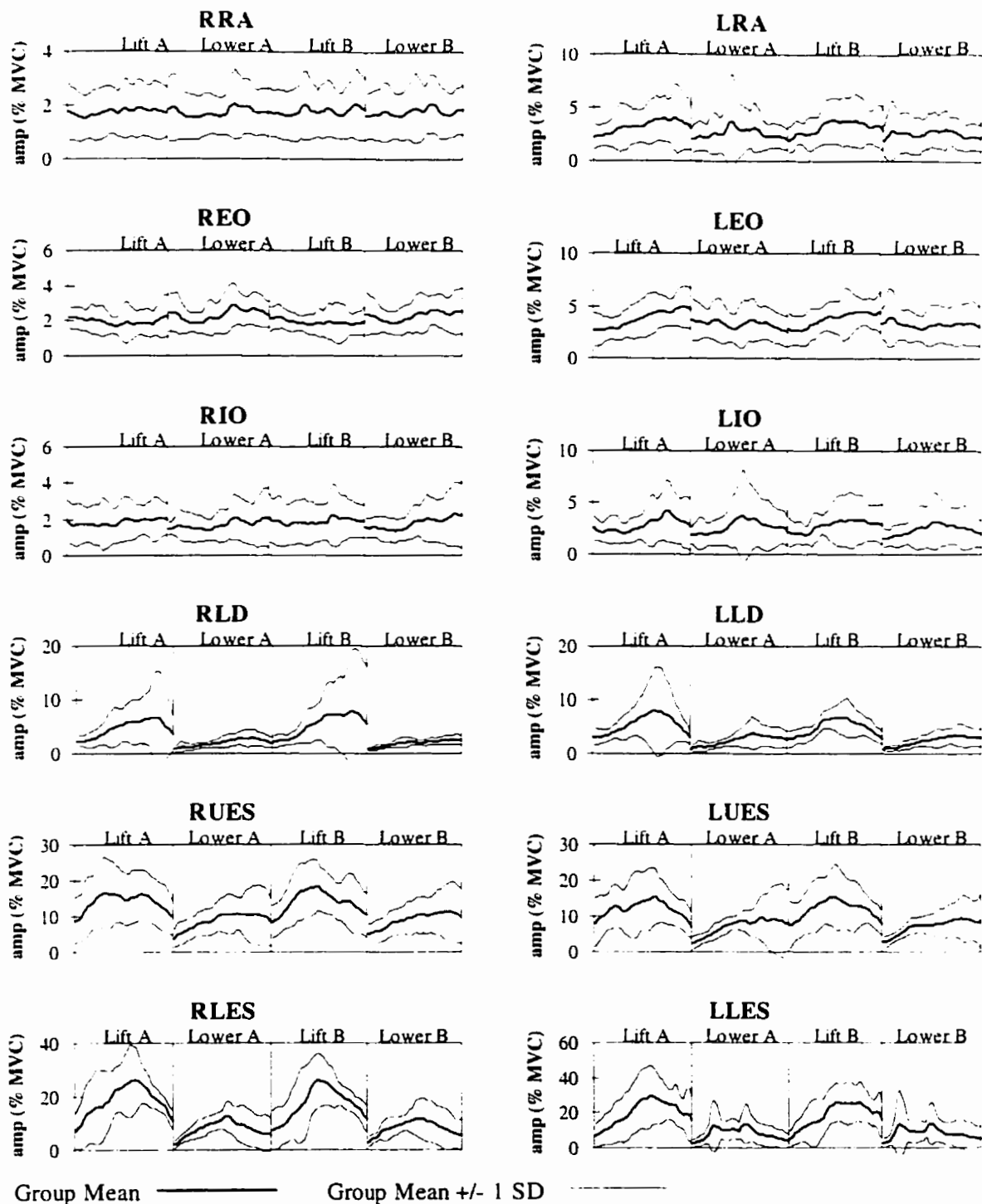


Figure 23: Summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 2, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

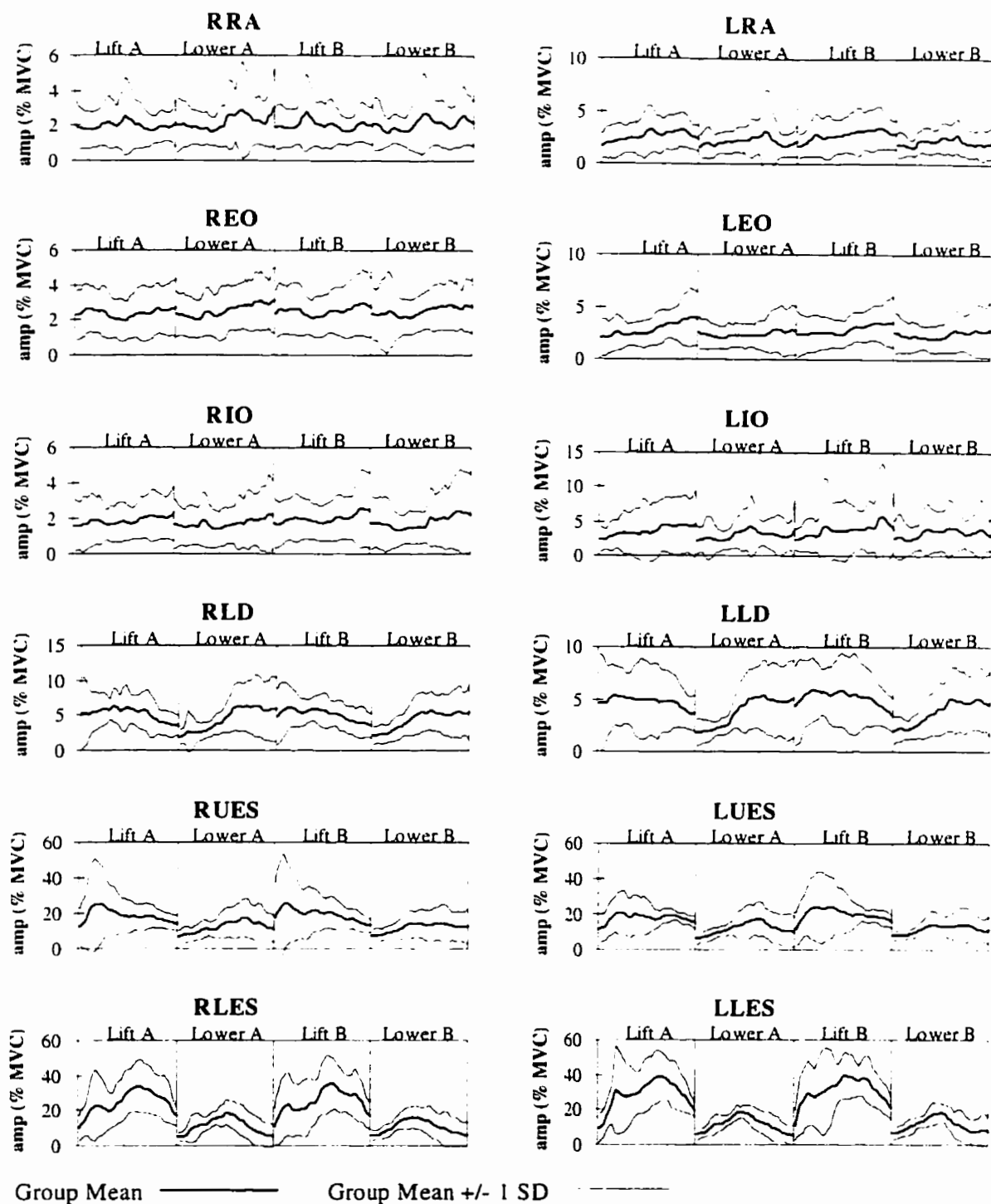


Figure 24: Summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (% MVC) for Day 1, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.



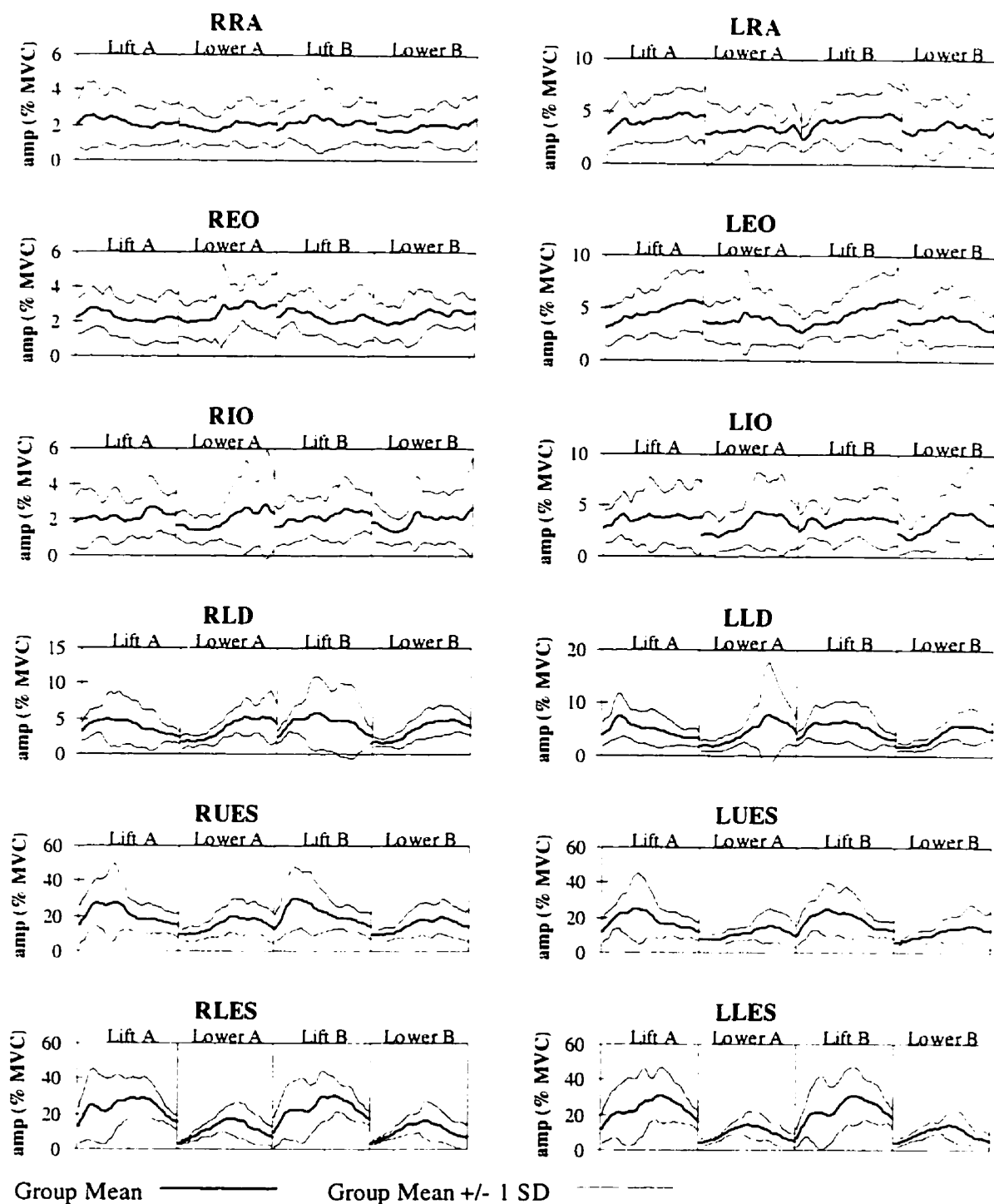


Figure 25: Summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

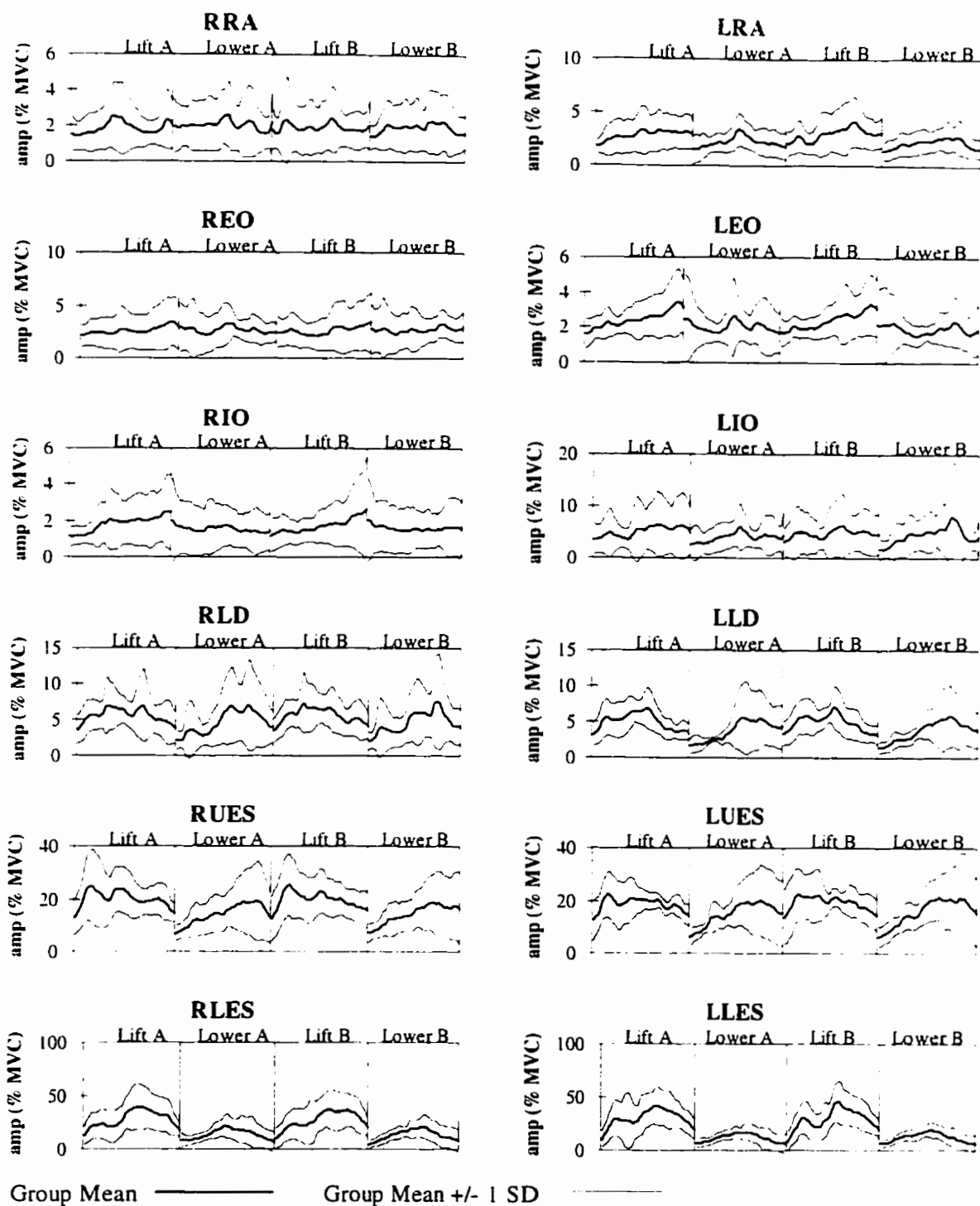


Figure 26: Summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (% MVC) for Day 1, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

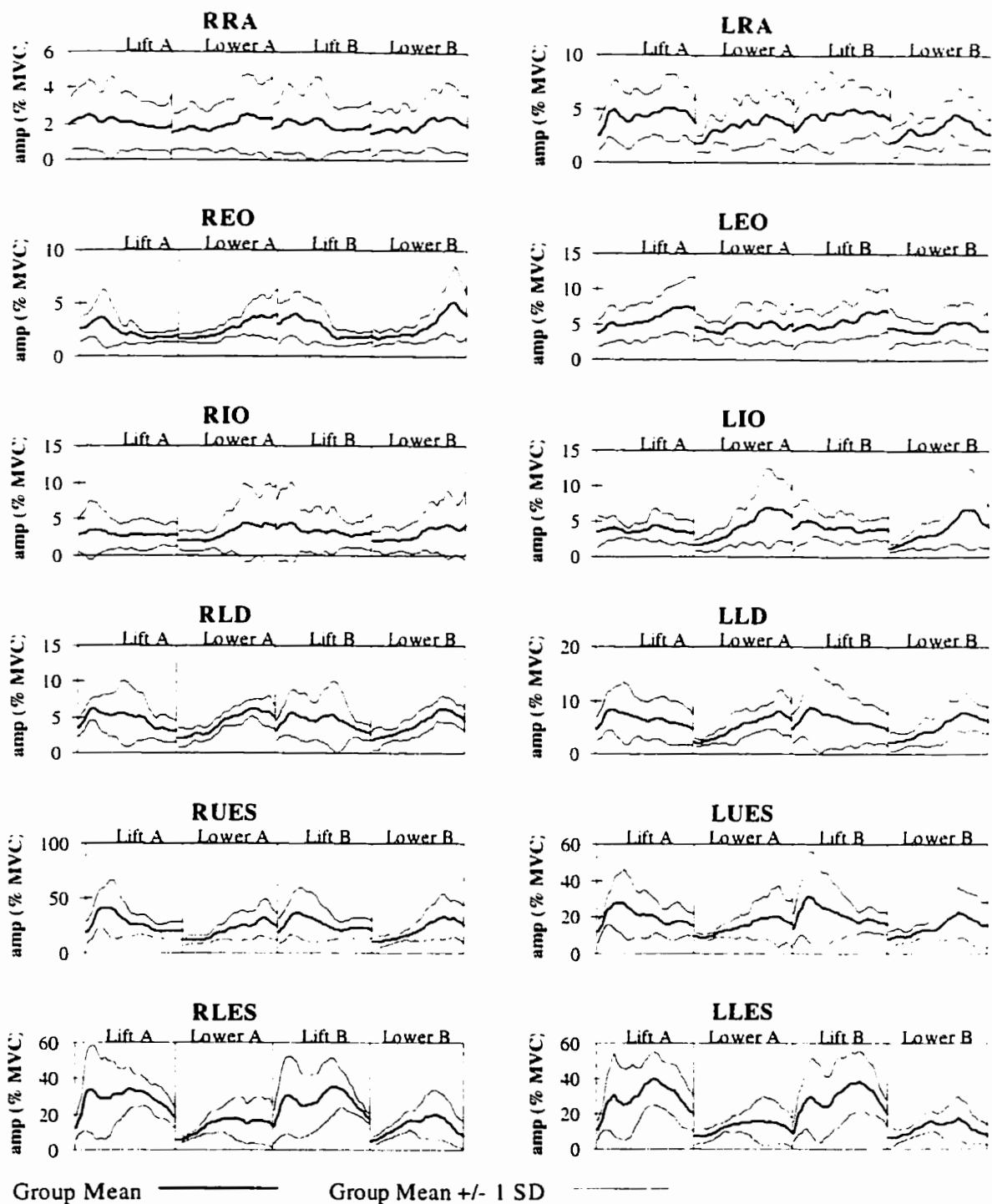


Figure 27: Summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

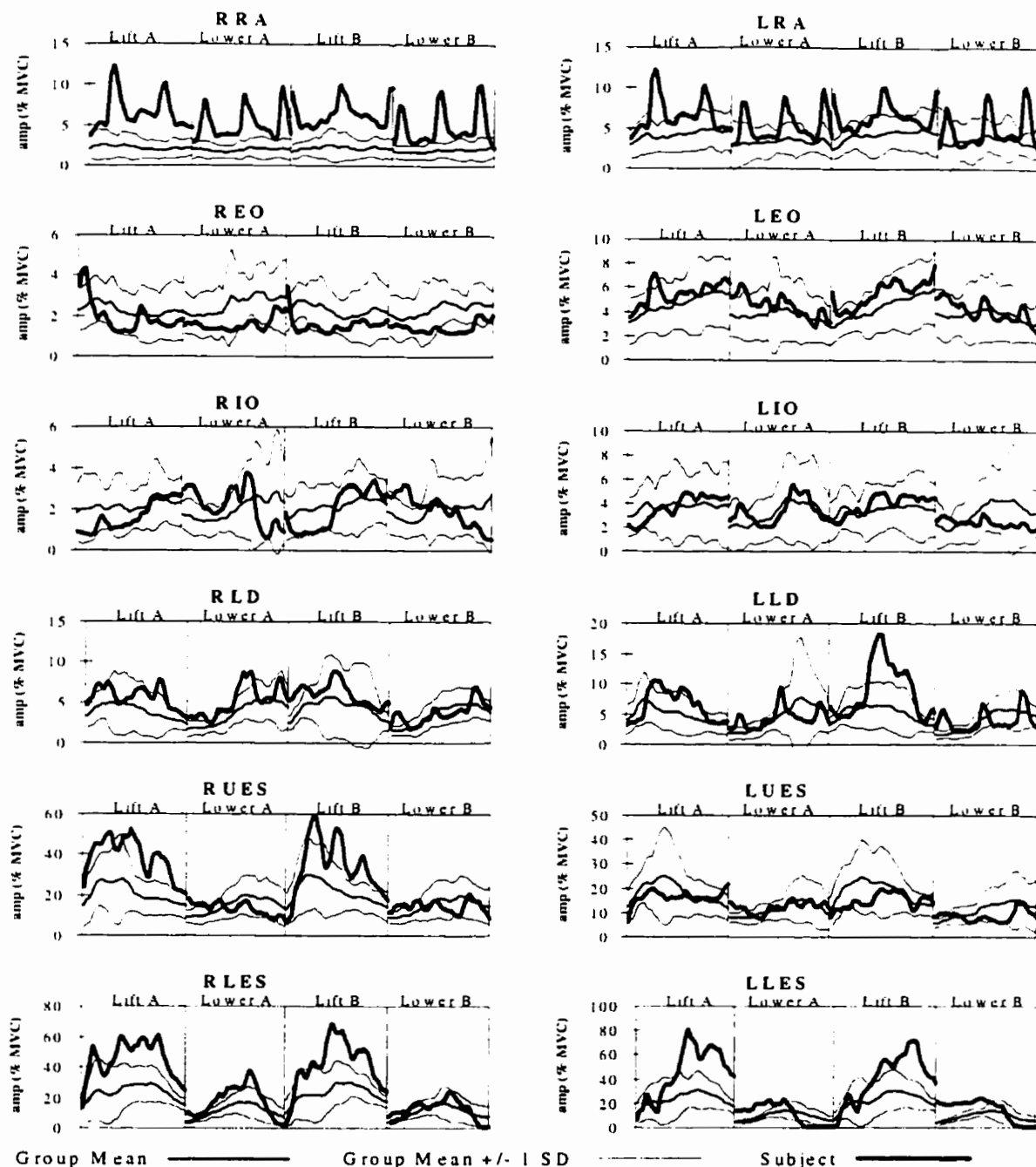


Figure 28: Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. This figure highlights the difficulty with normalizing to % MVC. Muscles that are not scaled to “true” MVCs appear to excessive amounts of activity. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

EMG - units of N·m

The RMS differences were calculated between the L4/L5 reaction moment time history, as calculated by the linked segment model, and the moment time histories produced using the six EMG-to-moment scaling factors (described in the Methods, Data Analysis, EMG -units of N·m section), for each day, load and lift and lower. The RMS differences were expressed as a percentage of the largest moment produced in each subject's trial. Across all conditions, the upper pair of electrodes produced the smallest mean RMS difference of 41.5 %, while the lower pair of electrodes had a mean RMS difference of 49.8 %. Based on these results, the right and left upper erector spinae electrode scaling factors were selected as the ones to use in order to estimate the L4/L5 reaction moment. The individual RMS differences for each of the days, loads

**Table 11** The mean (SD) RMS difference (% of maximum) in the L4/L5 reaction moment, as calculated by the linked segment model and the right and left lower erector spinae EMG. The values were expressed as a percentage of each subjects maximum L4/L5 moment (N.m) produced during a trial.

		Load (kg)					
		0		5		10	
Lift #		1	2	1	2	1	2
Day	1	43.1(17.0)	45.6(16.8)	47.8(17.4)	47.5(17.4)	49.0(15.4)	47.5(19.8)
	2	43.4(12.1)	44.2(13.5)	37.7(13.5)	41.0(13.4)	35.9(13.8)	39.3(15.6)
Lower #		1	2	1	2	1	2
	Day	1	60.9(11.9)	61.7(11.9)	60.1(10.0)	61.4(12.1)	58.7(12.4)
	2	55.1(10.8)	55.7(11.4)	50.2(13.3)	51.9(12.6)	48.9(16.4)	48.3(14.4)

and activities for the upper and lower electrode pair are listed in Tables 11 and 12, respectively.

**Table 12** The mean (SD) RMS difference (% of maximum) in the L4/L5 reaction moment, as calculated by the linked segment model and the right and left upper erector spinae EMG. The values were expressed as a percentage of each subjects maximum L4/L5 moment (N.m) produced during a trial.

		Load (kg)					
		0		5		10	
Lift #		1	2	1	2	1	2
Day	1	41.8(10.3)	43.4(12.1)	39.7(11.6)	37.3(10.1)	38.6(6.9)	37.6(8.8)
	2	37.9(8.8)	38.5(8.1)	29.5(8.5)	30.5(8.9)	28.0(9.0)	31.4(8.8)
Lower #		1	2	1	2	1	2
	1	54.2(15.7)	53.1(17.0)	48.7(13.2)	50.6(14.2)	51.0(6.8)	50.1(7.0)
Day	2	50.2(16.7)	49.4(15.5)	42.5(15.5)	41.9(15.1)	36.3(13.9)	34.7(15.0)

By using the scaling factor for the upper erector spinae electrode pair, the L4/L5 reaction moment time history was produced for each person, for each day, for the three loads of lifts and lowers. An ensemble average of the ASYMP group L4/L5 reaction moment, representing days, loads and trials was then produced (Figure 28). Figure 29 is an example of the L4/L5 moment time history for a SYMP individual (Case Study #3).

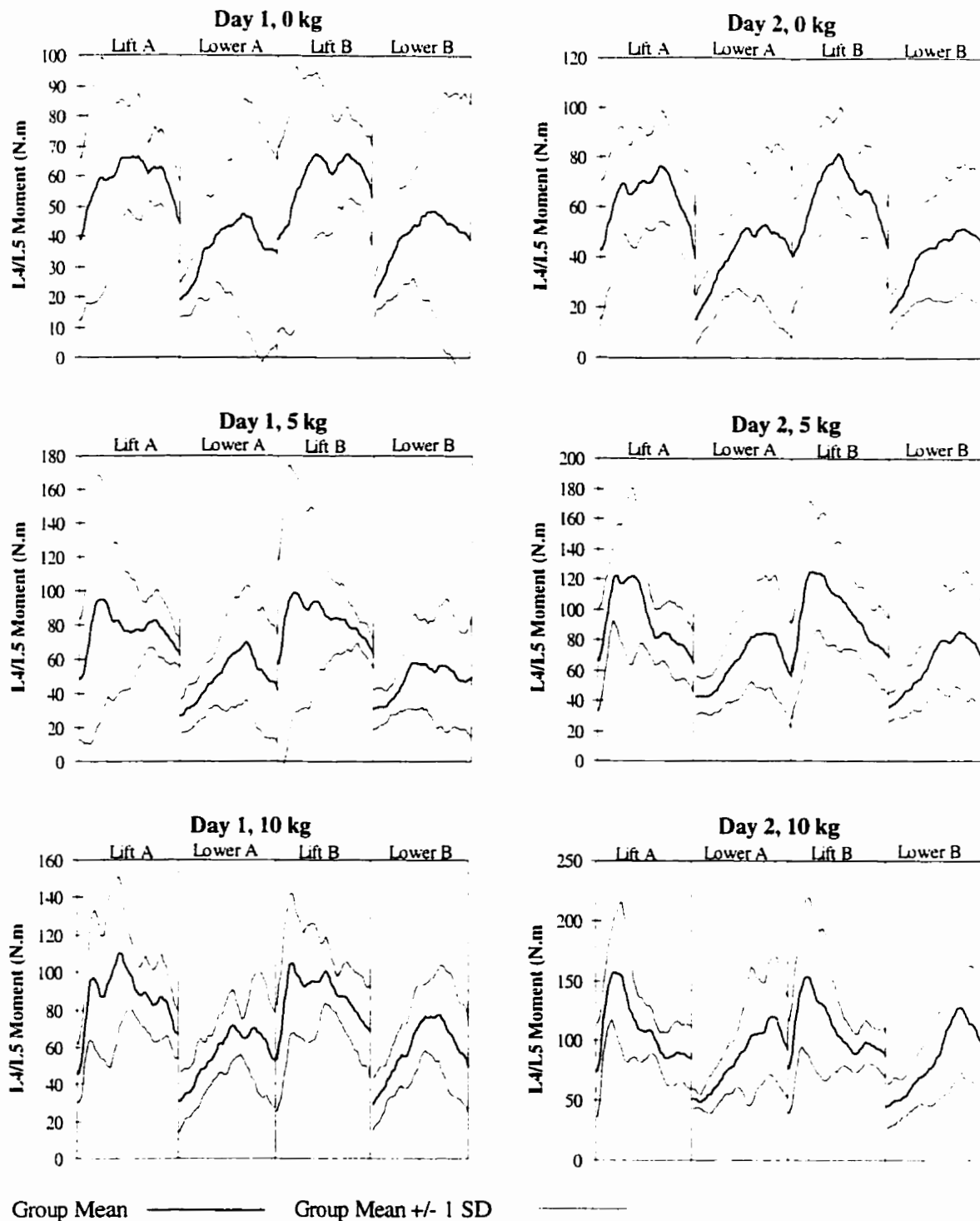


Figure 29: Summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects Day 1, 10 subjects Day 2) L4/L5 moment (N.m) calculated using moment normalized EMG from the left and right upper erector spinae electrodes. Each graph identifies a specific day and load. Within each graph, the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower.

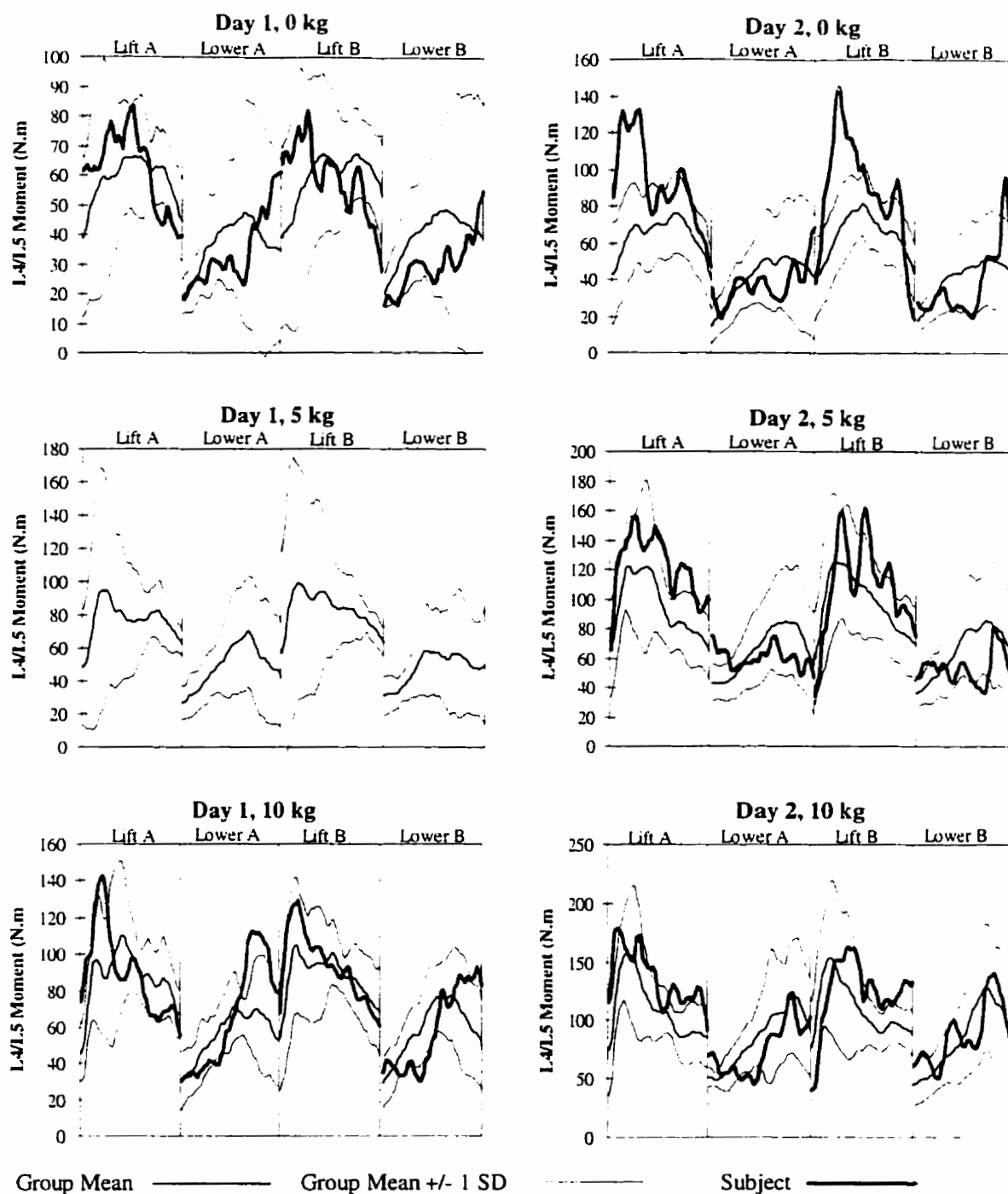


Figure 30: Case Study #3 graphed against the ASYMP mean ( $\pm 1$  SD) L4/L5 moment profile calculated using moment normalized EMG from the left and right upper erector spinae electrodes. Each graph identifies a specific day and load (N·m). Within each graph the panels indicate specific lifts (A or B extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Note: Day 2 = Bad Day and no data was available for the Good day, 5 kg load.



## Model Outputs

### Muscle Force

For each electrode site, the muscle forces produced by each muscle fascicle were summed. This allowed the results to be presented graphically as they were for the mV and %MVC EMG data. The profiles display the mean ASYMP muscle force per electrode, produced during the 0, 5 and 10 kg lifts and are presented in Figures 30 to 35. For each of the flexor muscles, the forces on the right side were less than those produced in the left. The latissimus dorsi muscle forces were greater for lifts than lowers, and increased slightly with increases in load, but the average force was less than 100 N. The upper erector spinae forces were also greater for the lifts and increased with the size of the load in the hands. The upper erector spinae forces were the largest and were greater in lifts than in lowers and increased with the load in the hands. The double peak evident in the EMG signals was not present in this display format.

Figure 36 is an example of the EMG patterns for a SYMP individual (Case Study #3).

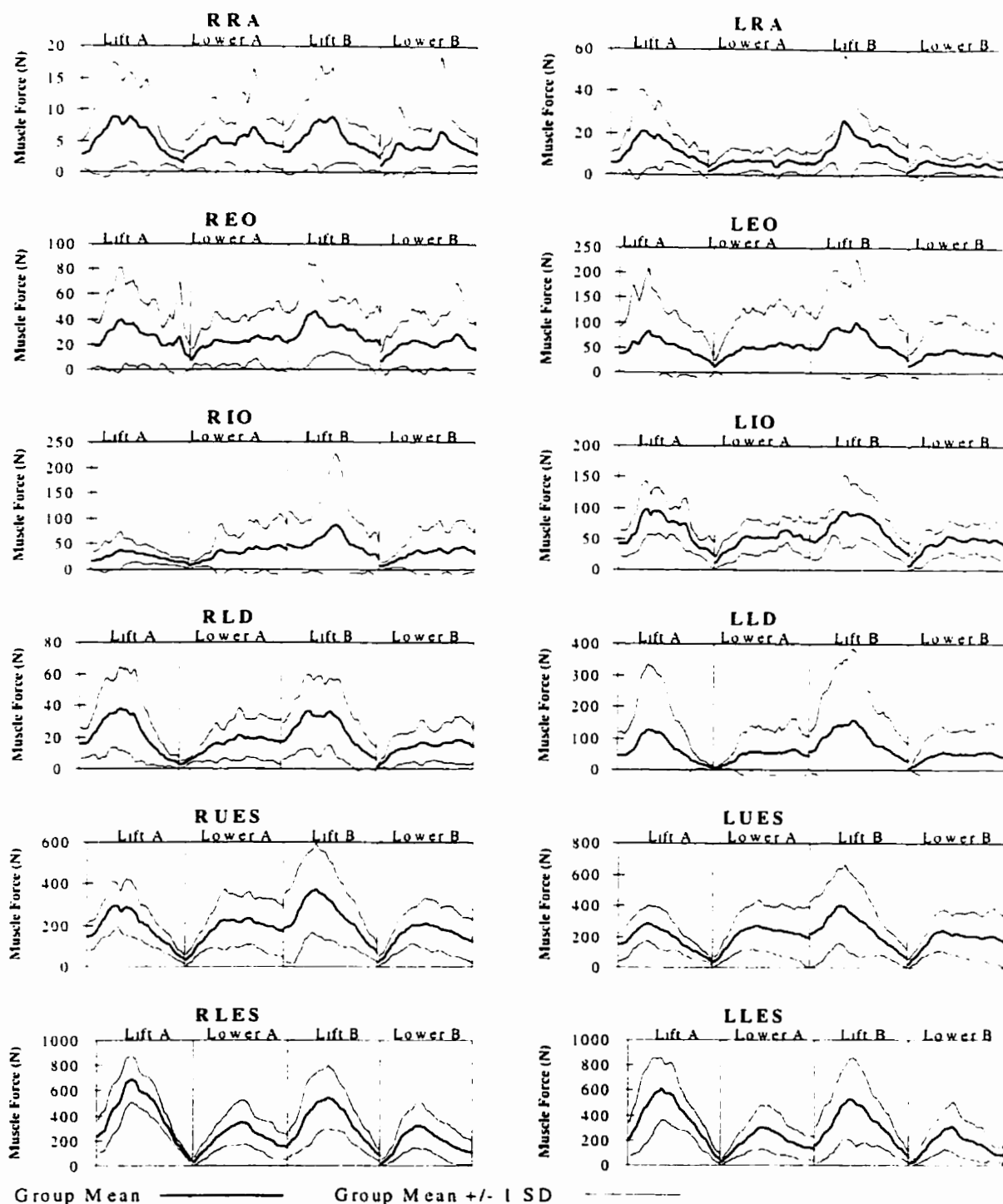


Figure 31: Summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) muscle force (N) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

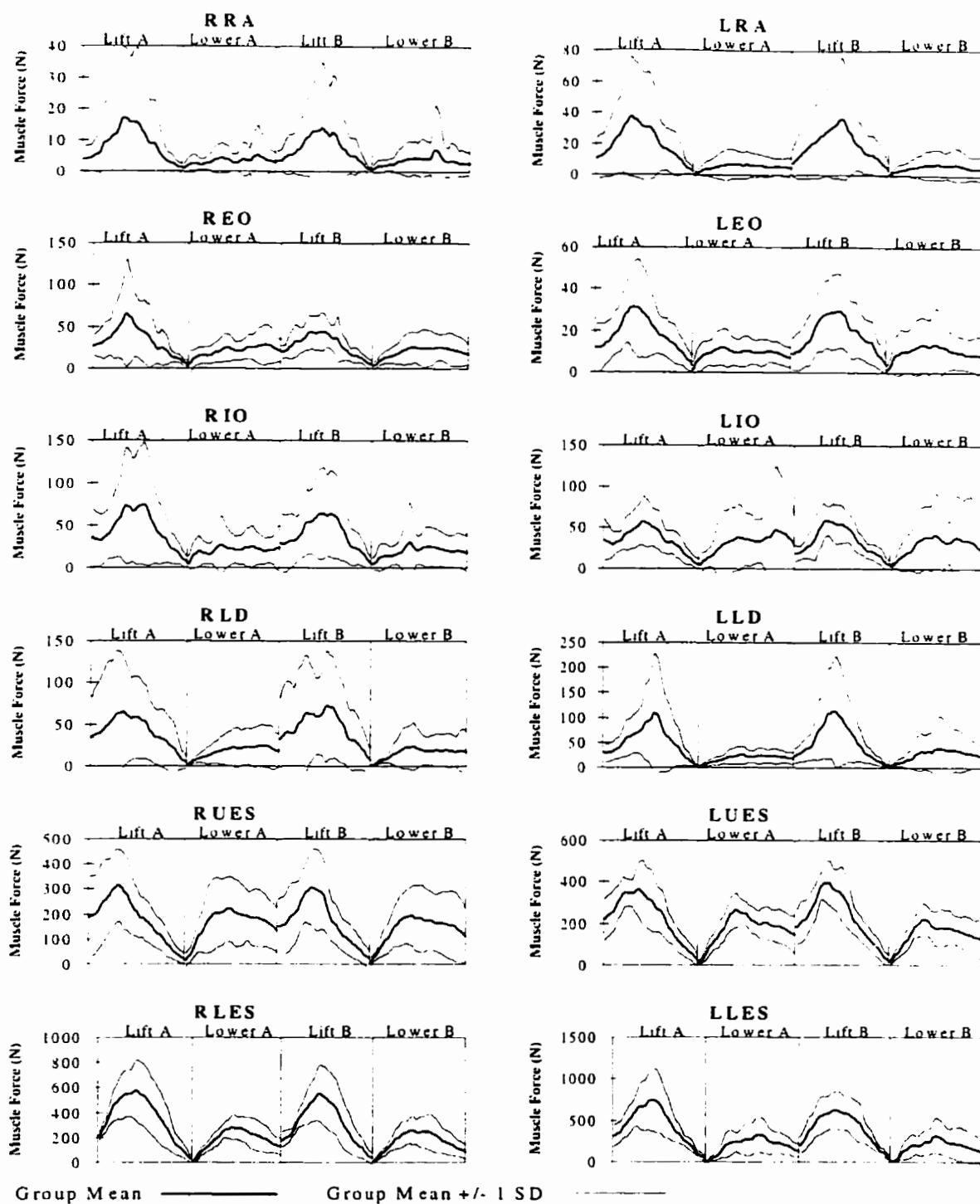


Figure 32: Summary profile of the mean ( $\pm$  1 SD) ASYMP (10 subjects) muscle force (N) for Day 2, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

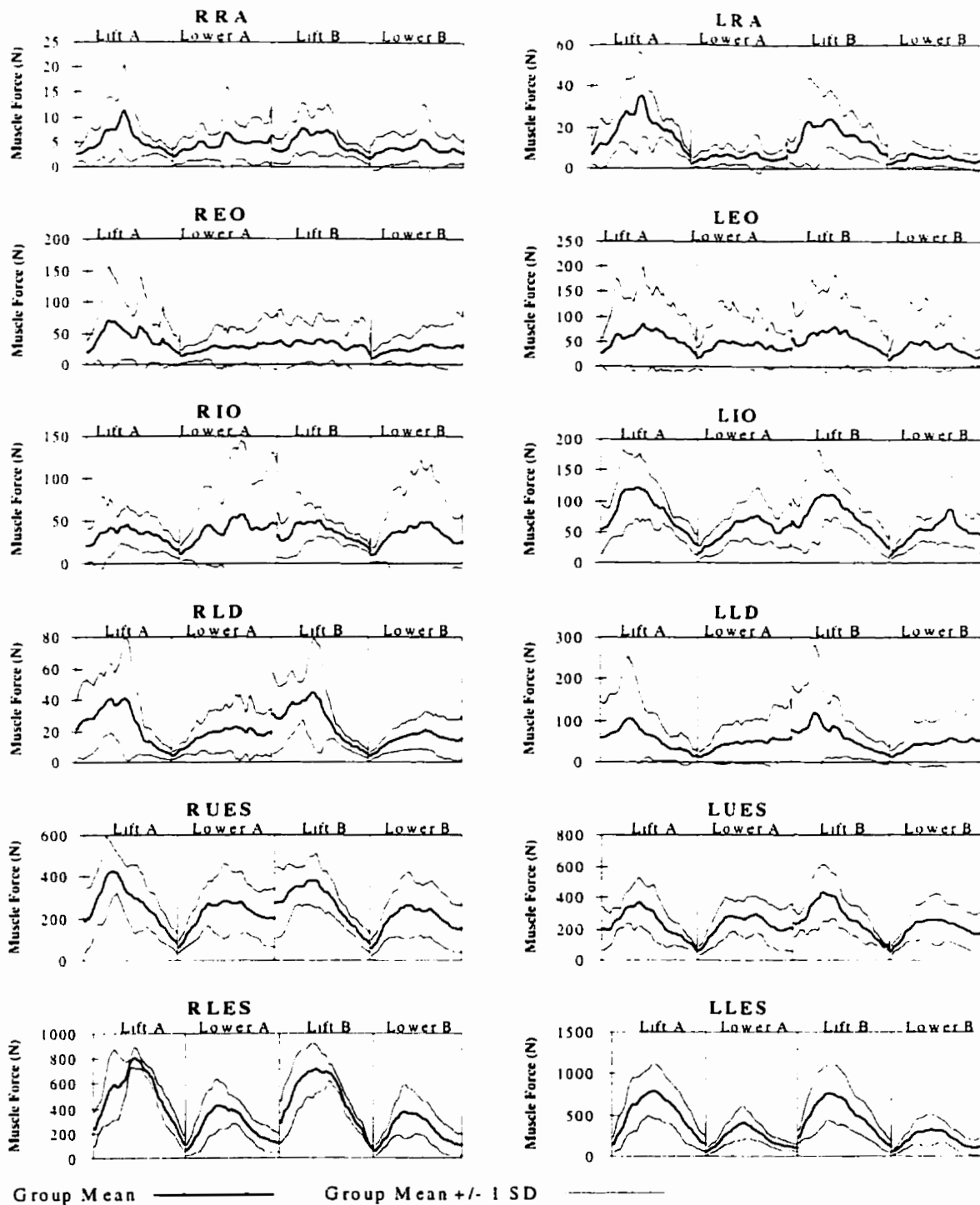


Figure 33: Summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) muscle force (N) for Day 1, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

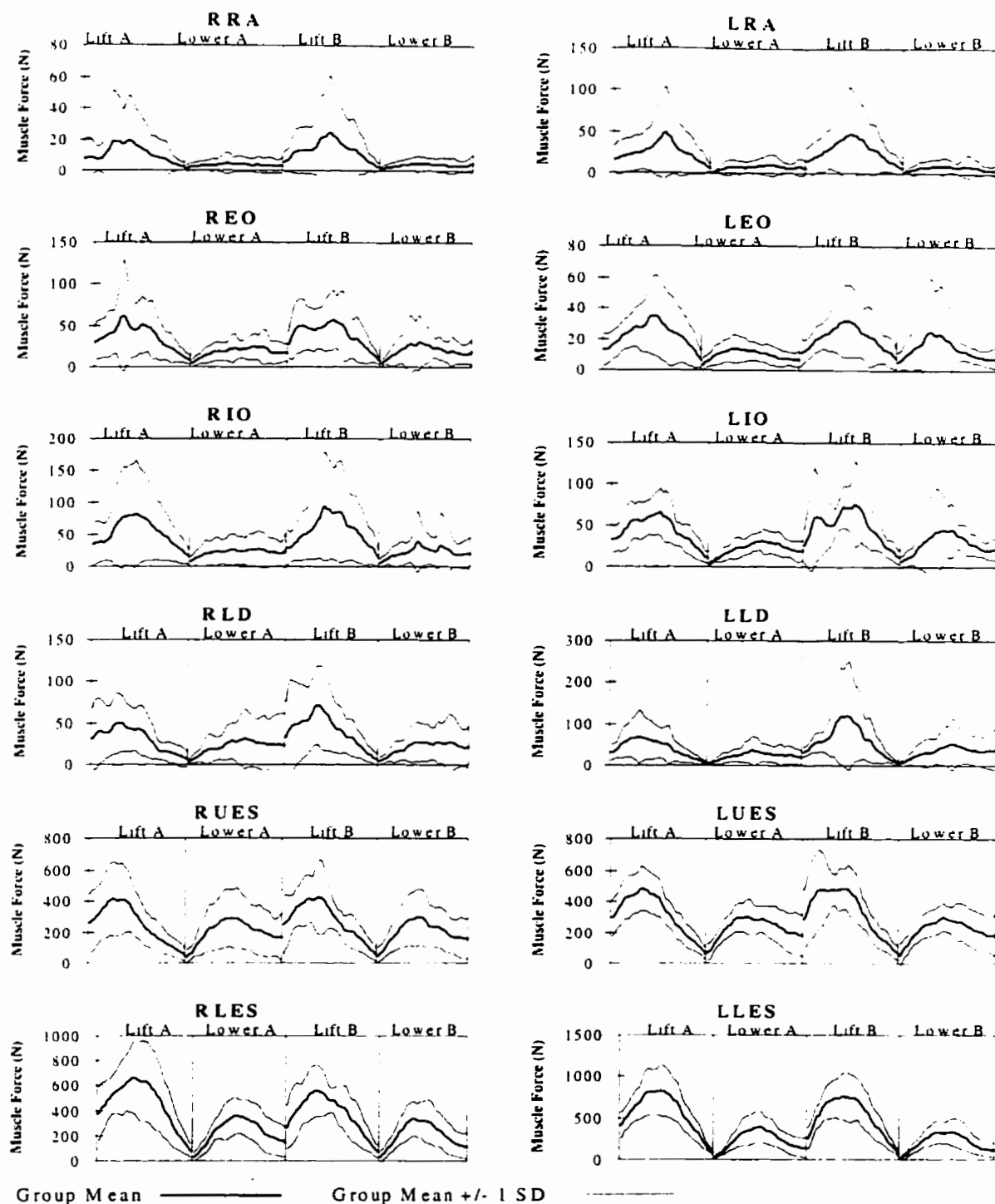


Figure 34: Summary profile of the mean ( $\pm$ 1 SD) ASYMP (9 subjects) muscle force (N) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

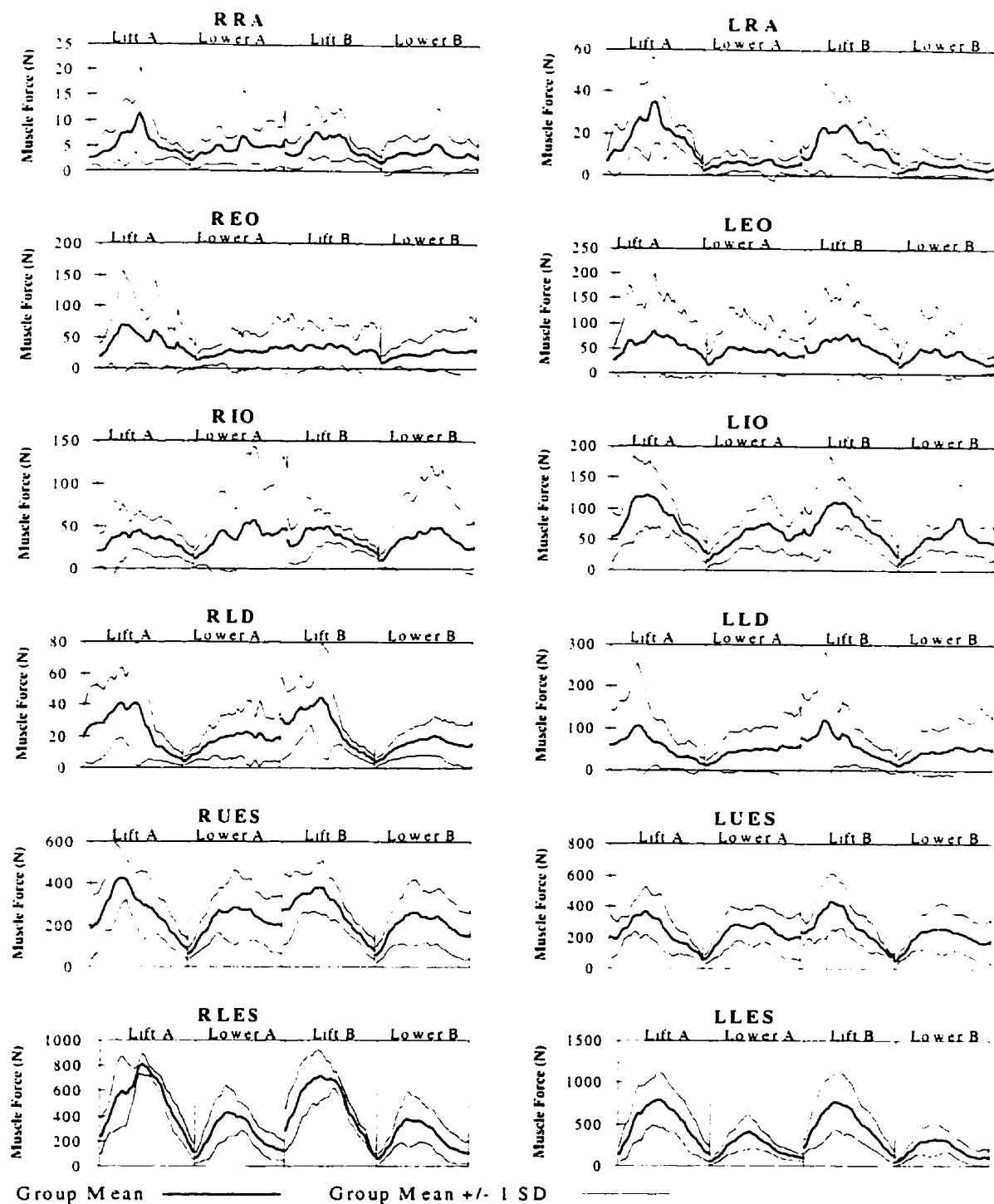


Figure 35: Summary profile of the mean ( $\pm$ 1 SD) ASYMP (8 subjects) muscle force (N) for Day 1, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA = rectus abdominis, EO = external oblique. IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae. LES = lower erector spinae.

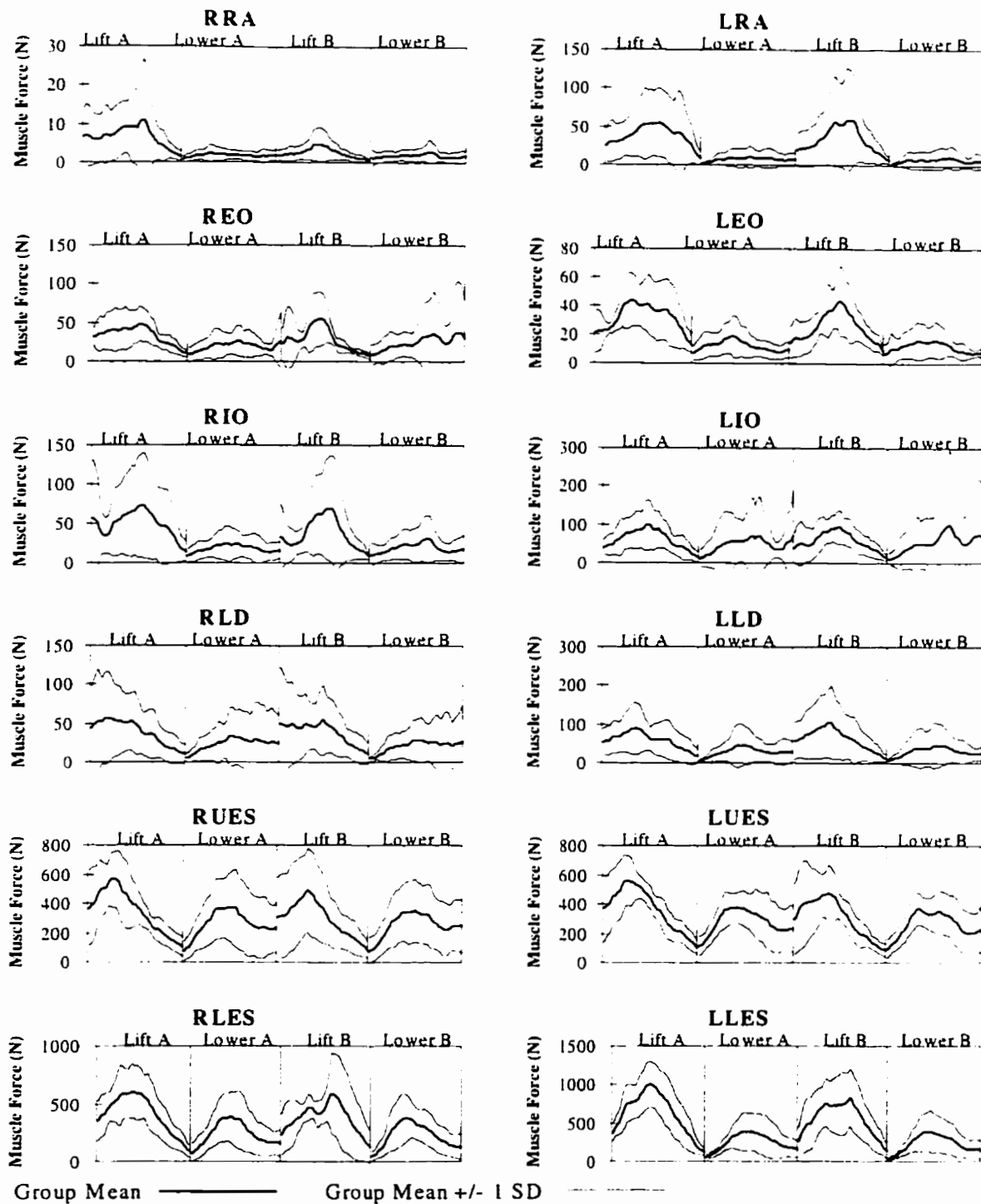


Figure 36: Summary profile of the mean ( $\pm$ 1 SD) ASYMP (8 subjects) muscle force (N) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

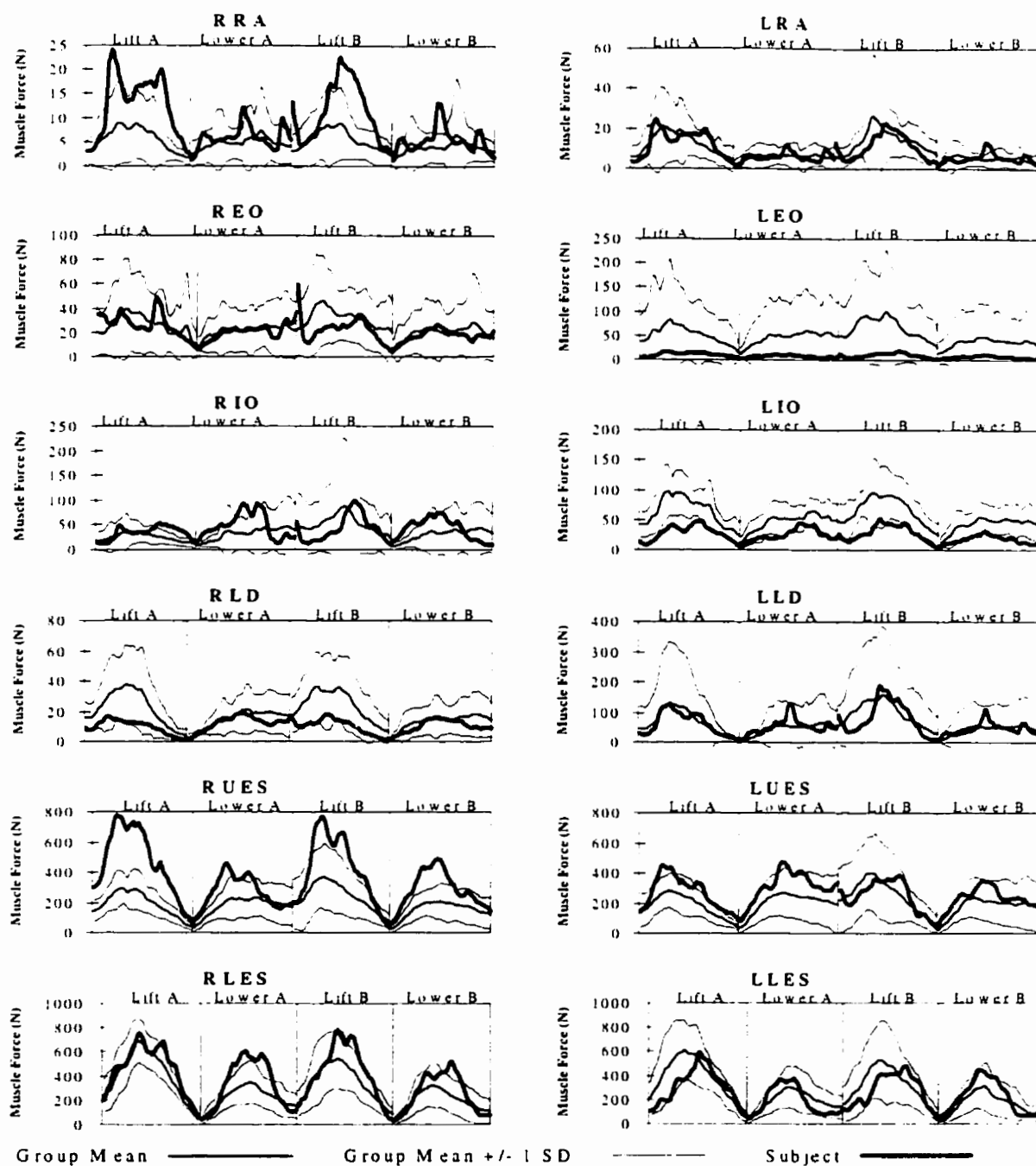


Figure 37: Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (8 subjects) muscle force (N) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. The  $EMG_{Force}$  processing method highlights regions of excessive activity (e.g. RUES) that are “normal” in the mV normalization and is not affected by submaximal efforts encountered in the MVC approach (e.g. RRA). Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.



The summary profiles provide a visual impression of the muscle force time histories. However, for a given individual, it is difficult to quantify the amount of time that the muscle force is above the mean + 1 SD level. To measure this, the muscle forces were first graphed as an amplitude probability distribution functions (APDF). Figure 37 shows the summary APDF for the asymptomatic individuals Day 1, 0 kg load condition. In Figure 38 the APDF of Case Study #3 has been superimposed. Periods of increased muscle force for Case Study #3 are indicated whenever that curve lies to the right of the ASYMP mean + 1 SD curve. To quantify the total time spent above the APDF criterion for a specific muscle, the number of observations to the right of the mean + 1 SD were divided by the total number of observations. This calculation was performed for each muscle, for each day, load, lift and lower. The total time above the APDF criterion was then summed for all twelve electrodes. The maximum amount of increased activity would be 1200% (12 muscles \* 100% increased activity). Dividing the total time above the APDF criterion by the maximum amount of time (1200%) and multiply by 100 expressed as a percentage (range = 0% to 100%) the amount of increased force production. This single number provides an overall indication of the amount of time that force production greater than the ASYMP criterion force (mean + 1 SD level) occurred. Figure 39 is an example of the summary APDF data for a SYMP individual (Case Study #3).

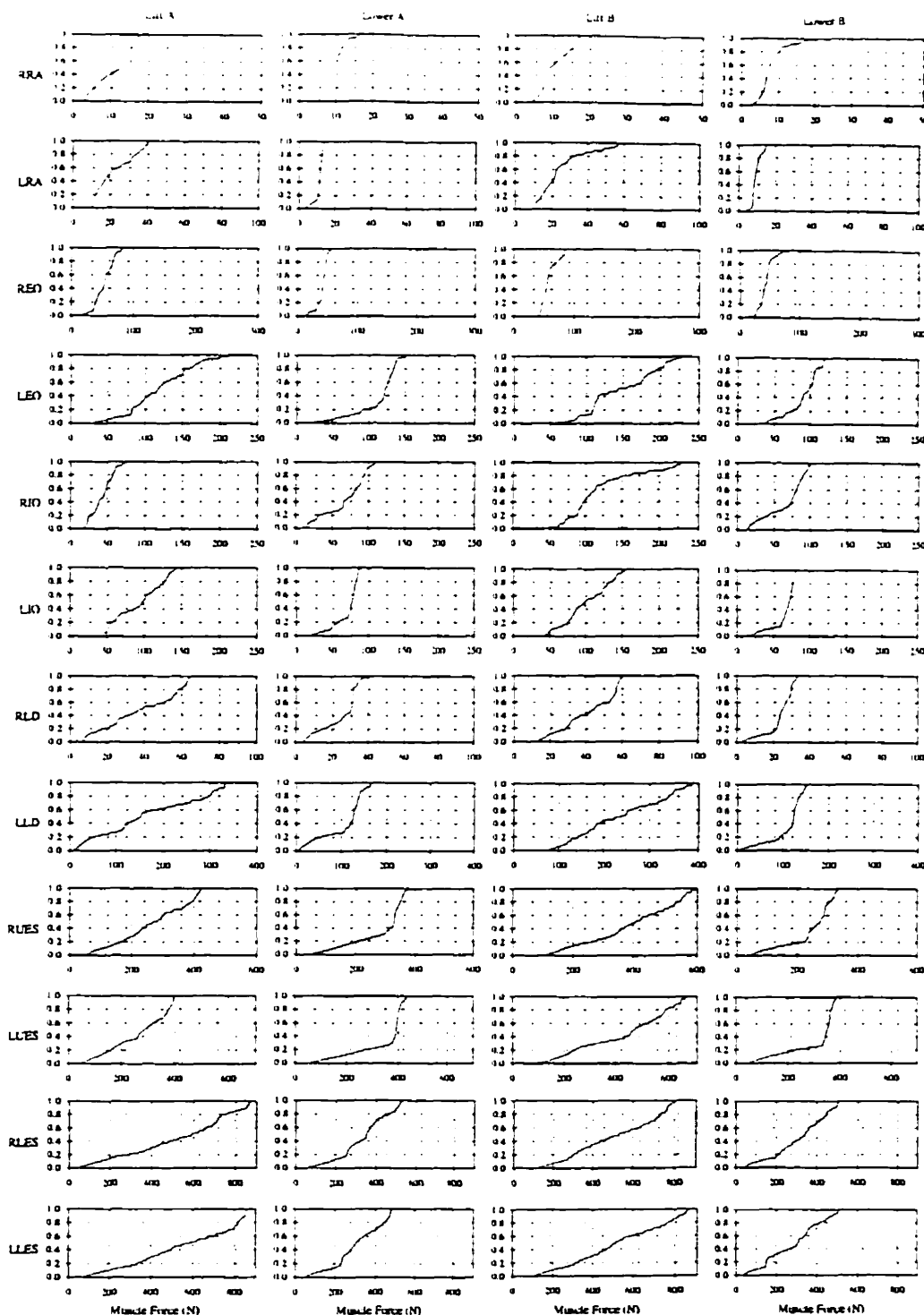


Figure 38: Summary APDF profile of the muscle forces (N) by each electrode site for Day 1, 0 kg load. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 9$  subjects) group. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability.

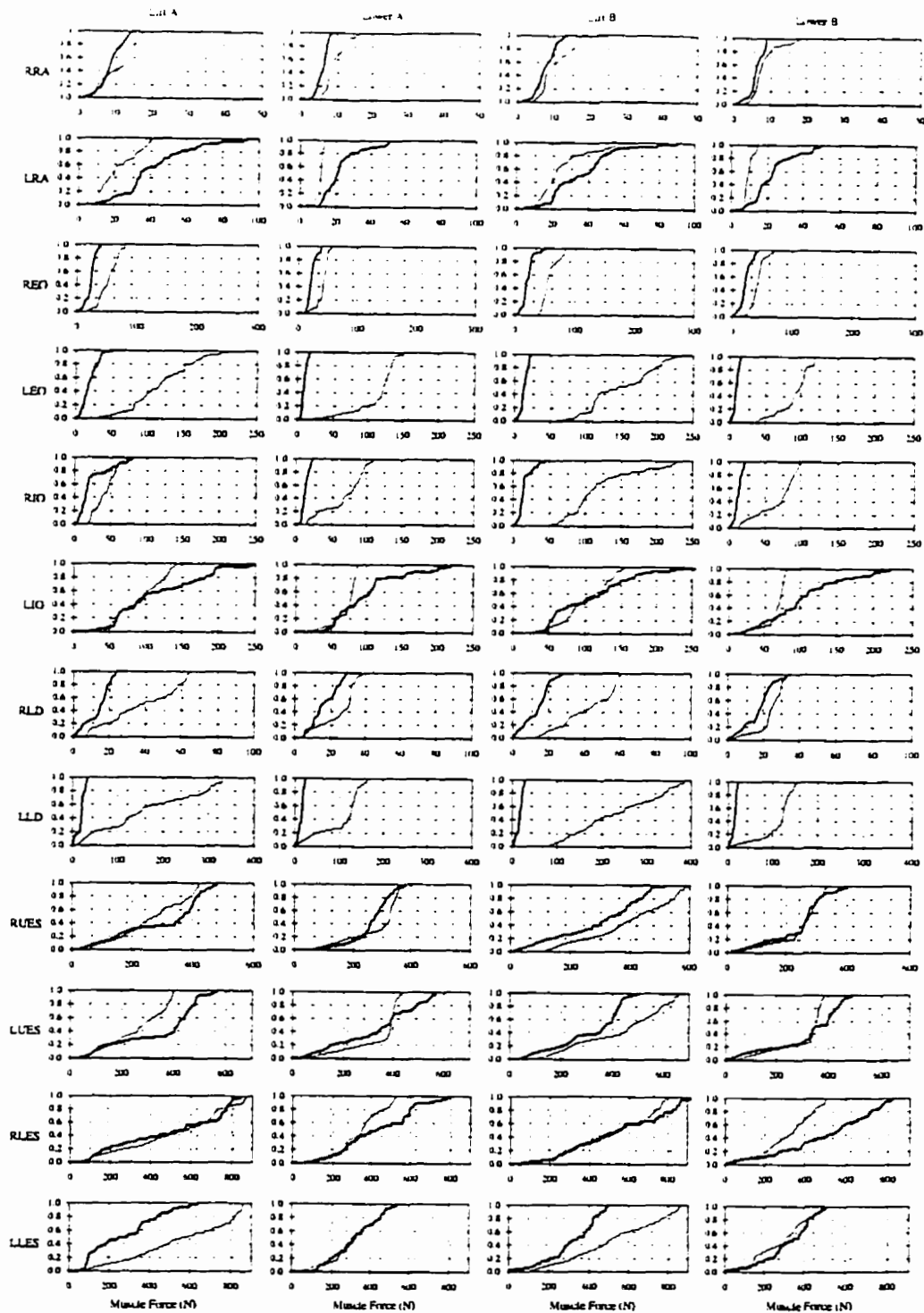


Figure 39: Summary APDF profile of the muscle forces (N) by each electrode site for Day 1, 0 kg load and Case Study #3. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 9$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability.

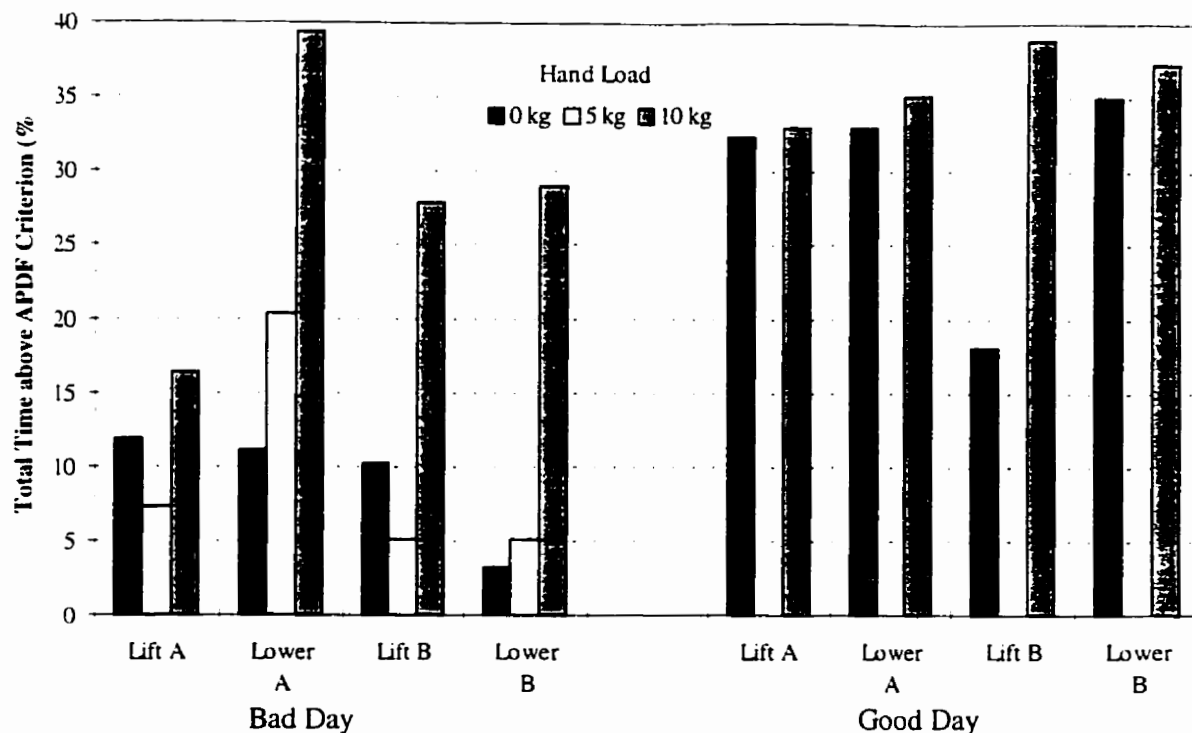


Figure 40: The cumulative time (%), for Case Study #3, in which the muscle force for each electrode site ( $n = 12$ ) was greater than the ASYMP criterion force (mean + 1 SD). Note: no data available for the Good day, 5 kg load.

### L4/L5 Compression

The time normalized, L4/L5 compression time histories estimated by the model for the ASYMP group were used to produce the ensemble averages for each day and load as illustrated in Figure 40. As expected, the load in the hands increased the peak and average compression values. Figure 41 is an example of the L4/L5 compression for a SYMP individual (Case Study #3).

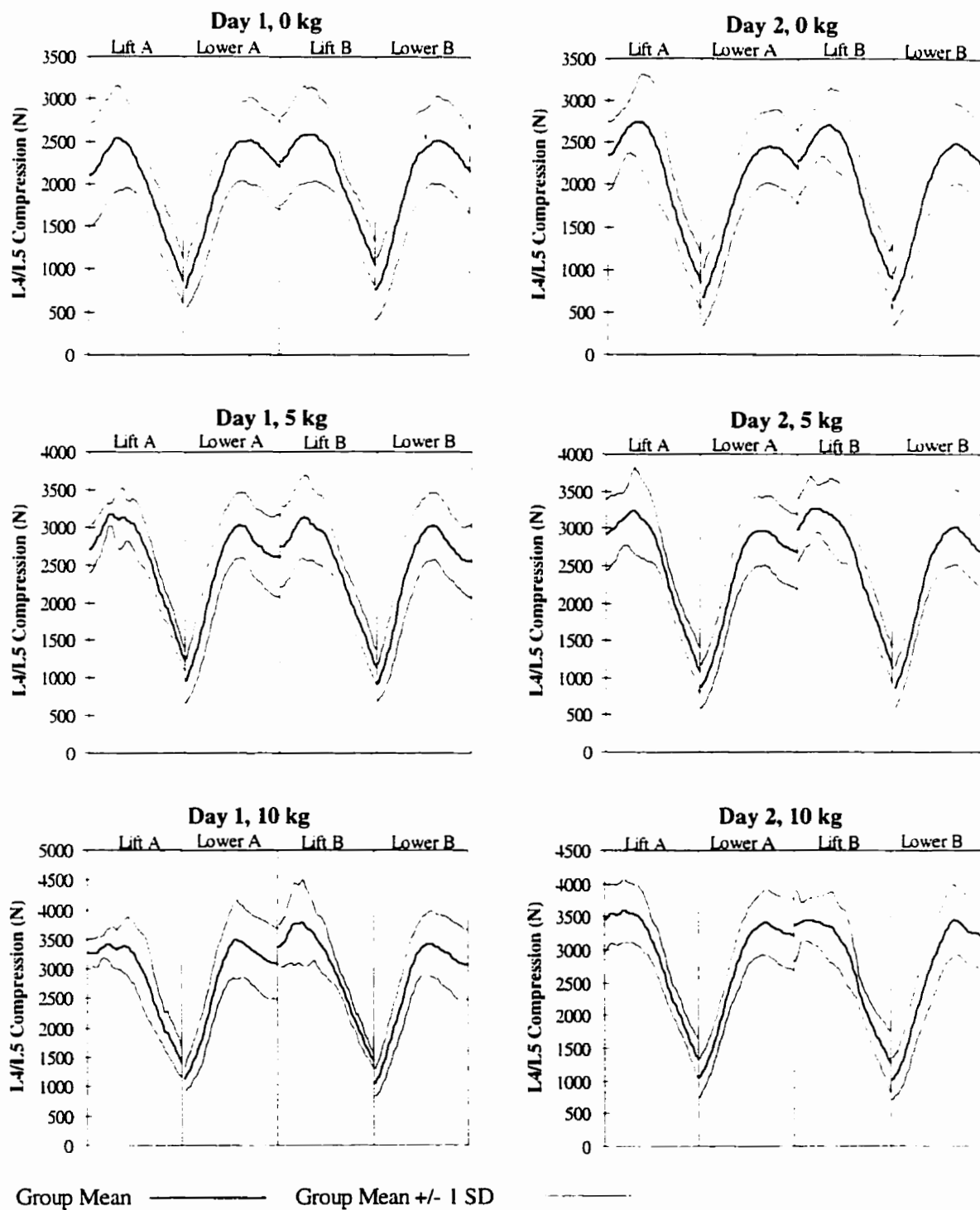


Figure 41: Summary profile of the mean ( $\pm$  1 SD) ASYMP L4/L5 compression force (N). Each graph identifies a specific day and load. Within each graph, the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower.

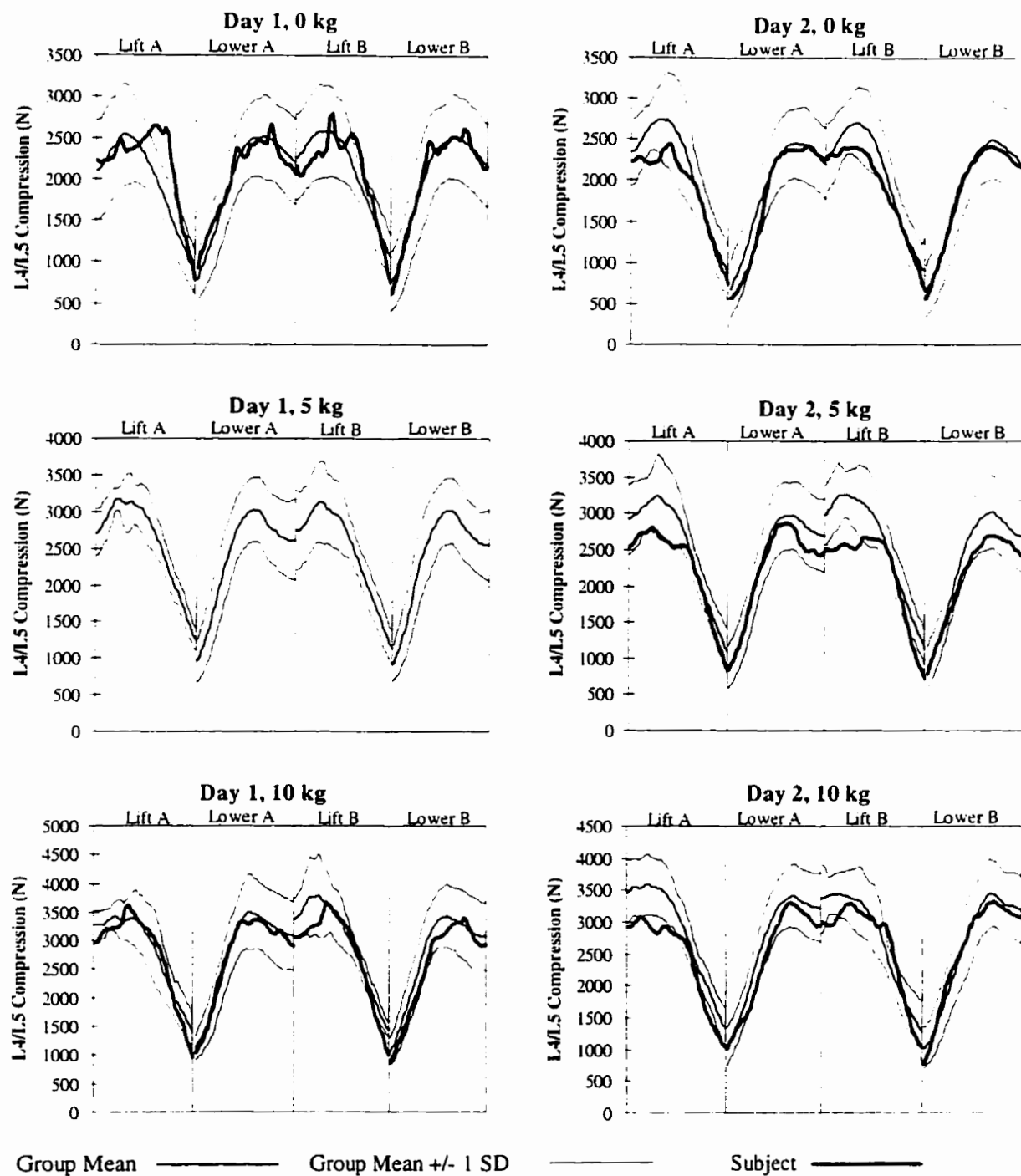


Figure 42: Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP ) L4/L5 compression force (N). Each graph identifies a specific day and load. Within each graph, the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Note: Day 2 = Bad Day and no data was available for the Good day, 5 kg load.

### **Case Study Presentations**

Four of the study participants were individuals symptomatic for recurrent low back pain. Each individual was tested on days that they described as a “good day” and a “bad day”. Inclusion in the study did not require a specific order for the good day/bad day. For each of the cases, the results for each of the analysis tools are presented.

Case Study #1

This 14 year old, 1.8 m, 78.0 kg high school student suffers from multiple joint dysfunction in the thoracolumbar and sacroiliac regions and an erector spinae strain. Despite these difficulties, this individual was very active in football and basketball. The right was typically worse than the left and he was negative for radiation of pain into the legs.

**Table 13** Comparison of “Bad” and “Good” day test results, by assessment tool, for Case Study #1.

Assessment Tool	Bad Day (Day 1)	Good Day (Day 2)
Oswestry Score (%)	11	11
VAS (following each test component)	4, 7, 8, 10, 12	2, 4, 7, 9, 10
B-200 OOC Back Dysfunction	None	None
- # of abnormal indicators	0	0
- # of non-physiological indicators	2	0
B-200 Profile - velocity	!rot, !f/e, !lf	✓rot, ✓f/e, .lf
- abnormal secondary moment	1	8
- abnormal tertiary moment	4	2
Moment Profile (N·m)		
- 0 kg	below upper limit	below upper limit
- 5 kg	below upper limit	below upper limit
- 10 kg	above upper limit	above upper limit
Muscle Force - APDFs (average time above APDF criterion (%))		
- Lifts (0, 5, 10 kg)	3, 7, 13	0, 4, 7
- Lowers (0, 5, 10 kg)	12, 22, 24	10, 11, 10
Compression		
- 0 kg	within normal limit	within normal limit
- 5 kg	within normal limit	within normal limit
- 10 kg	within normal limit	within normal limit



### Pain

The Oswestry low back dysfunction questionnaire rated the level of dysfunction as minimal. There was no difference in the score between days. A paired samples t test found that the VAS pain scores decreased significantly in value for the Good day,  $p < .009$ .

### Isostation B-200

The OOC test results reported normal back function for both test days and showed an increase in the maximal velocities for the Good day of testing. While there was no difference in the maximal rotation isometric strength between days, decreases of 6% in lateral flexion, 30% in flexion and 35% in extension were observed. The B-200 profile results show an increase in rotation and flexion-extension velocities into the normal range throughout the respective movement cycles. Although the lateral flexion maximal velocity improved, it remained below the normal range for both test days. The B-200 profile also revealed an increase in the lateral flexion moment produced during rotation on the Good day. The moment, which is greater than 40% MVC, is now well above the normal range. A decrease in the flexion moment produced during lateral flexion was also observed (Table 13).

### EMG - units of N·m

For both days of testing, the L4/L5 moment bordered on the upper boundary of the normal range during the lifts and lowers, for the 0 and 5 kg loads. For the 10 kg load, it exceeded this region on both test days (Figure 42).

### Muscle Force APDFs

On the Bad day, increased force was noted in the RLD and RUES for both lifts and lowers and the duration of the increased force increased with load (Figure 43). On the Good day, there was increased LUES force, primarily during the lowers, and an increase in RLES force, primarily during the lifts (Figure 43). Overall, on the Good day, there was less time in which the muscle forces exceeded the APDF criterion (Figure 44).

### L4/L5 Compression

For both test days, the compressive forces matched the average pattern, but the magnitude was typically at or below the mean -1 SD value (Figure 45).

### Interpretation

Significant improvements in pain from the Bad to Good Day were reflected by the VAS, but not the Oswestry scores. The normal back function reported by the OOC evaluation system is a combination of the conservative nature of the decision rules and the vigorous effort put forth by this individual. The OOC baseline rehabilitation data does reflect an improvement in performance, but indicates further rehabilitation is required (Appendix C). The B-200 profile, particularly the velocity profile, highlights a return to a more normal performance. The increased lateral flexion moment during rotation may be a consequence of the maximal effort produced throughout the testing. The decrease in the total muscle force above the APDF criterion level reflected the decreased magnitude of excessive force production. There was a shift in the muscles producing excessive forces from the upper right sided musculature (RLD, RUES), to the

left UES and right LES. It is a reasonable hypothesis that the increased EMG activity contributed to the right sided pain/dysfunction. Although the patient had a “Good” day, there are still some residual problems, as shown by the decreased lateral flexion velocity and the increased EMG moment produced when handling the 10 kg load.

**Table 14** Summary of increased secondary and tertiary axes moment activity for OOC 50% resistance tests, dynamic sequence 1 and 2, test days 1 and 2. Excessive secondary and tertiary activity is indicated by a ✓ and ✗, respectively. For rotation tests, lateral flexion is secondary, flexion-extension is tertiary. For lateral flexion tests, flexion-extension is secondary and rotation is tertiary. The shaded areas indicate the tests for the individual's “Bad” day.

Case Study # 1	Test Primary Axis (day - sequence #)	Movement Quadrant				Totals
		1	2	3	4	
Rotation	1 - 1					0, 0
	1 - 2	✓ ✗		✗	✗	1, 3
	2 - 1	✓	✓	✓	✓	4, 0
	2 - 2	✓ ✗				1, 1
Lateral Flexion	1 - 1					0, 0
	1 - 2				✗	0, 1
	2 - 1	✓			✓	2, 0
	2 - 2	✗			✓	1, 1
Totals	<b>Bad Day</b>	1, 1	0, 0	0, 2	0, 2	1, 4
	<b>Good Day</b>	3, 2	1, 0	1, 0	3, 0	8, 2

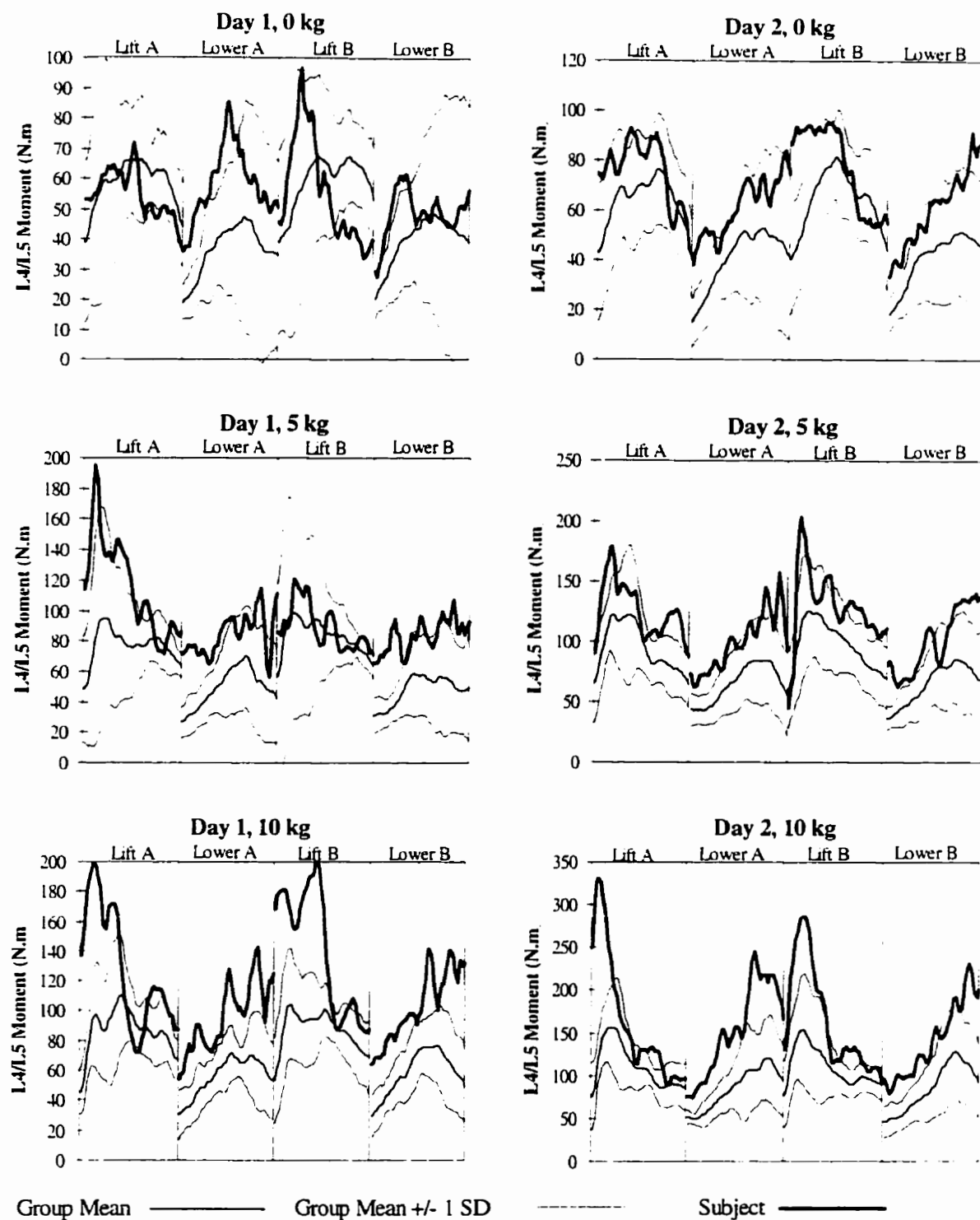


Figure 43: Case Study #1 graphed against the ASYMP mean ( $\pm 1$  SD) L4/L5 moment profile. Each graph identifies a specific day and load (N.m). Within each graph the panels indicate specific lifts (A or B extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Note: Day 1 = Bad Day.

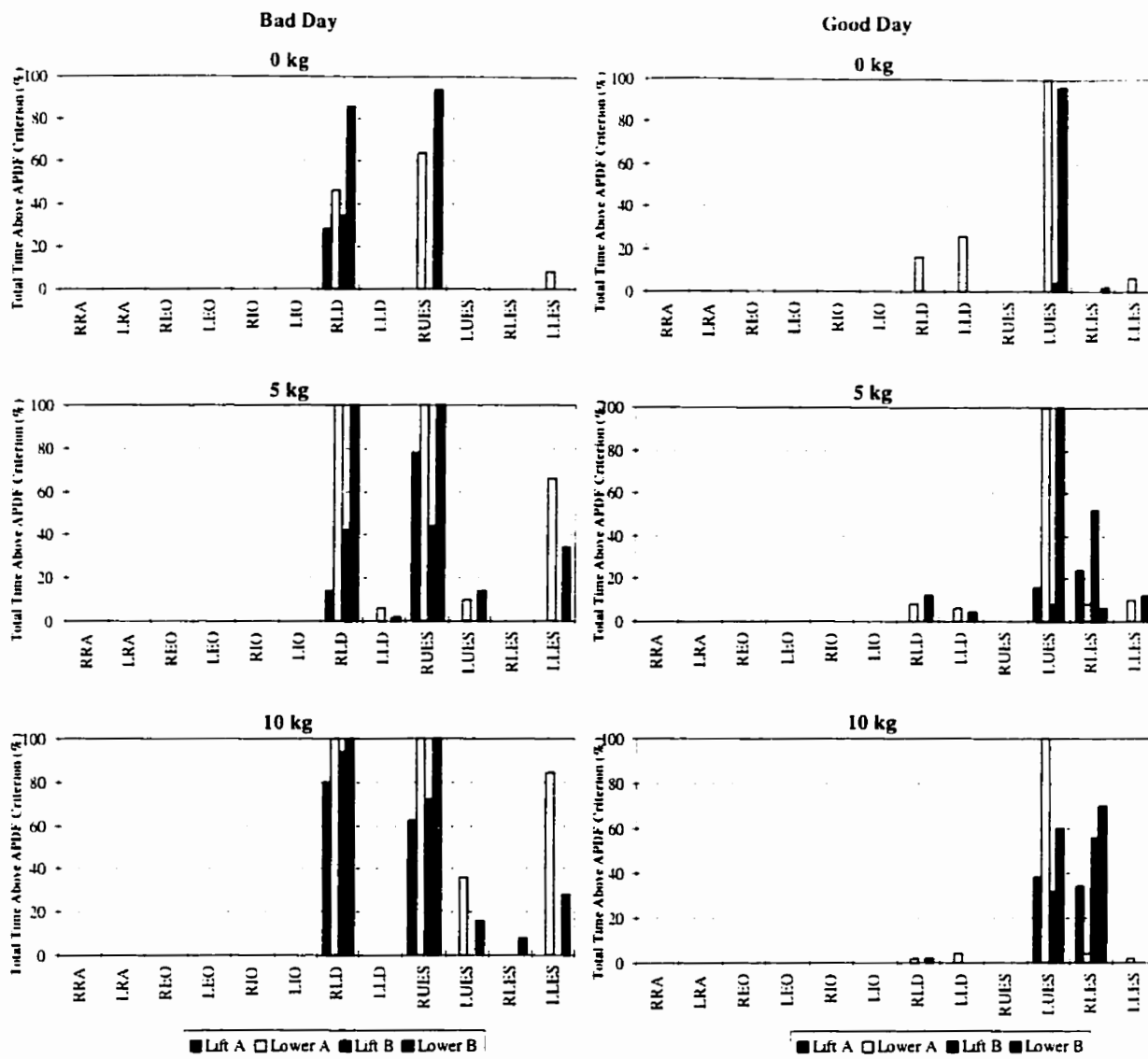


Figure 44: The time that the force for each muscle (n = 12) was greater than the ASYMP criterion force (mean + 1 SD) for Case Study #1. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

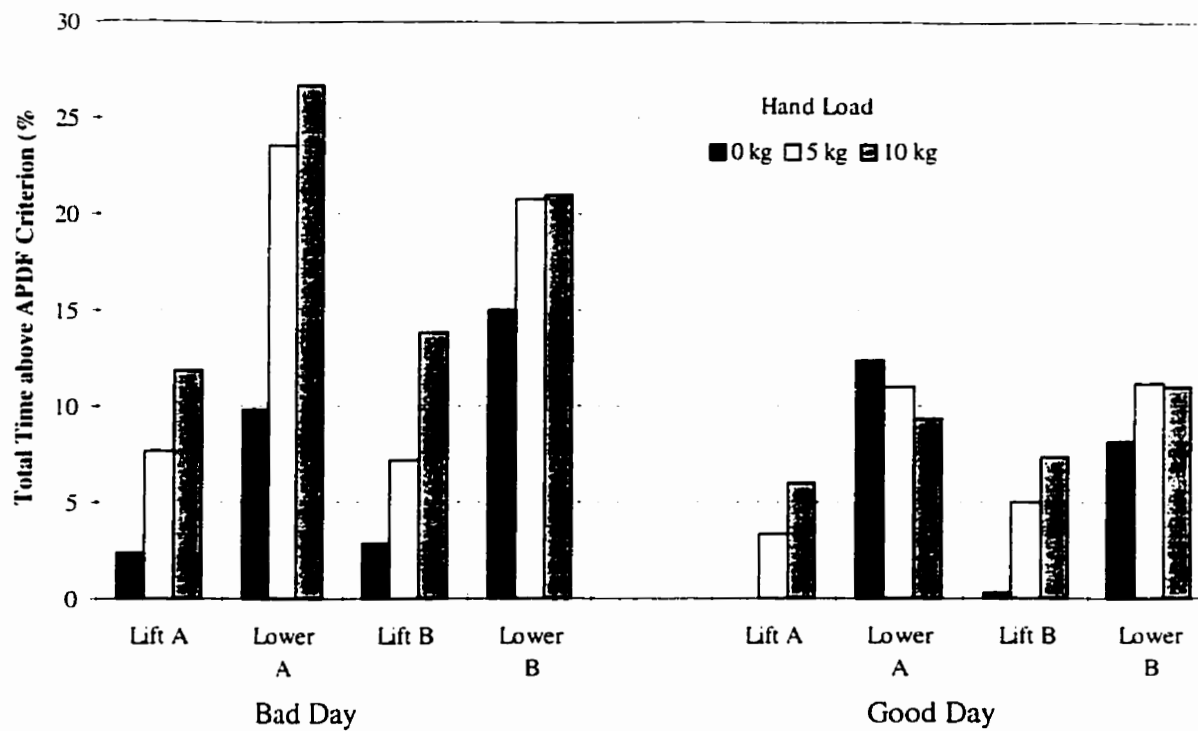


Figure 45: The cumulative time (%), for Case Study #1, in which the muscle force for each electrode site ( $n = 12$ ) was greater than the ASYMP criterion force ( $\text{mean} + 1 \text{ SD}$ ).

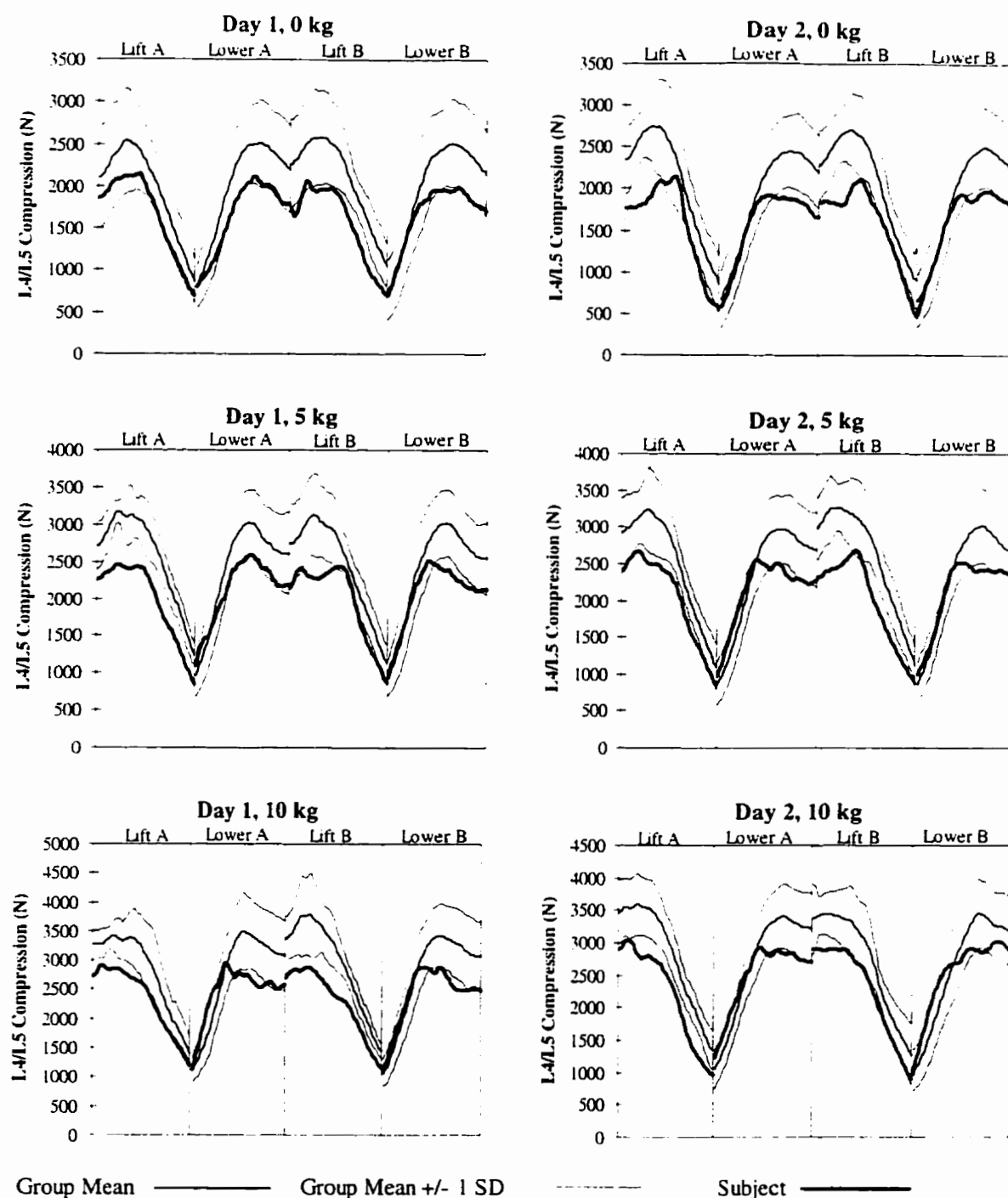


Figure 46: Case Study #1 graphed against the summary profile of the mean ( $\pm$  1 SD) ASYMP) L4/L5 compression force (N). Each graph identifies a specific day and load. Within each graph, the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Note: Day 1 = Bad Day.

## Case Study #2

This 33 year old, 1.70 m, 104 kg hockey official suffers from variable sided low and mid back pain due to chronic bilateral sacroiliac dysfunction and degenerative disc disease at L5/S1.

**Table 15** Comparison of “Bad” and “Good” day test results, by assessment tool, for Case Study #2. Note: B-200 profile data not available for Bad Day (Day 2).

Assessment Tool	Bad Day (Day 2)	Good Day (Day 1)
Oswestry Score (%)	26	24
VAS (following each test component)	7, 7, 8, 8, 9	5, 5, 8, 4, 8
B-200 OOC Back Dysfunction	None	None
- # of abnormal indicators	0	0
- # of non-physiological indicators	1	0
B-200 Profile - velocity	-	✓rot, ✓f/e, ✓lf
- abnormal secondary moment	-	3
- abnormal tertiary moment	-	7
Moment Profile (N·m)		
- 0 kg	above upper limit	below upper limit
- 5 kg	above upper limit	at upper limit
- 10 kg	below upper limit	lowers exceed limit
Muscle Force - APDFs		
Average Time above APDF Criterion (%)		
- Lifts (0, 5, 10 kg)	53, 46, 34	29, 32, 31
- Lowers (0, 5, 10 kg)	75, 58, 39	31, 32, 34
Compression		
- 0 kg	at upper limit	above upper limit
- 5 kg	at upper limit	above upper limit
- 10 kg	at upper limit	above upper limit



### Pain

The Good day showed a 2% decrease in the Oswestry score. This reflected a decrease in his pain intensity rating from moderate to very mild. However, the Oswestry classification is still moderate dysfunction. Paired samples t-tests revealed a non-significant trend in decreased VAS scores between days ( $p < .053$ ), although four of the five lower scores occurred on the Good day and the fifth score was unchanged.

### Isostation B-200

The OOC test reported normal back function on both test days. On the Bad day, the peak isometric strength scores were reduced by 12% in rotation, 39% in flexion, 22% in extension and 43% in lateral flexion. On the Good day, maximum velocities increased in all dynamic tests, with the exception of the second flexion-extension series. The B-200 test data was not stored for the Bad day, so it was not possible to produce a complete B-200 profile analysis. On the Good day, this individual's average velocity profiles for all tests were well within the normal band, except for the second flexion extension test at 50% resistance. During rotation, the amount of lateral flexion moment generated followed the group profile curve very closely. This was coupled with an extension moment (peak moment of 30% extensor MVC), that was well beyond the normal band, for the entire rotation test. However, during lateral flexion, the individual utilized a flexion moment, that peaked at 50% of their isometric strength. This value also exceeded the normal range (Appendix D).

### EMG - units of N·m

On the Good day, the L4/L5 moment was near the mean + 1 SD upper limit during the lifts (extensions) and lowers (flexions). For the 10 kg lowers, the moment exceeded this band. For the Bad day 0 kg condition, the L4/L5 moment exceeded the upper boundary during lifts and lowers. The addition of the 5 and 10 kg loads produced more typical patterns (Figure 46).

### Muscle Force APDFs

For the Bad day, a majority of the flexor forces were above the APDF criterion level for the lifts and lowers (Figure 47). This excessive force diminished during the loaded conditions. The RLD and LLD had excessive force during the 0 kg condition and this also decreased with the addition of the 5 and 10 kg loads. The LUES was above the criterion for all three loads, while the LLES was primarily above the criterion during the 0 kg condition. The RUES and RLES showed minimal excessive force production. For the Good day, the right and left RA exceeded the APDF criterion level. The RLD and RUES were also above the criterion for the lifts and lowers for all three loads. The LUES activity increased with loading. The average values for the APDFs across muscles showed a decrease from the Bad to the Good day (Figure 48).

### Compression

For the Good day of testing, the L4/L5 compressive forces exceeded the mean + 1 SD region for the lifts and lowers for all three loads. On the Bad day of testing, the compressive values were much closer to the mean during the lifts, but were at or above the upper boundary for the lowers (Figure 49).

### Interpretation

The VAS scores reflected the difference between the Good and Bad days better than the Oswestry low back pain questionnaire. The normal back function reported by the OOC evaluation system appears to be a combination of the conservative nature of the decision rules and the level of effort produced by this individual. The muscle force APDFs reflect an increased amount of flexor force production, which may be serving to help stabilize or splint this person. His problem is also revealed during the performance of lowers (eccentric contractions). Increased levels of muscle activity were found on the Good day (EMG-moment, 10 kg; Compression, all loads) and Bad day (EMG-moment, 0 kg; Compression, all loads). The EMG based techniques allow the upper erector spinae musculature to be included in the evaluation, providing a more thorough assessment of individuals.

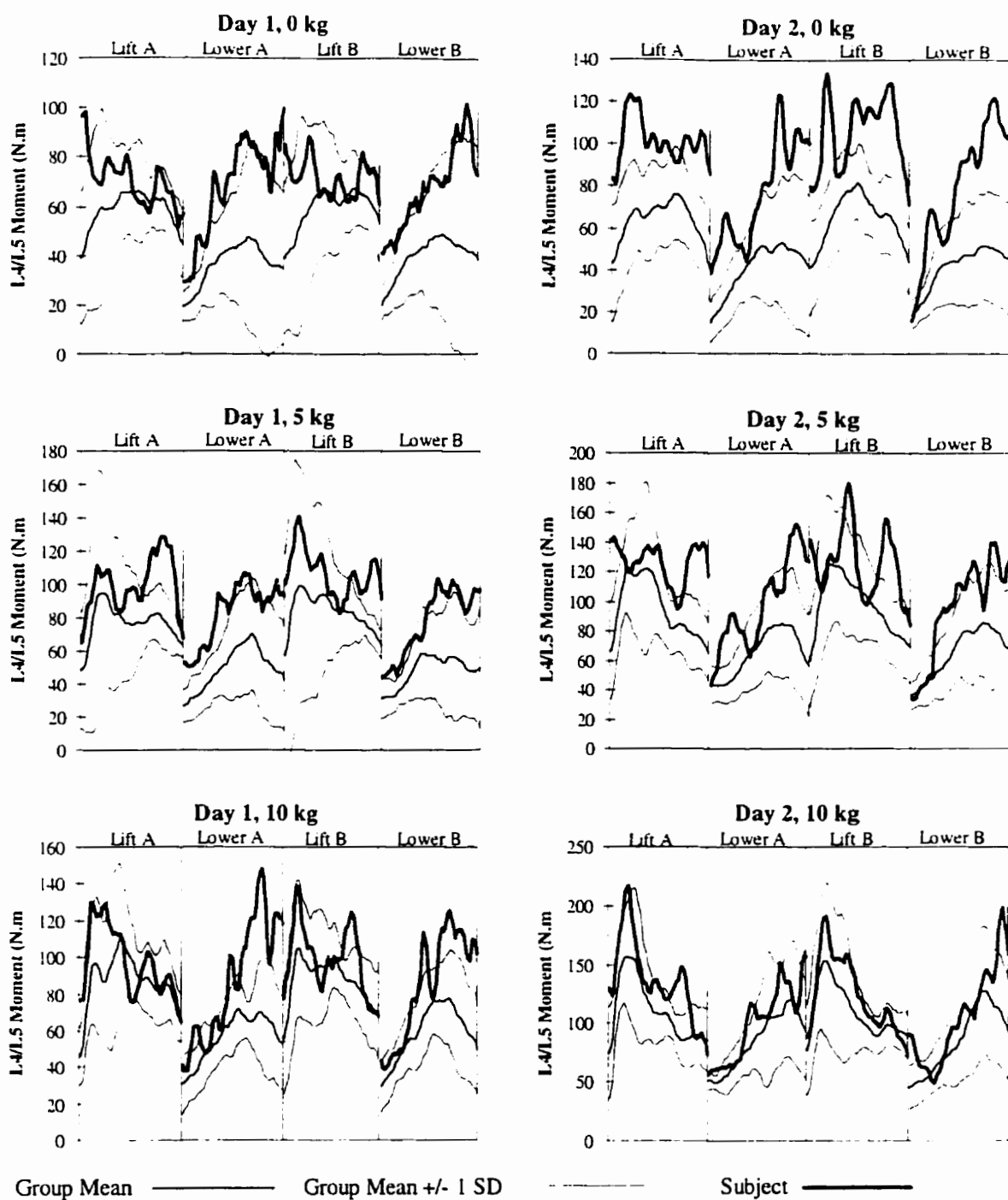


Figure 47: Case Study #2 graphed against the ASYMP mean ( $\pm 1$  SD) L4/L5 moment profile. Each graph identifies a specific day and load (N.m). Within each graph the panels indicate specific lifts (A or B extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Note: Day 2 = Bad Day.

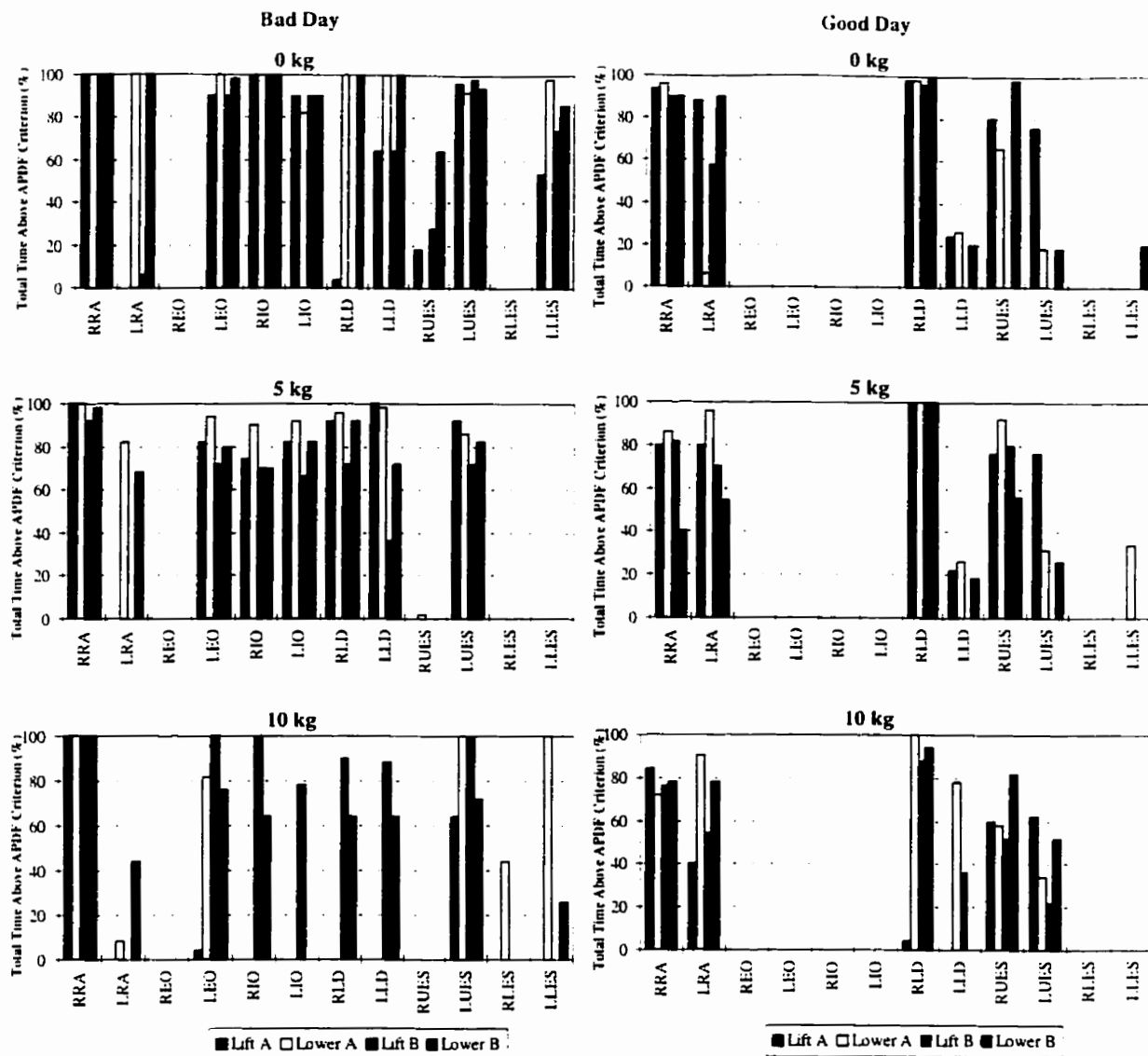


Figure 48: The time that the force for each muscle (n = 12) was greater than the ASYMP criterion force (mean + 1 SD) for Case Study #2. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae.

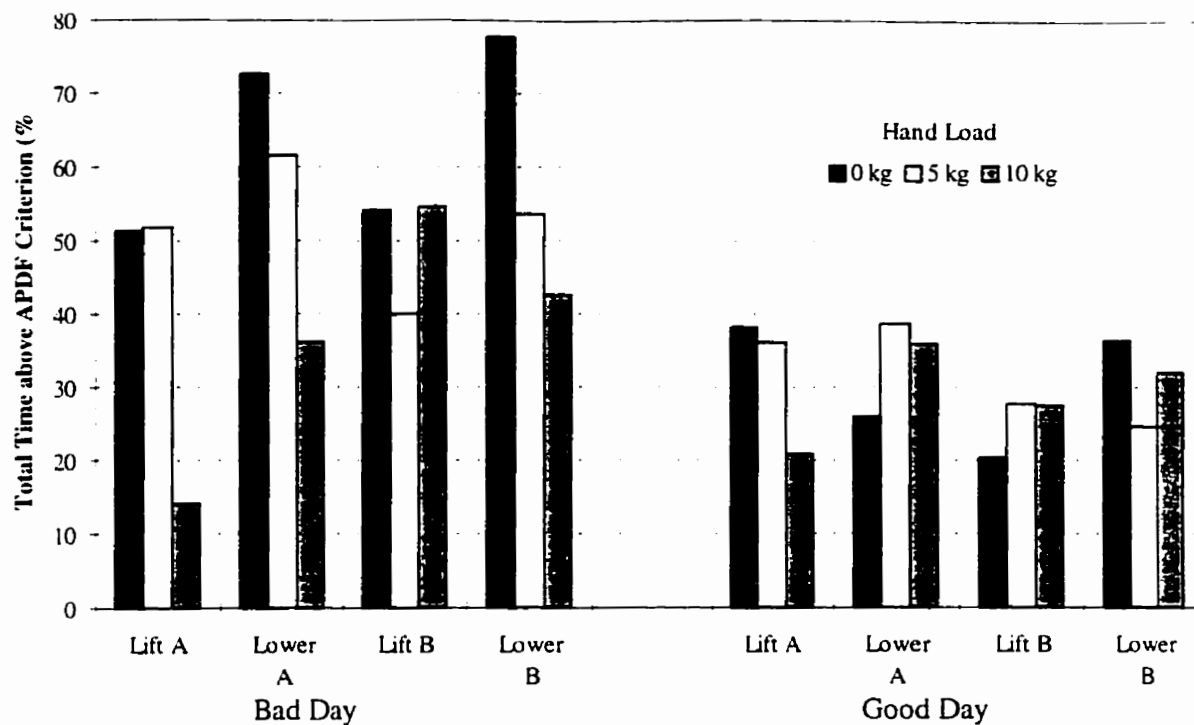


Figure 49: The cumulative time (%), for Case Study #2, in which the muscle force for each electrode site ( $n = 12$ ) was greater than the ASYMP criterion force (mean + 1 SD).

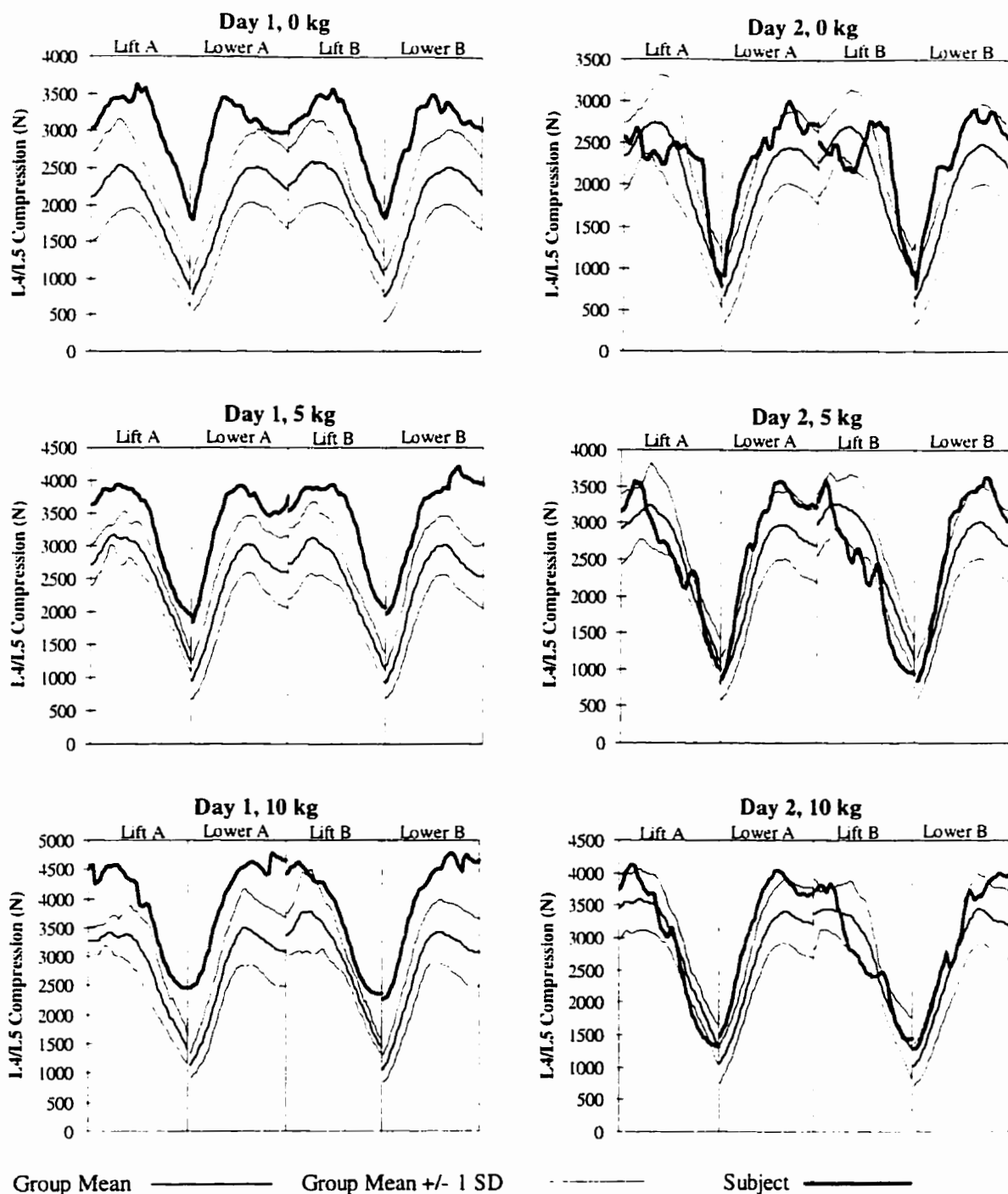


Figure 50: Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP) L4/L5 compression force (N). Each graph identifies a specific day and load. Within each graph, the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Note: Day 2 = Bad Day.

Case Study #3 This 33 year old, 1.86 m, 88.8 kg manual materials handler/truck driver suffers from myofascial pain syndrome. This includes upper thoracic spine pain and low back pain. His right side is typically worse than his left. Still performs all activities of daily living and pursues recreational activities, including goal-tending in ice hockey.

**Table 16** Comparison of “Bad” and “Good” day test results, by assessment tool, for Case Study #3. Note: no data available for the Good day, 5 kg load.

Assessment Tool	Bad Day (Day 2)	Good Day (Day 1)
Oswestry Score (%)	24	26
VAS (following each test component)	7, 10, 10, 13, 11	8, 11, 9, 12, 12
B-200 OOC Back Dysfunction	Moderate	Mild
- # of abnormal indicators	9	4
- # of non-physiological indicators	0	0
B-200 Profile - velocity	↓rot, ↓f/e, ↓lf	✓rot, ↓f/e, ↓lf
- abnormal secondary moment	12	7
- abnormal tertiary moment	6	4
Moment Profile (N·m)		
- 0 kg	lifts, above limit	within normal limit
- 5 kg	at upper limit	-
- 10 kg	at upper limit	within normal limit
Muscle Force - APDFs		
Average Time above APDF Criterion (%)		
- Lifts (0, 5, 10 kg)	11, 6, 22	25, -, 36
- Lowers (0, 5, 10 kg)	7, 13, 34	34, -, 36
Compression		
- 0 kg	within normal limit	within normal limit
- 5 kg	within normal limit	-
- 10 kg	within normal limit	within normal limit



### Pain

The Oswestry scores were 26% and 24% for the Good and Bad days, respectively, ranking the disability as moderate. This individual described his pain as being fairly severe on his Good day and moderate for the Bad day. The VAS scores also reflected this anomaly, with the pain recorded as an 8 and 7, for the Good and Bad days respectively (Table 17). A paired samples t-test found no significant difference between the VAS scores for the two days of testing,  $p < .704$  (Table 17).

### Isostation B-200

The B-200 OOC test results indicated Moderate dysfunction on the Bad day, and Mild dysfunction on the Good day. Increases in isometric strength were observed in all directions on the Good day, except for right rotation, which decreased by 5%. On the Good day (Day 1), decreased peak velocities were observed in the 25% and 50% Flexion-Extension tests and the 25% Lateral Flexion tests. There was also a decreased peak Lateral Flexion moment noted during the 25% resistance Rotation tests. On the Bad day (Day 2) in addition to these variables still being decreased there was also a decreased maximum velocity in the 25% and 50% Rotation tests, a decreased maximum Flexion-Extension moment during Lateral Flexion at 25% resistance, and a decreased isometric Flexion moment (Appendix E). For the Bad day compared to the Good day, the B-200 profile analysis showed an increase from, 6 to 12 and 4 to 7, in the number of positive responses for the secondary and tertiary axes, respectively. The increases occurred during the tests in the rotation axis. On the Bad day, there was a substantial increase in the magnitudes of the Lateral Flexion and Flexion-Extension moments produced throughout the

rotation range of motion (Table 17). Interestingly, the OOC report marked some peak secondary axes moments as being abnormally low. However, in comparing them to the B-200 profile these values were found to fall within the normal range (Appendix E).

#### EMG - units of N·m

For the Good day, the L4/L5 moments are within the normal range. On the Bad day, the 0 kg load condition showed a particularly excessive moment during lifting. The rest of the activity was within the normal range (Figure 50).

#### Muscle Force APDFs

The average time above the APDF criterion values in Table 16 are greater for the Good day than for the Bad day. On the Good day, the LRA and LIO produced excessive forces for all load conditions. The RUES, RLES, LUES and LLES also had excessive force production for lifting and lowering and this increased with load. For the Bad day, the RUES was active for all loads. As the load increased, so did the excessive force production in the RRA, LRA, RIO and LIO (Figures 51 and 52).

#### L4/L5 Compression

The L4/L5 compressive forces are within the normal range for both days (Figure 53).

### Interpretation

It is surprising that even though the subjects back felt “better” for the Good day, it was not reflected in the pain scales. Perhaps there are other intrinsic factors that this individual used in evaluating how their back felt. The OOC system reflected the difference between the two days, as the dysfunction increased from Mild to Moderate for the Bad day. The B-200 Profile reflected this change via the increased secondary and tertiary moments. Improved functioning in the Good day resulted in decreased abnormal EMG based moments, particularly for the 0 kg load. The excessive muscle forces produced on the Good day, may serve to increase the stabilization of the spine. This improved stability may be one of the intrinsic factors used in evaluating performance and may also be the source of the increased pain.

**Table 17** Summary of increased secondary and tertiary axes moment activity for OOC 50% resistance tests, dynamic sequence 1 and 2, test days 1 and 2. Excessive secondary and tertiary activity is indicated by a ✓ and ✗, respectively. For rotation tests, lateral flexion is secondary, flexion-extension is tertiary. For lateral flexion tests, flexion-extension is secondary and rotation is tertiary. The shaded areas indicate the tests for the individual's "Bad" day.

Case Study # 3	Test Primary Axis (day - sequence #)	Movement Quadrant				Totals
		1	2	3	4	
Rotation	1 - 1		✗		✗	0, 2
	1 - 2					0, 0
	2 - 1	✓	✓✗	✓✗	✓✗	4, 3
	2 - 2	✓✗	✓	✓✗	✗	3, 3
Lateral Flexion	1 - 1	✓✗		✓	✓	3, 1
	1 - 2	✗	✓	✓	✓	3, 1
	2 - 1		✓	✓	✓	3, 0
	2 - 2	✗	✓	✓		2, 1
Totals	Bad Day	2, 2	4, 1	4, 2	2, 2	12, 7
	Good Day	1, 2	1, 1	2, 0	2, 1	6, 0

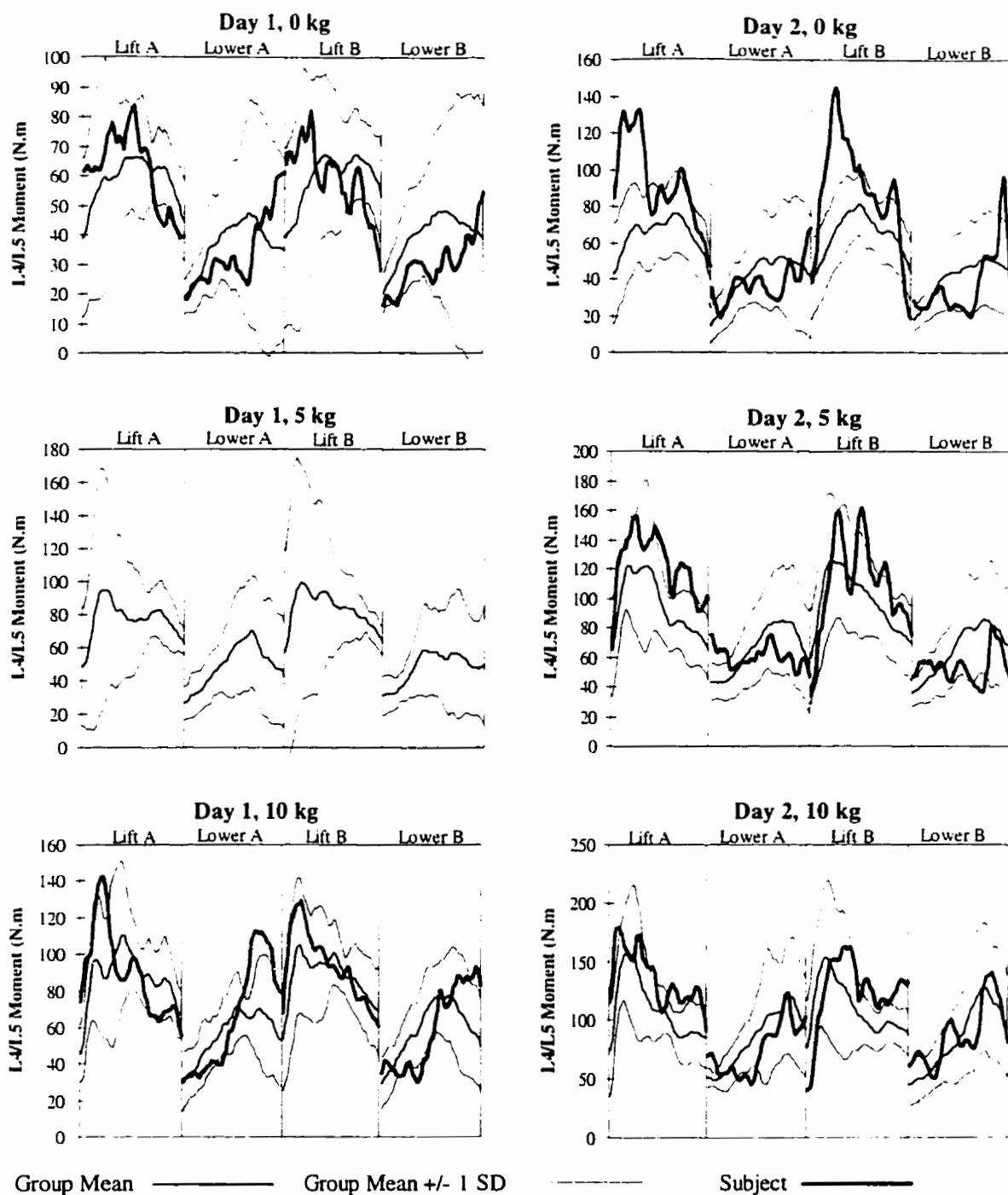


Figure 51: Case Study #3 graphed against the ASYMP mean ( $\pm 1$  SD) L4/L5 moment profile. Each graph identifies a specific day and load (N·m). Within each graph the panels indicate specific lifts (A or B extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Note: Day 2 = Bad Day and no data was available for the Good day, 5 kg load.

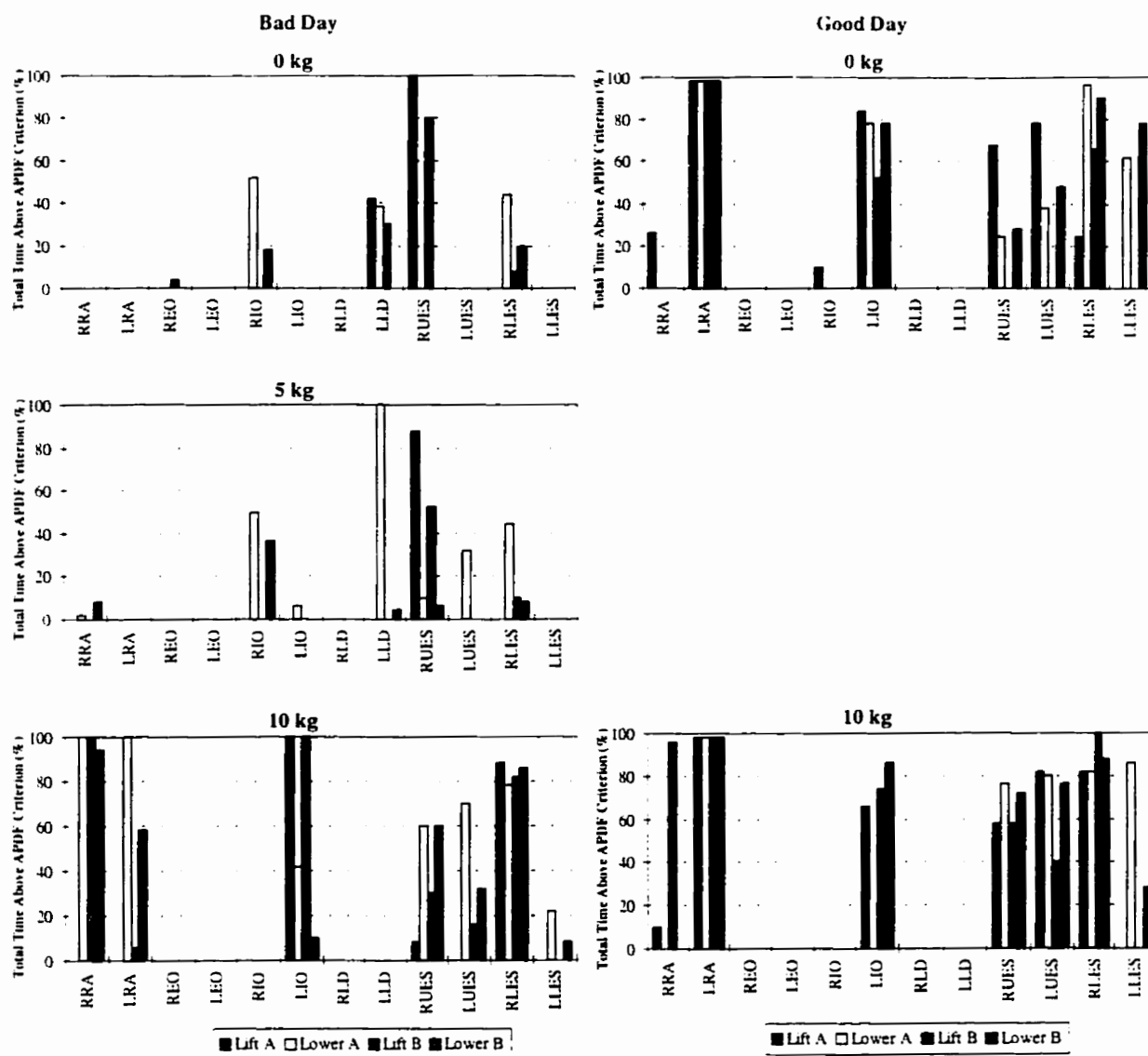


Figure 52: The time that the force for each muscle (n = 12) was greater than the ASYMP criterion force (mean + 1 SD) for Case Study #3. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: no data available for the Good day, 5 kg load.

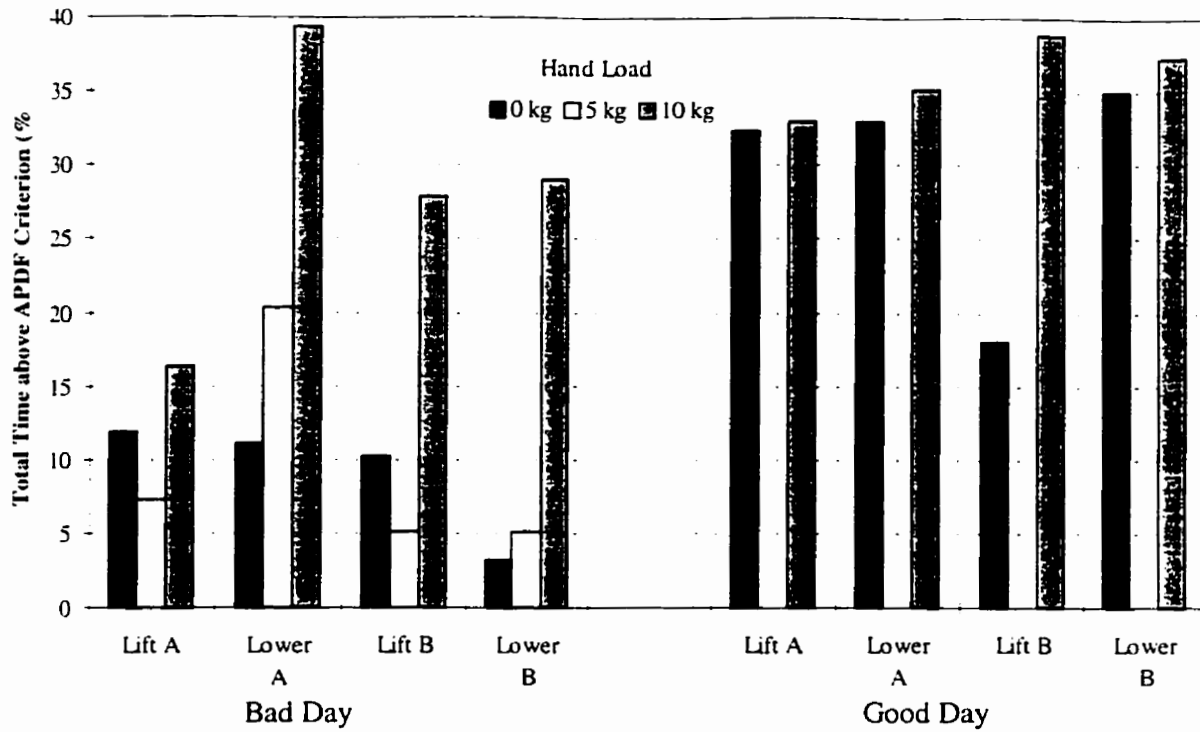


Figure 53: The cumulative time (%), for Case Study #3, in which the muscle force for each electrode site ( $n = 12$ ) was greater than the ASYMP criterion force (mean + 1 SD). Note: no data available for the Good day, 5 kg load.

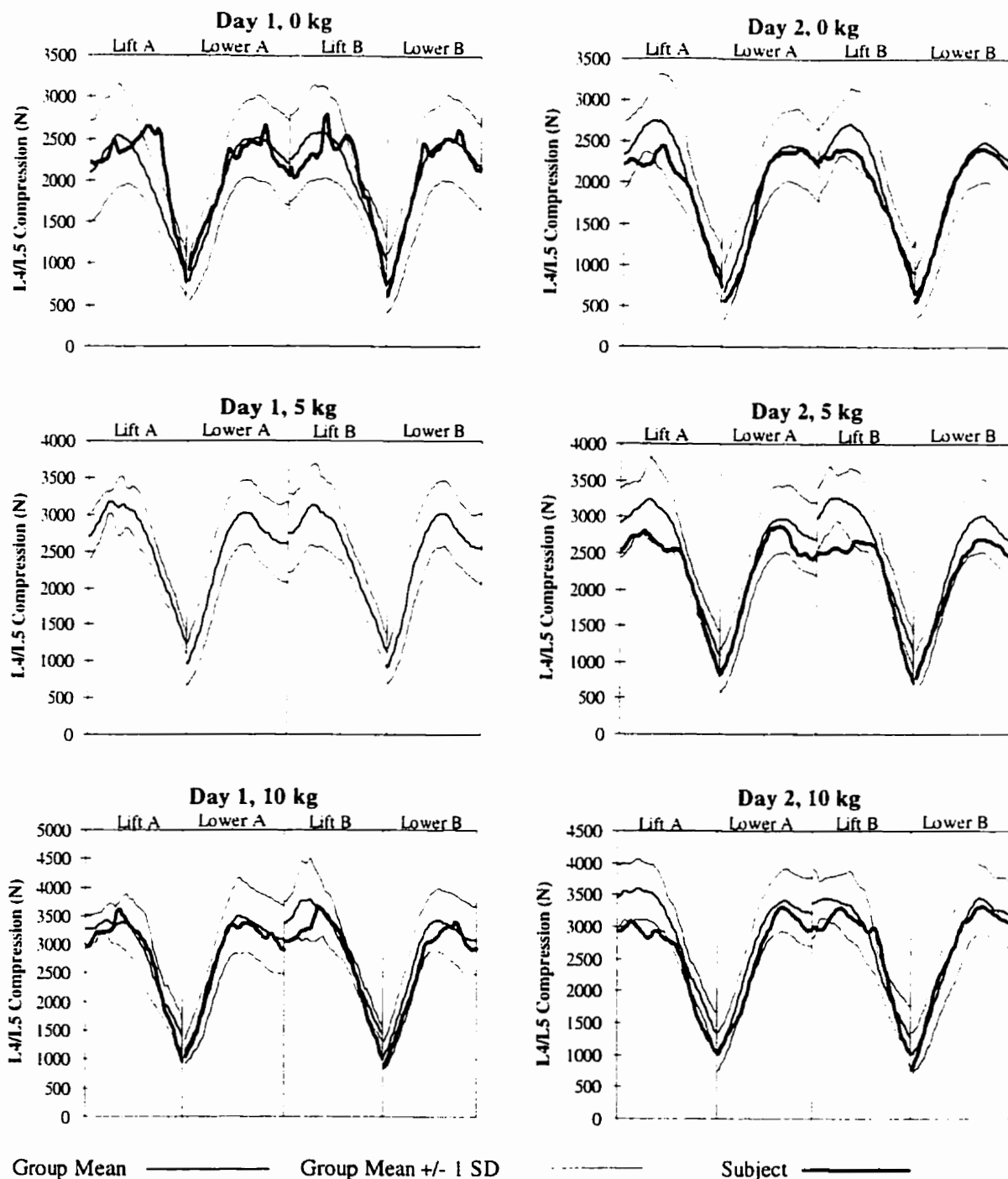


Figure 54: Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD ) L4/L5 compression force (N). Each graph identifies a specific day and load. Within each graph, the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Note: Day 2 = Bad Day and no data was available for the Good day, 5 kg load.



Case Study #4 - This 40 year old, 1.79 m, 91.4 kg massage therapist suffers from myofascial pain syndrome which includes chronic left sacroiliac dysfunction and low back pain. The left side is typically worse than the right, but he still performs all of his activities of daily living.

**Table 18** Comparison of “Bad” and “Good” day test results, by assessment tool, for Case Study #4.

Assessment Tool	Bad Day (Day 1)	Good Day (Day 2)
Oswestry Score (%)	14	8
VAS (following each test component)	5, 2, 1, 3, 1	3, 4, 3, 6, 5
B-200 OOC Back Dysfunction	None	None
- # of abnormal indicators	0	0
- # of non-physiological indicators	0	0
B-200 Profile - velocity	✓rot, ↓ f/e, ↓ lf	✓rot, ↓ f/e, ↓ lf
- abnormal secondary moment	7	7
- abnormal tertiary moment	7	8
Moment Profile (N·m)		
- 0 kg	within normal limit	-
- 5 kg	within normal limit	above upper limit
- 10 kg	lower exceeds limit	above upper limit
Muscle Force - APDFs		
Average Time above APDF Criterion (%)		
- Lifts (0, 5, 10 kg)	22, 22, 49	- , 19, 22
- Lowers (0, 5, 10 kg)	6, 22, 47	- , 28, 23
Compression		
- 0 kg	above upper limit	-
- 5 kg	above upper limit	at upper limit
- 10 kg	above upper limit	at upper limit

### Pain

The Oswestry scores were 14% and 8% for the Bad and Good days, respectively, ranking the disability as minimal. The pain at the start of the session on the Bad day was scored as moderate and this decreased to very mild for the Good day. The Good day initial VAS score of 3, was lower than the Bad day score of 5 (Table 18). However, all of the remaining scores for the Good day were greater than those of the Bad day (see Table 18). Therefore, a paired-samples t test found a non-significant difference in VAS scores ( $p < .152$ ).

### Isostation B-200

The OOC protocol rated this individual as having Normal back function for both days. Isometric strength increased in rotation and flexion. The extension strength decreased by 9% and the lateral flexion strength was unchanged. Peak velocities were greater in all tests for the Good versus Bad day (Appendix F). The B-200 profile reflected this improvement in both the rotation and flexion-extension axes. However, despite the increased velocity, the lateral flexion profile still remained below the normal range. The B-200 profile produced the same number of positive responses, 7, for the secondary axes and an decrease from 8 to 6 for the tertiary axes for the Good and Bad days, respectively. However, there was a shift in the movement as to where the increased secondary and tertiary moments were produced. For the Good day, the positive responses were produced at the start and end of the repetitions, Q1 and Q4, respectively. For the Bad day, the positive responses occurred in the middle of the repetitions, Q2 and Q3 (Table 19).

### EMG - units of N·m

On the Bad day, the EMG-to-moment profile for the 0 kg load shows a decreased moment during the lift and an average pattern during the lowers. The magnitudes increased with loading, resulting in an excessive moment being produced when lowering the 10 kg load. Also, the moments during the lowers peaked earlier than normal profile. For the lifts, the peak moments occur later than in the normal profile. On the Good day, excessive moment was produced during the lifts and lowers, for all loads. The pattern of moment production is similar to that of the Bad day. For the lowers, this occurs at the start of the cycle and for the lifts it occurs at the end of the movement (Figure 54).

#### Muscle Force APDFs

On the Bad day, there is an increase in the force produced by the RRA, REO, RIO and RLD during the load handling. Increasing the load resulted in increased activity in the RLES and LLES. On the Good day, the RRA, RIO, LEO, LIO and LLES showed excessive force production (Figure 55). The cumulative total time above the APDF criterion decreased from the Bad day to the Good day (Figure 56).

#### L4/L5 Compression

On the Bad day, the L4/L5 compressions exceeded the normal range at the onset and during the terminal portion of the lifts. The peak compressive values were also greater for the lowers. For the Good day, the lifts matched the normal profile in both magnitude and timing. The lowers marginally exceeded the upper boundaries (Figure 57).

### Interpretation

The initial VAS scores reflected the improvement between the two days. However, each of the test components was scored as being more painful on the Good day. This may have been a function of increased effort, as both the B-200 and prone MVC's were greater for the Good day. Both the OOC and B-200 profile systems documented the increased velocity of movement on the Good day. The B-200 moment profiles reflected the change in how the performance was occurring. The EMG based measures showed the difference in the magnitudes and patterns of utilization, especially for the 10 kg load. The differences in the moment profiles may reflect this particular person's style of lifting. The increased flexor activity on the Bad day may indicate an increased need for stabilization .

**Table 19** Summary of increased secondary and tertiary axes moment activity for OOC 50% resistance tests, dynamic sequence 1 and 2, test days 1 and 2. Excessive secondary and tertiary activity is indicated by a ✓ and ✗, respectively. For rotation tests, lateral flexion is secondary, flexion-extension is tertiary. For lateral flexion tests, flexion-extension is secondary and rotation is tertiary. The shaded areas indicate the tests for the individual's "Bad" day.

Case Study # 4	Test Primary Axis (day - sequence #)	Movement Quadrant				Totals
		1	2	3	4	
Rotation	1 - 1	✓ ✗			✓	2 , 1
	1 - 2	✓ ✗			✓ ✗	2 , 2
	2 - 1		✓ ✗	✗	✗	1 , 3
	2 - 2		✓		✓	2 , 0
Lateral Flexion	1 - 1		✓	✗	✓	2 , 1
	1 - 2		✗	✓ ✗		1 , 2
	2 - 1	✓ ✗	✗	✓	✗	2 , 3
	2 - 2	✗		✓	✓ ✗	2 , 2
Totals	<b>Bad Day</b>	2 , 3	1 , 1	1 , 2	3 , 1	7 , 6
	<b>Good Day</b>	1 , 2	2 , 2	1 , 1	3 , 3	7 , 8

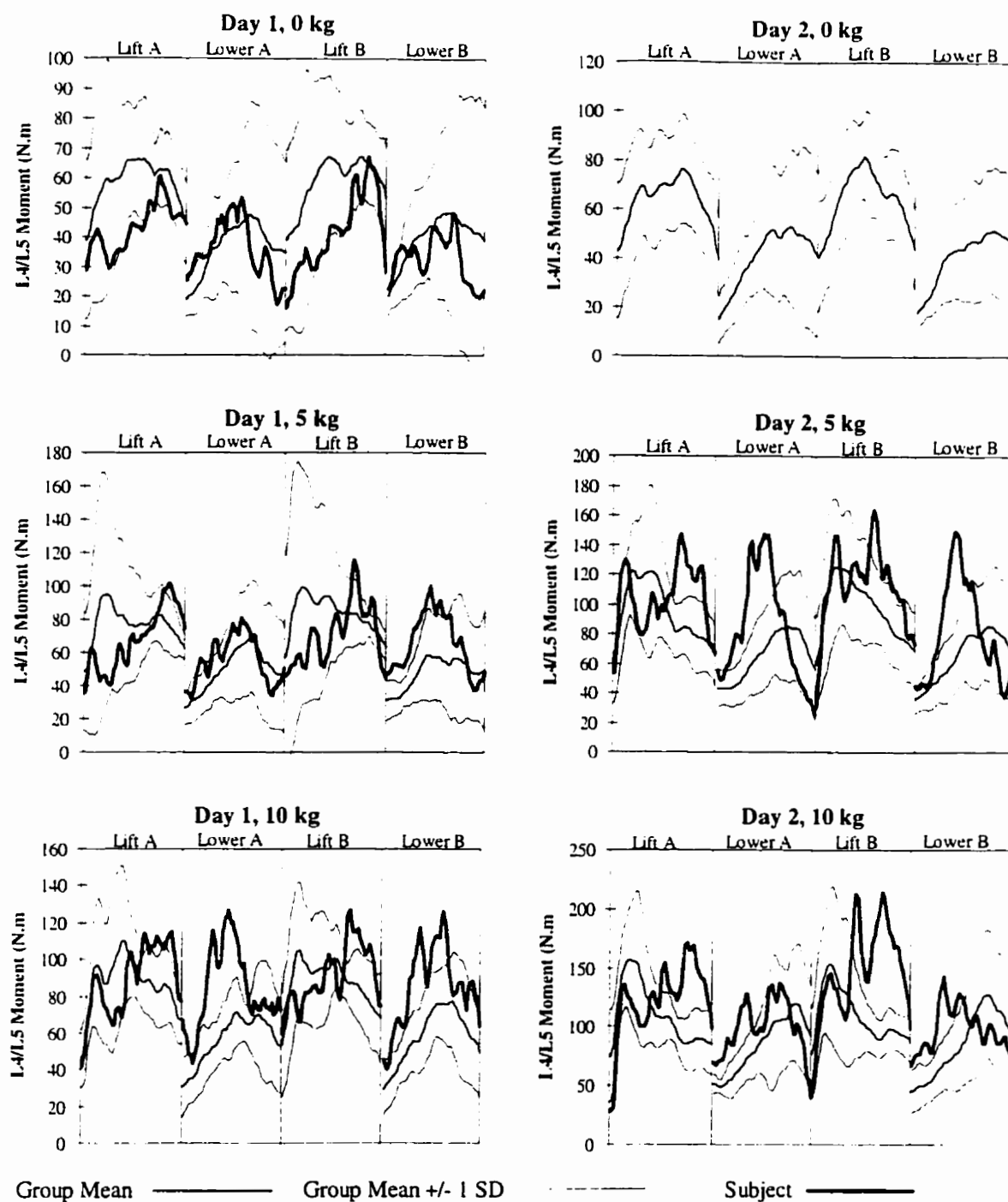


Figure 55: Case Study #4 graphed against the ASYMP mean ( $\pm$  1 SD) L4/L5 moment profile. Each graph identifies a specific day and load (N.m). Within each graph the panels indicate specific lifts (A or B extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Note: Day 1 = Bad Day and no data was available for the Good day, 0 kg load.

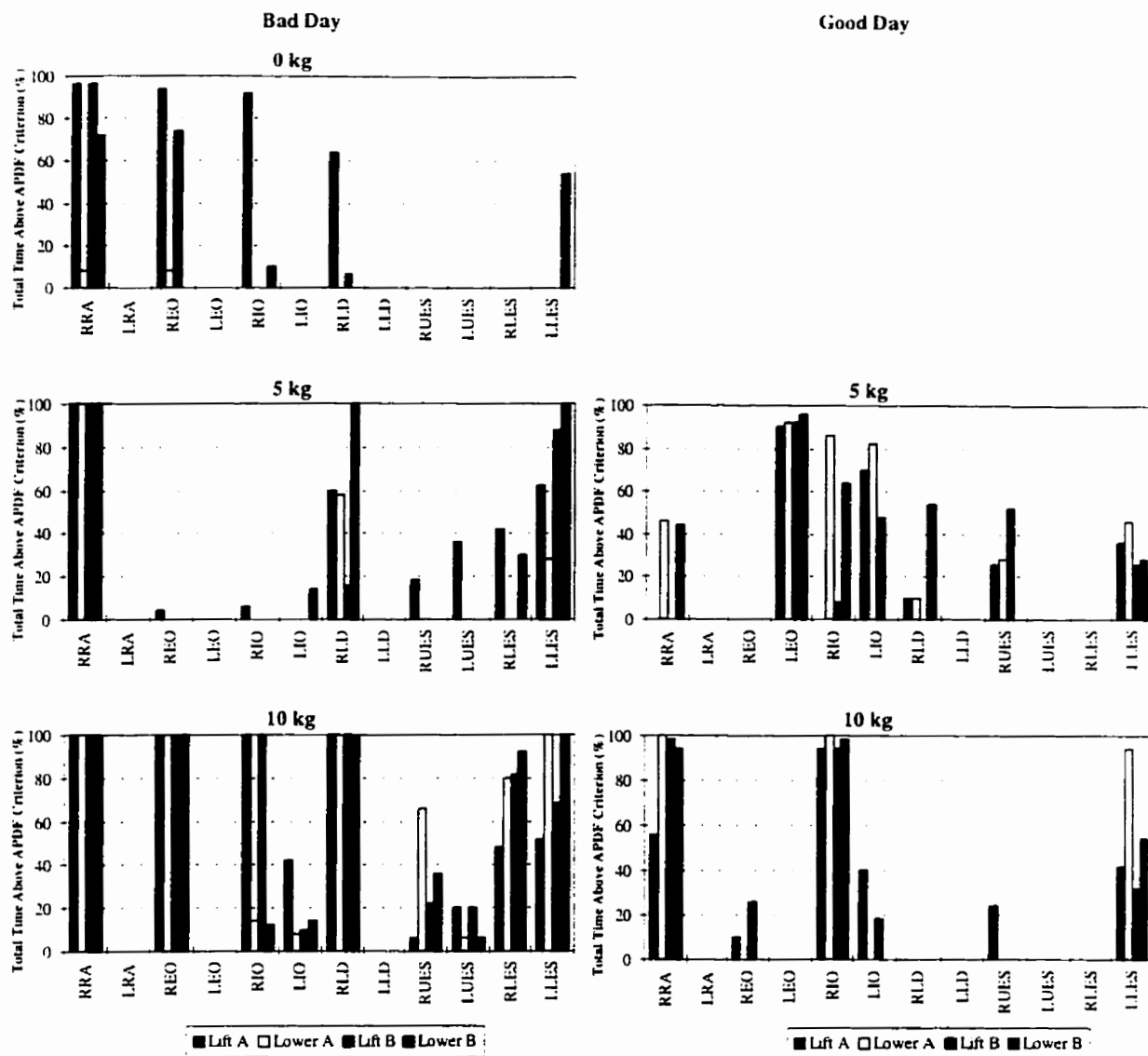


Figure 56: The time that the force for each muscle (n = 12) was greater than the ASYMP criterion force (mean + 1 SD) for Case Study #4. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: no data available for the Good day, 0 kg load.

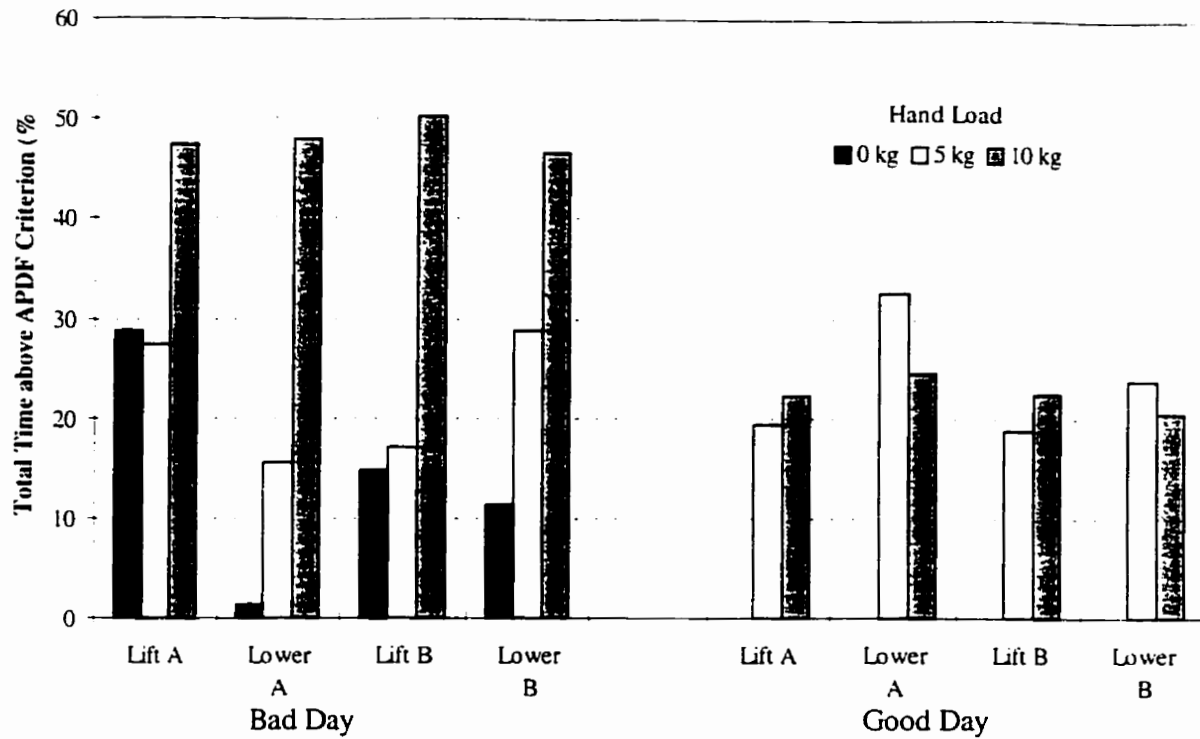


Figure 57: The cumulative time (%), for Case Study #4, in which the muscle force for each electrode site ( $n = 12$ ) was greater than the ASYMP criterion force (mean + 1 SD). Note: no data available for the Good day, 0 kg load.

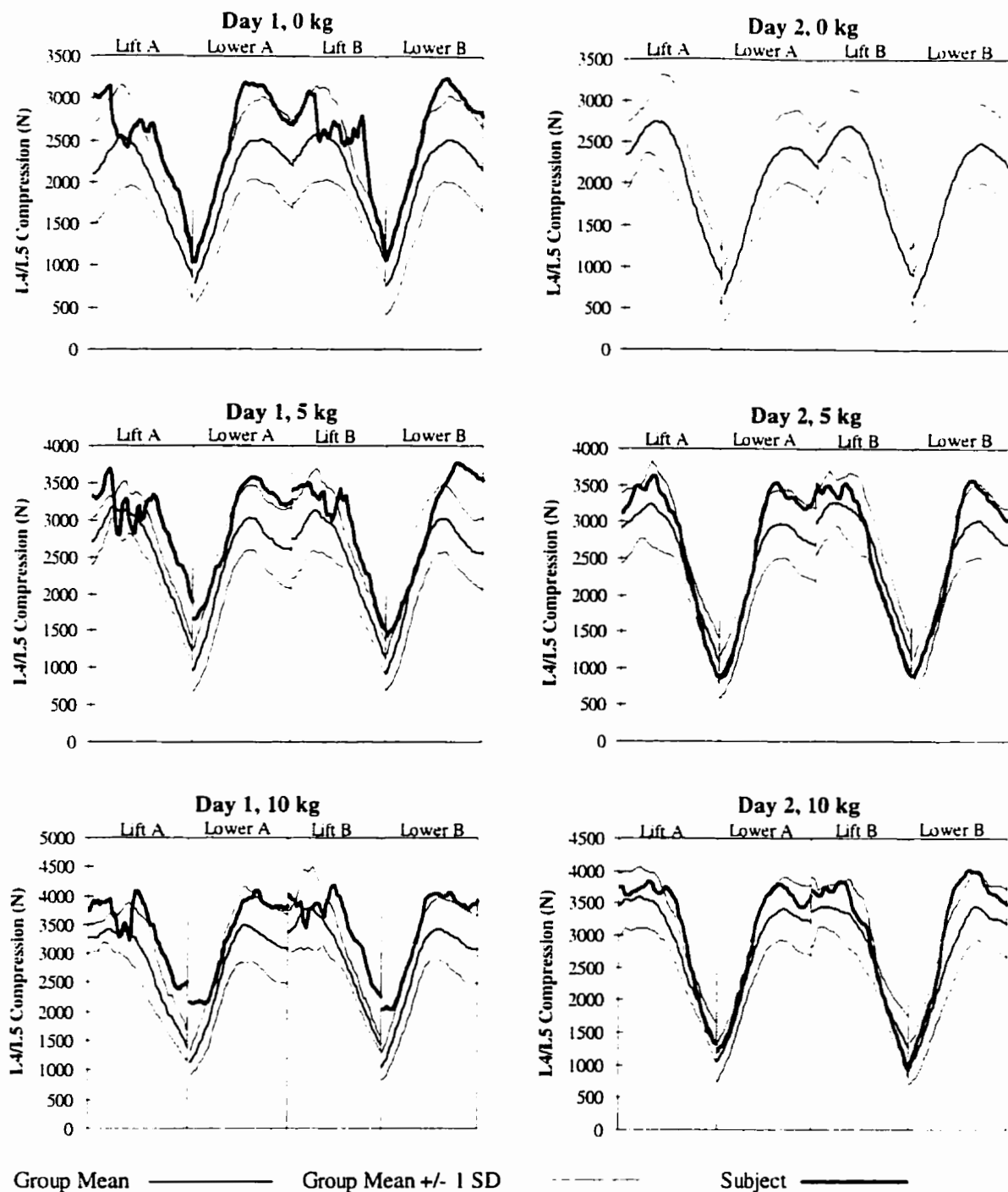


Figure 58: Case Study #4 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP) L4/L5 compression force (N). Each graph identifies a specific day and load. Within each graph, the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Note: Day 1 = Bad Day and no data was available for the Good day, 0 kg load.



## Discussion

The purpose of this study was to determine whether the understanding of spine movement dysfunction, as indicated by abnormal displacement, velocity and torso moment data measured by a clinical evaluation system, was augmented by a flexion-extension task and the knowledge of EMG activity and/or by knowledge of individual torso tissue force estimates from an EMG assisted, dynamic three dimensional spine model. Pain scales, custom Isostation B-200 movement profiles, EMG of the spinal musculature and an EMG assisted model of the lumbar spine were found to provide more information regarding the low back function of the individuals symptomatic and asymptomatic for low back pain (LBP) that volunteered for this study.

Individuals with recurrent low back pain were targeted as the specific symptomatic population for this study. These individuals have a strong feel for their back status. They have periods where their back dysfunction impinges minimally on their daily routine, followed by episodes, typically of predictable recurrence, which limit their activities and their daily routine. However, during the periods of recurrence, they know they will reach a state where their back dysfunction will once again be in remission. This made these individuals ideal candidates in which to investigate differences in spine movement dysfunction. Another advantage of this group is that these were not first time injuries and they would not be expected to become chronic. Prolonged or chronic back pain (i.e. greater than 3 months) is complicated by psychological concerns and secondary gain issues (Spitzer et al., 1987, Gatchel, et al., 1995). These factors should be minimal in the groups used in this study. Also, Spitzer et al. (1987) found that recurrent episodic cases should be treated by an approach used for acute cases. Therefore,

techniques or results produced by studying these recurrent cases would be reasonable starting points for future work with acute cases.

In this study, the symptomatic individuals were tested on days they identified as a Good day and a Bad day. Their pain status was evaluated using the Oswestry low back pain disability questionnaire (Fairbanks et al., 1980) and a Visual Analog Scale (VAS). The functional daily differences were not reflected by the overall Oswestry rating and the VAS only differentiated one of the individuals. This may reflect the nature of the measurement scales and the participants in this study. The Oswestry questionnaire classifies disability using the following categories and scores: Minimal Disability (0-20%), Moderate Disability (20-40%), Severe Disability (40-60%) and Crippled (60-80%). Scores of 80-100% indicate a person is bed-bound or exaggerating their symptoms. These scores are based upon the self reported amount of pain felt at the time of testing and the ability to perform in nine areas related to activities of daily living. The visual analog scale (VAS) is used as a graphic indication of pain by marking a 10 cm line.

Using the Oswestry scores, two of the individuals were classified as having "minimal disability" on their Bad day. Therefore, they could not improve their classification per se, on the Good day. Yet on the Good day, one score decreased from 14 to 8% and the other remained at 11%. For the two individuals with moderate disability, one score decreased from 26 to 24% for the Good day, but the other score *increased* from 24 to 26% for the Good day. The differences in both scores reflect changes in the pain levels on the Good day (i.e. Case 3's pain increased). The responses to the other nine sections were almost identical between days.

Likewise, the VAS scores showed a significant improvement for only one subject (Case #1). The VAS also revealed that Case #3 rated their pain at the start of testing greater on the

Good day then on the Bad. Given that the symptomatic individuals felt that they had improved function, but the rating instruments failed to reflect this, there must be other intrinsic and extrinsic factors besides pain (e.g. stability) and the performance of activities of daily living, that are evaluated by individuals when evaluating their functional state.

The clinical evaluation system utilized in this study was the Isostation B-200 and the OOC software system (Version 3.1). Two reported strengths of this system are its ability to comprehensively evaluate back function and monitor changes that occur throughout treatment (Deutsch, 1991). In this study, the system produced reports of normal back function, on both test days, for three of the four cases. The moderate dysfunction on the Bad day for the other case (Case #3) improved to mild dysfunction on their Good day.

There are several possible explanations for this anomaly. Perhaps the three cases truly have normal back function. However, it seems unlikely that this was just an increase in symptoms not associated with some type of back dysfunction. Also, this was not the first time that these individuals had been symptomatic for low back pain and they had actively sought treatment from a chiropractor and received relief during previous LBP episodes.

Another explanation may lie in the system's very conservative scoring of abnormal performance. Before being considered as an indicator of back dysfunction, the isometric and secondary axes strength values must fall below the 2.5<sup>th</sup> percentile level in the comparison database. The peak velocity parameter must fall below the 5<sup>th</sup> percentile. For the secondary axes moments and the peak velocity parameter values, this requirement must occur in both of the dynamic test sequences, for the same resistance (Deutsch, 1991). Perhaps the recurrent LBP participants in this study, who were accustomed to their dysfunction and used to performing

activities with it, were not as limited as might be expected. Or perhaps the peak parameters were within the normal range, but a majority of the time histories were abnormal.

The number of individuals in the normal comparison database may be a factor. The OOC software (Version 3.1) compares an individual's test results to a database of performance scores for 62 people, matched for gender. Although the data base represents a wide range of ages, heights and weights (Deutsch, 1991), there may not be a sufficient number of people within the database to produce a Gaussian distribution or sufficient variability. Thus, a small change in a performance parameter may be enough to shift the scoring of a result from abnormal to normal.

Finally, the underlying approach to scoring the data may contribute as well. One of the strengths of the OOC system is that relative percentages are used for each individual in determining the primary axis loads for dynamic tests. However, in the scoring of the peak secondary axes moments, the absolute magnitude of the peak moment is used. This creates two problems. By expressing the moment in absolute terms, a person may be classified as having a low secondary/tertiary axis peak moment, but relative to their own isometric strength they may be producing a typical peak secondary/tertiary moment. This would represent a Type I or False Positive error. Secondly, a single number (i.e. the peak) may indeed be in the normal range, while a majority of the time history is beyond the boundary. This reflects a Type II error or False Negative.

The B-200 profile system developed in this study attempted to address these issues. The single page graphical results facilitates the analysis by providing an overview of the velocity, secondary and tertiary moment parameters for each test. However, this technique is still labor intensive because a separate summary page is produced for each of the twelve dynamic tests that

are conducted in an OOC evaluation. Therefore, a scoring system was developed to facilitate the B-200 profile analysis even further. This system used a less stringent criterion ( $\pm 1$  SD) than the OOC software (2.5 and 5.0 Percentile) and concentrated on the primary axis velocity and the secondary axes moments produced during dynamic testing. Using this system, performance differences from the normal profiles and changes between the Good and Bad day tests were quantified for the three subjects that had complete raw data files. In comparing the differences between days, one individual's results shifted more towards normal, another stayed the same and the third one shifted further from normal. One of the major advantages of the B-200 Profile analysis system developed in this study is that the ensemble average for the middle three repetitions is the data that are analyzed. This prevents spurious peaks from biasing test results. Also, the moment variables are expressed relative to the isometric strength that an individual could produce in each of the effort directions. This further highlights any left or right sided asymmetries that an individual may have, as illustrated in this study.

The profiles presented here must be interpreted and generalized with caution. The Isostation B-200 was certainly a novel testing experience for the participants. With the pelvis restrained, a significant mass strapped to the upper trunk and the off-plane axes "locked" at maximal resistance, it was assumed that each participant would produce "natural" motions while moving as hard and as fast as they could. The resulting performance reflected both the "natural" muscular recruitment pattern and an individual's efforts in learning to deal with constraints imposed by the dynamometer and the test protocol.

Other researchers have published normal values for Isostation B-200 parameters (Levene et al., 1989; Gomez et al., 1991; McIntyre and Glover, 1993). However, these studies have

utilized peak and average parameter values for describing and comparing populations. Szpalski et al. (1996) have incorporated ensemble averaging of the velocity data for 50% resistance flexion-extension tests in trying to document differences between normals, individuals with spinal stenosis and posterior bulging discs. Instead of analyzing the entire flexion and extension velocity curves, they used four points from within each motion. When the flexion and extension velocities were normalized to the peak flexion velocity, these authors found significant differences in the motion patterns among all three groups. Although they only investigated velocity in one axis, these authors felt that diagnosis-specific spinal movement signatures do exist. If this is indeed the case, then the system developed in this study should facilitate in defining the signatures even further.

This study also incorporated a flexion-extension task designed to determine if EMG activity could augment the understanding of low back dysfunction. The EMG profiles for the ASYMP group were normalized to be expressed in terms of mV and %MVC. However, inspection of the SYMP EMG profiles plotted against the ASYMP EMG profiles showed the method did not augment the analysis for the SYMP group because the curves for the SYMP individuals typically fell within the mean  $\pm 1$  SD range. Of the twelve electrode sites, the upper erector spinae site consistently showed the most excessive activity. For the 10 kg trials, the variability ranged from 20 - 25% MVC at the time of peak extensor activity. Sutarno (1993) found similar patterns and peak magnitudes for %MVC normalized EMGs, for spinal flexor and extensor muscles of males performing a similar flexion-extension task with 0 and 10 kg loads. However, the variability was much smaller. For a 10 kg load, the standard deviation was less than 6.5% at a peak extensor activity of 40% MVC. The specific nature of the task is one factor

which affects the magnitude of the variability. Although Sutarno's (1993) subjects performed a flexion-extension task, the lifting posture and type of lift (stoop) were much more controlled than in this study. When the subjects speed of movement was paced, the variability was reduced even further. During a fully flexed, static posture, the upper erector spinae electrode of McGill and Kippers (1994) had a standard deviation of 8.9%, while Callaghan and McGill (1995) found variability of over 10% MVC for static postures that required similar thoracic and lumbar spinae activation levels as in this study.

Another reason for not using the trials normalized to % MVC was illustrated by the SYMP group in which the difficulty in trying to obtain a true MVC was apparent. Producing an effort less than 100% resulted in the rectus abdominis contraction level exceeding 10% MVC (Figure 27). Whether fear of re-injury, pain or decreased motivation, the ASYMP group did not appear to produce true MVCs. Performance of a MVC is a skill and obtaining a maximum value may require numerous test positions (McGill, 1991) and even then a researcher cannot be confident that a true maximum was obtained.

Combining the EMG and the link segment model together to produce a signal calibrated in N·m was an attractive alternative to the EMG profiles because it provided a calibrated signal which did not require MVCs. In this study, the average RMS differences across all loads and days were 41.5% and 49.8% for the thoracic and lumbar electrode sites, respectively. Potvin et al., (1990) calibrated the EMG signal to represent the L4/L5 compressive force and found RMS differences of 39% and 58% using a thoracic and lumbar electrode site, respectively. Wells et al., (1997) also used this approach as a means of estimating exposure in the work place. However, in the present study, calibrating the EMG signal to estimate the L4/L5 reaction moment produced

equivocal results. Changes between the Bad and Good day test sessions, which could be attributed to improved functionality, were observed for some of the symptomatic individuals. However, the variability present in the normals minimized the utility of these profiles. The N·m calibrated EMG method was advantageous because a single output was produced which incorporated information from two of the most active spinal muscles for the flexion/extension task used in this study. This technique could very easily be applied to a variety of tasks. It requires minimal hardware and software, making it a useful instrument for use in a clinical environment. This method could also be useful as an educational tool in industry, demonstrating the different muscular loading responses that occur with different lifting styles and postures.

The assessment with muscle forces estimated via the computerized spine model augmented the analysis of spine movement dysfunction the most. This method corrected for the normalization difficulties encountered when expressing the EMG in units of mV or %MVC. The ability to observe which of the flexor and/or extensor electrode locations resulted in excessive muscle forces allowed a more detailed assessment of the performance to be made. However, the twelve electrode sites, three loads, two lift/lower cycles and two test days made understanding and interpreting the comparison profiles in order to pinpoint problematic muscles difficult.

The Amplitude Probability Distribution Function (APDF) was incorporated as a method through which the amount of time in which muscles had excessive force generation could be quantified. This produced a two step analysis for the SYMP group. The first was a breakdown by day, task, load and electrode location (e.g. Figure 43). This graphical summary of the APDF identified both the muscle groups and the effect of the load in the hand on excessive muscle force production. They also illustrated that for the flexion-extension task utilized in this study, the



eccentric phase appeared to be more important in documenting differences in recovery because more excessive force production was found to occur during these activities (see Figure 44). The second part of the analysis was the overall summary for each day and load which quantified the percentage of time that the muscle forces were above the APDF criterion level (see Figure 44). Using this approach, changes in excessive muscle force production were quantified for each SYMP individual in this study. The importance of this result is underscored by the fact that the ASYMP group was only “mildly” abnormal as indicated by the Oswestry and VAS scores. On the Good day, three of the cases (Case #1, 2, 4) had decreases in the amount of excessive force production. The other individual showed an increase in excessive force production. This person also had increased pain on this day, despite reporting their back function to be improved.

These results illustrate that decreased pain may not necessarily reveal improvements in function and they underscore the importance in assessing function and not focusing solely on pain. Functionality may depend upon a number of intrinsic and extrinsic factors. These results also highlight a possible limitation in studying a recurrent low back pain population. As their functionality improves, these individuals perform a greater number of, and more demanding, activities. This provides a greater opportunity for an increase in pain or perhaps even a relapse. The Cases in this study had full-time responsibilities and were typically tested at the end of their work day. So even though they may have felt functionally improved, there may be an increased cost (e.g. pain) associated with being able to participate in or perform more activities.

One of the difficulties with the APDF method is the loss of timing information (i.e. when did the excessive forces occur?). This limitation may be overcome by breaking the specific tasks

into discrete movement phases. This would allow specific periods to be identified and focused upon. However, it does increase the number of events that would need to be analyzed.

Another drawback to the criterion strategy used in this study is that the magnitude of the excessive force production was not considered. Thus two individuals with different force profiles would produce identical evaluations. For example, one individual may have a muscle force profile which was only slightly greater (e.g. 1 N) then the criterion level for the entire time history and another person may have a profile which was much greater (e.g. 100 N) then the criterion level for the entire time history. Both of these individuals would be identified as having excessive force production for the entire time history, but the difference in magnitude would be ignored. Also, an individual may have identical force-time histories for a right and left electrode site, but the "normal" APDF criterion level profiles (mean + 1 SD) may be different for the right and left electrode sites. This could result in one muscle being to the right of the criterion and be considered to be producing "excessive" force while the other which was to the left of the criterion would be considered normal. Integrating the area between the "normal" APDF criterion curve and an individual's APDF curve would allow the excessive muscle force production to be quantified and it could be normalized to the entire area encompassed by the total APDF criterion.

The spine model used in this study also estimates the L4/L5 compressive force and this parameter was incorporated into the SYMP profile analysis. Although the overall L4/L5 compressive force profile was a one page analysis, the variability in the range of compressions (200 to 900 N) introduced by the absence of the specific lifting/lowering instructions did not differentiate the SYMP individuals from the ASYMP individuals. However, because compression is one of the few tissue tolerance standards that are available, its inclusion is

worthwhile as it provides insight regarding the relative risk of injury (NIOSH, 1981). The assessment of compression estimates from the model are also advantageous because the effects of muscular co-contraction are also reflected. The L4/L5 compression estimates would also be useful in demonstrating to individuals how their particular movement profile affects their relative risk of injury for the performance of a particular task.

In this study the participants performed a freestyle, self paced flexion-extension task. This allowed each SYMP individual to perform the task so they could make accommodation for their particular injury, either by avoiding a problematic posture or moving in a manner which minimized pain. The resulting assessments measured these accommodations by quantifying deviations from normal. However, it would have been possible to incorporate a task which involved a constrained movement posture and/or pace. This type of task may also have produced responses which deviated from normal because the constraints exacerbated a painful condition. However, this method increases the risk of injury aggravation and individuals may elect not to perform the task at all. Incorporating a combination of freestyle and constrained tasks would be an approach which would result in a very thorough assessment of spine movement dysfunction.

The conclusions of the current study must be considered in light of the assumptions and limitations inherent in the research design and methodology. The results are specific to the two populations utilized in this study. Although an effort was made to incorporate older ASYMP individuals so that they would better reflect the anticipated SYMP group, the use of a university based student population made this challenging. Increasing the number of individuals in each group would have been advantageous and helped to decrease the variability for the ASYMP group profiles. In this study the normal range was defined as the mean  $\pm$  1 SD. Statistically, this

range only incorporated 68% of the “normal” population, leaving 16% to fall above this range and 16% to fall below this range. Increasing the range to include the mean  $\pm$  2 SD would have decreased the number of occurrences where spine movement dysfunction was documented, particularly for the B-200 profile system. However, for the muscle force profiles the conclusions would not have been altered because the symptomatic individuals muscle force production was excessive for so many of the EMG electrode sites. Due to the planar nature of the flexion-extension task, kinematic symmetry was assumed between the right and left sides. However, this would not have significantly altered the magnitude of the muscle force estimates.

The use of surface EMG meant that not all of the muscles that forces were estimated for could be monitored (e.g. psoas), requiring the EMG time history from synergistic muscles to be used (e.g. internal oblique for psoas). This assumption is supported by data from McGill et al. (1996) who found that well selected surface electrode locations did provide a representation of deeper muscles with RMS differences of 2-15% MVC found during the performance of clinical tasks. The approach of using synergistic EMG time histories in symptomatic individuals may produce abnormal results in and of itself. For example, if the right internal oblique muscle had abnormal function or timing then the right psoas muscle fascicles would also be credited with abnormal function or timing, producing an incorrect assessment. Likewise, if the right internal oblique was functioning normally but there was a problem with one of the psoas fascicles, then it would not be identified because the right internal oblique time history was used to partition the muscle force. Therefore, one must be cautious in the interpretation of dysfunction.

Some researchers have used optimization as a method to assist in muscle force partitioning. However, this technique is insensitive to both EMG electrical silence and the

different load sharing strategies that occur between people. As previously mentioned, the  $EMG_{Force}$  method for calculating the EMG scaling factors for the model produces underestimates of the L4/L5 compressive forces. Modifications in the strategy used to collect the scaling factors would help to minimize this difference.

This study utilized EMG and an EMG assisted model of the lumbar spine in conjunction with a flexion extension task to try and improve the understanding of spine movement dysfunction as indicated by a clinical evaluation system. A more thorough analysis of the data produced by a clinical evaluation system enhanced the assessment of the SYMP group. The estimation of muscle forces for the symptomatic individuals documented that changes in muscle performance are correlated with improvements in function. In future research, it would be beneficial to incorporate tasks (e.g. asymmetrical loading) that would concentrate on specific muscles sites. This may further isolate dysfunctional muscle performance.

### **Conclusions**

1. The APDF presentation technique provided a visual summary of the muscle force data that immediately highlighted the intra-individual differences between loads and days, and the inter-individual differences between each symptomatic case and the asymptomatic population. The APDF method also qualitatively indicated for the symptomatic individuals which of the flexor and extensor electrode sites were most affected for each load and test day.

2. The “percentage of time above the APDF criterion level” calculation quantified the intra- and inter-individual differences in the flexor and extensor muscle forces for each of the electrode locations, hand loads and test days. Expressing the EMG in units of mV or %MVC did not distinguish these differences.
3. Improved functionality, as indicated by decreases in Oswestry and visual analog scale scores, was typically (3 out of 4 cases) associated with decreases in excessive spinal flexor and/or extensor muscle force production.
4. The eccentric (flexion) component of the flexion/extension task was the better activity for distinguishing between the symptomatic individuals and the asymptomatic population. The differences were most pronounced for the largest loads (i.e. 10 kg).
5. A custom profile analysis of the B-200 moment-time histories, obtained during the performance of a clinical evaluation protocol, was found to augment the identification of spine movement dysfunction. The system developed in this study identified the abnormal performances and documented changes in performance associated with recovery. The OOC clinical evaluation protocol provided a rating of spine dysfunction for one of the case studies, but was insensitive to some types of spine dysfunction.

6. The moment normalized spinal EMG method (calibrated in N.m) was equivocal in its ability to reflect the changes and improvement for the individuals symptomatic for low back pain when compared to the asymptomatic population.
  
7. EMG profiles of the spinal flexor and extensor musculature activity, produced while performing a dynamic flexion-extension task and presented as mV or %MVC, did not enhance the understanding of the spine movement dysfunction. The variability in the profiles made the identification of abnormal activity difficult.

## **Chapter VI**

### **Discussion, Conclusions, Recommendations**

#### **Discussion**

This study is one of the first to evaluate spine movement dysfunction using a commercial lumbar spine dynamometer, EMG and an EMG assisted model. For each of the evaluation instruments a group of individuals asymptomatic for low back pain were used to develop "normal" performance profiles. Comparison of individuals symptomatic for low back pain to the normal profiles allowed the appropriateness of each technique for evaluating spine movement dysfunction to be determined. Repeating the evaluations for each of the participants over a period of time allowed changes in performance for the symptomatic individual's "Good" and "Bad" day to be documented.

The results of the study were very encouraging because each of the methods could be incorporated into a technique that could be utilized for the assessment of spine movement dysfunction. However, the conclusions reached in this study are limited by the small number of participants, particularly for the symptomatic group. This was a function of recruiting only individuals with recurrent low back pain who were selected primarily because of their experience with low back pain. The individuals in this study had to be familiar enough with their back "status" so that they could be evaluated on a good and a bad day in order to see which of the evaluation techniques could document the changes in spine movement dysfunction.

Each of the major questions will now be discussed in terms of how they were answered by the results of these studies.



***Question #1:***

Can the dynamic spine model be used for people in pain?

**RATIONALE:** The model requires maximum voluntary contractions to normalize the EMG. These contractions are problematic for individuals with low back pain. Is it possible for asymptomatic individuals to perform sustained, submaximal contractions of sufficient intensity in order to induce maximal electrical activation of the flexor and extensor musculature? This would avoid the typical maximal voluntary contractions.

The model was successfully adapted so that it could be used for people in pain. An original assumption was that sustained, isometric contractions of sufficient intensity would produce maximum amplitude EMG. This assumption was based on observations from work performed using the biceps brachii. However, this technique was unsuccessful when applied to the flexor and extensor musculature of the lumbar spine. Although the flexor and extensor EMGs increased by 35% and 20%, respectively, they failed to produce an EMG of 100% MVC. Small shifts in the test posture and the recruitment of other musculature to produce the required flexor and extensor moment were cited as explanations for these results.

Attempts were made to predict the maximal EMG amplitude by using submaximal contractions of varying intensity. Difficulties in replicating the test posture may have contaminated these results as well. On an individual basis, none of the subjects produced a substantial number of correlations in the flexor and extensor musculature that would have made the method viable.

An alternative technique was developed to calculate EMG-to-muscle force scaling factors which allowed the spine model to be used without performing MVCs. The construction of the model in its modular format made it possible to determine muscle forces for a particular level of contraction, provided the maximum amplitude that was observed was considered to be the maximum or 100% activation level. A sensitivity study which manipulated activation level

revealed no significant difference in the resultant muscle force calculations by using this approach.

This very important observation meant that for a pair of isometric contractions of different intensity there would be two levels of muscle force and two levels of EMG activation which could then be used to produce EMG-to-force scaling factors. As a result, the performance of appropriate calibration trials combined with modifications in the model's software allowed muscle forces to be estimated that were not dependant upon performing MVCs.

To test the model's output, groups of individuals asymptomatic and symptomatic for low back pain performed dynamic flexion and extension efforts. It was shown that the extensor muscle forces were not significantly different using either the MVC or the  $EMG_{Force}$  processing methods. However, the  $EMG_{Force}$  method was shown to produce significantly smaller lumbar spine compressions, flexor and extensor muscle moments and flexor muscle forces.

***Question #2:***

Is movement dysfunction identification, as documented by abnormal peak displacement, velocity and torso moment data output from a commercial dynamometer (i.e. the Isostation B-200) confirmed and/or augmented by the assessment of the entire moment-time history?

**RATIONALE:** Displacement, velocity, and moments may be obtained from a variety of dynamometers. The Isostation B-200 is a specific commercial dynamometer that utilizes this information to produce a "Back Dysfunction" rating. Individuals with low back pain have been found to produce abnormally low magnitudes of peak torque production in primary and secondary axes during isometric and dynamic testing. But the comparison is made using absolute instead of relative values, which would consider strength differences. Would a comparison of the relative moment magnitudes over the whole time history enhance the assessment?

The commercial dynamometer (Isostation B-200) and clinical assessment software (OOC, Version 3.1) used in this study found normal back function in three of the four case studies. The

fourth showed improvement from Moderate to Mild back dysfunction when evaluated on the Bad and the Good day, respectively. Several possible reasons were discussed for this observation with the conservative scoring system and utilization of peak values the most likely explanation for this anomaly.

Using the raw data from each of the clinical tests to produce a custom B-200 performance profile allowed abnormal performances to be quantified for each of the symptomatic individuals. The custom performance profile incorporated the primary axis velocity and the secondary and tertiary axes moments. However, when evaluating the changes between the Bad day and the Good day it was observed that the number of abnormal indicators did not necessarily decrease. One individual's score decreased, another person's stayed the same and the third person's increased. The velocity of movement showed improvement in 3 of the 4 cases for the Good day of testing. The custom B-200 performance profile is an alternative assessment method that has the potential to be used in developing profiles for both specific populations and diagnoses.

***Question #3:***

Does EMG obtained from select abdominal and back extensor muscles help specify a more precise location of spine movement dysfunction?

**RATIONALE:** The B-200 quantifies a level of dysfunction, but it oversimplifies the lumbar spine and the tests are not representative of tasks typically encountered in the activities of daily living. EMG may be utilized to assess muscular function. Would EMG profiles constructed from asymptomatic individuals performing a flexion-extension task provide insight into normal muscle function and serve as a reference for symptomatic individuals, assisting in providing a more specific identification?

Presenting the EMG signals as mV or % MVC did not help specify a more precise location of spine movement dysfunction. Overall, the right and left upper erector spinae

electrode sites were the two locations that illustrated excessive activity on a consistent basis. However, comparing an individual case's profile to the asymptomatic profiles was not particularly insightful due to the amount of variability present for the dynamic flexions and extensions. The major contributing factor to the variability was the freestyle nature of the task. The participants were allowed to perform the lifts and lowers using a self-selected style and speed.

The 0, 5 and 10 kg loads were selected for use in this study because they were thought to be typical of loads encountered in the performance of activities of daily living (e.g. lifting and lowering). However, the combination of the load size and the freestyle nature of the task were insufficient to produce a profile that would be feasible to use as a means of specifying a more precise location of dysfunction.

The right and left upper erector spinae EMG signals were also used to create a moment normalized spinal EMG profile. Combining the EMG and the link segment model together to produce a signal calibrated in N·m was an attractive alternative to the EMG profiles because it provided a calibrated signal which did not require MVCs. The RMS difference produced using this approach was very similar to that found by Potvin et al. (1990). However, calibrating the EMG signal to estimate the L4/L5 reaction moment during the performance of the flexion and extension tasks produced equivocal results. Although changes between the Bad and Good day test sessions, which could be attributed to improved functionality, were observed for some of the symptomatic individuals, the variability present in the normals minimized the utility of these profiles. These profiles also incorporated only two electrode sites. This restricts the musculature upon which the assessment is based to one anatomical region of the spine.

**Question #4:**

Is a more precise identification of the region of the back involved in the movement dysfunction, if not which tissue, possible, using the information provided by the spine model?

**RATIONALE:** The spine model provides force/time histories for 50 muscles, 12 ligaments and compression and shear forces acting on the L4/L5 motion unit. Does this level of knowledge provide more specific identification of dysfunctional structures?

The results of this study showed that a more precise identification of the region of the back involved in the movement dysfunction is produced by knowledge of the muscles forces estimated by the spine model. By summing the muscular forces associated with each EMG electrode, it was possible to produce a muscle force profile for each electrode site, day, load and task. When this information was presented via an Amplitude Probability Distribution Function (APDF) (Figure 38), it visually highlighted which of the flexor and/or extensor muscles had excessive force production, and for which part of the flexion/extension task (e.g. flexion). A criterion level (mean + 1 SD) for muscle force was determined for each electrode site for the asymptomatic population. The calculation of the "percentage of time above the criterion" allowed the excessive force production to be quantified for each day, load and task. The graphical presentation of this data (e.g. Figure 43) clearly identifies which of the flexor and/or extensor electrode sites have excessive activity and which muscles show improvement between the Good and Bad test days. Collapsing the excessive force production across all muscles created an overall summary (Figure 44) and allowed a single number to represent each load and task. Excessive muscle force production occurred in 3 of the 4 cases for the Bad day compared to the Good day. Excessive force production was not restricted to only the extensor muscles as excessive flexor musculature force production was also observed. This observation emphasizes the need to estimate both flexor and extensor muscle forces

**Question #5:**

What are the effects of recovery from spine movement dysfunction on the phase and magnitude of the lumbar spine model tissue force/time histories during isometric and dynamic contractions and do these force-time profiles provide more information than either the EMG or B-200 alone?

**RATIONALE:** The EMG assisted, dynamic, three dimensional spine model partitions the reaction moments produced by a linked segment model into the restorative moments generated by the 50 muscle fascicles, 12 ligamentous components, and the non-linear elastic intervertebral disc. Muscle fascicles that are functionally similar are assigned activation patterns from common surface EMG electrodes. Therefore, if changes in muscle function occur with recovery, these should be observed in the model muscle force outputs. Also, if changes in muscle function occur, these may also be observed in the EMG profiles and the B-200 profiles.

The effect of recovery from spine movement dysfunction was manifested by a decrease in excessive muscle force production, as observed in three of the four cases. For each case this could be observed by the summary APDFs and quantified by the percentage of time above the APDF criterion level. The summary APDFs also illustrated that for the flexion-extension task utilized in this study, the eccentric phase appeared to be more important in documenting differences in recovery because more excessive force production was found to occur in this region of activity.

It was anticipated that changes in muscle function should be observed in the EMG profiles and the B-200 profiles as well. For the EMG profiles, the upper erector spinae electrode site revealed some differences. The moment normalized EMG profile (calibrated in N·m) reflected some changes towards the “normal” profile for the asymptomatic individuals. However, the overall results were equivocal for this method. Improved performance was noted in the B-200 OOC clinical evaluation report with respect to greater peak velocities. Changes that were observed in the custom B-200 performance profiles could be due to improved coordination.

strength and/or decreased activation of antagonists. Overall it was impossible to attribute the improvement to one specific factor.

***Question #6:***

What are the specific resources necessary for each method?

**RATIONALE:** this allows a cost/benefit analysis to be performed so that for a specific scenario, the most appropriate method may be used.

The Isostation B-200 and clinical evaluation software system had several strengths. The duration of the testing time was relatively short. The comparison of each individual's results to a gender matched database provides some degree of breadth to the assessment. Minimal time was required to output the standard report. Also, the B-200 software has the capability of providing custom graphical analysis for a particular test. These advantages must be weighed against several weaknesses. The most important is that the mechanical axes of the machine do not align with the mechanical axes of the lumbar spine. The cost of the dynamometer may be prohibitive to some (≈\$70,000 U.S.). The clinical database sample size is limited to only 62 gender matched individuals. The number of individuals in the database cannot be increased, nor can it be customized in anyway (e.g. height or weight ranges). The software does allow the raw data to be displayed graphically, but no visual comparison to the OOC database is possible.

The custom B-200 profile provides an advantage in database development and management. A user would have the ability to construct a database specifically tailored to their clinical population and needs (e.g. diagnostic category, age, occupation). Currently, data reduction and report generation for the custom B-200 profile method is a time consuming process

because each of the raw data files must be converted into ASCII format by the manufacturer's proprietary software program.

The EMG methods allowed a profile to be developed, but normalization issues, especially for individuals in pain make its utility questionable. The issue of producing a "true" MVC will always persist. The technique is a reasonable method to utilize in the clinic because the costs are minimal in terms of time, and equipment costs are moderate, depending on the number of channels to be utilized.

The muscle force information provided by the computerized lumbar spine model clearly made it the most informative method. However, it was also the most costly in terms of equipment, processing time and personnel. The numerous steps required to collect and process the data currently make it feasible only in a laboratory situation.

### **Conclusions and Recommendations**

The primary purpose of this thesis was to evaluate a clinical dynamometer, EMG and an EMG assisted model of the lumbar spine to determine if more may be learned about spine movement dysfunction. Chapters III and IV addressed methodological subproblems that had to be solved before Chapter V, the primary study, could be performed. Chapter III investigated a method to obtain a muscles maximal EMG amplitude without performing maximal voluntary contractions. Chapter IV developed a method of obtaining EMG-to-muscle force scaling factors, that did not require the performance of maximal voluntary contractions. These scaling factors were then used in place of the maximal EMG amplitude scaling factors required by the EMG assisted lumbar spine model. In Chapter V individuals asymptomatic and symptomatic for low



back pain were compared using the clinical dynamometer, EMG and an EMG assisted lumbar spine model to learn more information about spine movement dysfunction. Each of the evaluation methods had its own strengths and weaknesses. However, there were components of each method which were shown to be associated with improvements in the symptomatic individuals. The following sections summarize the conclusions from this research and present suggestions for future research.

### **Conclusions**

1. The maximal back extension efforts required to calibrate EMG to units of muscle force in a complex EMG assisted model, that were necessary prior to this study, can now be replaced by submaximal efforts. This is a necessity for use of this model with individuals in pain or afraid of injury. It would also be advantageous to use in situations (e.g. industry) where individuals may be unfamiliar with producing maximal effort muscular contractions. Regardless of the method used, there was no difference in the calculated extensor muscle forces. The submaximal effort method did result in smaller flexor muscle forces and lumbar compressive forces.
2. The APDF presentation technique provided a visual summary of the muscle force data that immediately highlighted the intra-individual differences between loads and days, and the inter-individual differences between each symptomatic case and the asymptomatic population. The APDF method also qualitatively indicated for the symptomatic

individuals which of the flexor and extensor electrode sites were most affected for each load and test day.

3. The "percentage of time above the APDF criterion level" calculation quantified the intra- and inter-individual differences in the flexor and extensor muscle forces for each of the electrode locations, hand loads and test days. Expressing the EMG in units of mV or %MVC did not distinguish these differences.
4. Improved functionality, as indicated by decreases in Oswestry and visual analog scale scores, was typically (3 out of 4 cases) associated with decreases in excessive spinal flexor and/or extensor muscle force production.
5. The eccentric (flexion) component of the flexion/extension task was the better activity for distinguishing between the symptomatic individuals and the asymptomatic sample. The differences were most pronounced for the largest loads (i.e. 10 kg).
6. According to the data, the day of testing did not significantly affect the model outputs of flexor or extensor muscle force, flexor or extensor muscle moment, or spinal compression. This is an important conclusion for utilization of the model with studies that require multiple test sessions.

7. A custom profile analysis of the B-200 moment-time histories, obtained during the performance of a clinical evaluation protocol, was found to augment the identification of spine movement dysfunction. The system developed in this study identified the abnormal performances and documented changes in performance associated with recovery. The OOC clinical evaluation protocol provided a rating of spine dysfunction for one of the case studies, but was insensitive to some types of spine dysfunction.
8. The moment normalized spinal EMG method (calibrated in N.m) was equivocal in its ability to reflect the changes and improvement for the individuals symptomatic for low back pain when compared to the asymptomatic population.
9. Maximal EMG amplitude of the flexor and extensor musculature of the spine can not be reliably determined using sustained, submaximal exertions. Although the EMG amplitude did increase by more than 20% and 35%MVC for the flexors and extensors, respectively, the final amplitudes were too variable for this method to be used.
10. Maximal EMG amplitudes of the flexor and extensor musculature of the spine can not be reliably predicted by means of linear regression and extrapolation from the maximal amplitude observed from submaximal contractions of varying intensity. Changes in posture and the recruitment of other musculature may have contributed to the poor predictions obtained in this study.

11. EMG profiles of the spinal flexor and extensor musculature activity, produced while performing a dynamic flexion-extension task and presented as mV or %MVC, did not enhance the understanding of the spine movement dysfunction. The variability in the profiles made the identification of abnormal activity difficult.

### **Future Considerations**

1. The calibration posture incorporated in this study is too cumbersome. The current prone and supine postures are difficult to position a person in, are uncomfortable once the person is positioned, and the moments induced by the torso are very large, limiting the calibration range. Having a person perform the test contractions in a standing position would greatly facilitate the calibration procedure.
2. The calibrations should be performed in the range of lumbar moments that will be produced in the study. The prone and supine postures used in this study produced large moments due to the torso mass. As a result, the EMG-to-Force calibration method overestimated the amount of muscle force produced per unit EMG. Utilizing a standing posture would minimize this effect.
3. This study utilized a two point, linear calibration method. Adding a third point would help to minimize the overestimation of muscle force per unit EMG in the EMG-to-Force method. This would also allow a curvilinear calibration to be performed.

4. The task utilized should be more stressful. If a flexion/extension task similar to the one in this study is to be used, the load should be greater than 10 kg. This should amplify the responses of the symptomatic individuals.
  
5. The performance of the task should be more structured. One of the limitations of the EMG profiles was the amount of variability present. This was due to the freestyle nature of the task where only the location of the load was controlled. Restricting posture and controlling the speed of movement may perturb the musculature in such a manner that the responses of the symptomatic individuals are amplified with respect to the asymptomatic individuals.
  
6. The symptomatic individuals used in this study are representative of most clinical low back research studies in that whoever is available is recruited. This is partly due to the clinical nature of low back pain in that you never know who will be suffering and when. Now that the model may be applied to those with low back pain, it would be possible to develop a database of individuals evaluated early on in their symptomatic period and then continually evaluate them at future points in time (e.g. more than the two used in this study). The results for specific populations could then be analyzed using a number of sorting criteria (e.g. physical findings, age, gender, treatment protocol), providing further insight into the assessment and treatment of spine movement dysfunction.

7. The "moment normalized" EMG processing method which incorporated the left and right upper erector spinae musculature produced equivocal results. This was due to the variability in the normal profile, which was partly a function of the task. Incorporating a more stringent task may improve its utility. It would also be beneficial to present the output using the APDF format and criterion level approach incorporated in this study. If this type of system were capable of identifying periods of excessive EMG activity, then the approach would be very beneficial in a clinical environment. The equipment and time costs that an EMG based system would require are minimal. This would allow the excessive activity to be represented using a variety of interfaces (e.g. computer screen, audio signal, LED panel) so that a personal training/rehabilitation system could also be developed. If this information was present in an APDF and a criterion level established, it may differentiate between the asymptomatic and symptomatic individuals. It may require a more stringent task as well. This method would not require use of the model which would be more attractive to clinicians. Once a normal profile group was established, then there would be several methods in which excessive activity could be displayed (e.g. computer screen, LED panel).
8. The APDF format used for expressing the excessive muscle force production could be improved by integrating the area between an individual muscle's APDF curve and the APDF criterion level. This would help to further quantify the magnitude of excessive force production. It would also be possible to incorporate pattern recognition technology

with the APDF profiles in an effort to determine if specific muscle force profiles or signatures existed for specific types of spinal dysfunction.

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## **Appendix A**

### Oswestry Low Back Pain Disability Questionnaire

The Oswestry Low Back Pain Disability Questionnaire  
 Fairbank et al., (1980), *Physiotherapy*, 66, 271-273.  
 Baker et al., (1989), In: (Roland, MO, Jenner, JR, eds.)  
*Back Pain: New Approaches to Rehabilitation and Education*, 174-186.

This questionnaire has been designed to give us information as to how your back pain has affected your ability to manage in everyday life. Please answer every section, and mark in each section **ONLY THE ONE BOX** which applies to you. We realise you may consider that two of the statements in any one section relate to you, but **PLEASE JUST MARK THE BOX WHICH MOST CLEARLY DESCRIBES YOUR PROBLEM.**

### Section 1 - Pain Intensity

- I have no pain at the moment.
- The pain is very mild at the moment.
- The pain is moderate at the moment.
- The pain is fairly severe at the moment.
- The pain is very severe at the moment.
- The pain is the worst imaginable at the moment.

### Section 2 - Personal Care (Washing, Dressing, etc.)

- I can look after myself normally without causing extra pain.
- I can look after myself normally but it causes extra pain.
- It is painful to look after myself and I am slow and careful.
- I need some help but manage most of my personal care.
- I need help every day in most aspects of self care.
- I do not get dressed, wash with difficulty and stay in bed.

### Section 3 - Lifting

- I can lift heavy weights without extra pain.
- I can lift heavy weights but it gives extra pain.
- Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned e.g. on a table.
- Pain prevents me from lifting heavy weights but I can manage light to medium weights if they are conveniently positioned.
- I can only lift only very light weights.
- I cannot lift or carry anything at all.

### Section 4 - Walking

- Pain does not prevent me walking any distance.
- Pain prevents me from walking more than 1 mile.
- Pain prevents me from walking more than 1/2 mile.
- Pain prevents me from walking more than 1/4 mile.
- I can only walk using a stick or crutches.
- I am in bed most of the time and have to crawl to the toilet.

**Section 5 - Sitting**

- I can sit in any chair as long as I like.
- I can sit in my favourite chair as long as I like.
- Pain prevents me from sitting more than 1 hour.
- Pain prevents me from sitting more than 30 minutes.
- Pain prevents me from sitting more than 10 minutes.
- Pain prevents me from sitting at all.

**Section 6 - Standing**

- I can stand as long as I want without extra pain.
- I can stand as long as I want but it gives me extra pain.
- Pain prevents me from standing more than 1 hour.
- Pain prevents me from standing more than 30 minutes.
- Pain prevents me from standing more than 10 minutes.
- Pain prevents me from standing at all.

**Section 7 - Sleeping**

- My sleep is never disturbed by pain.
- My sleep is occasionally disturbed by pain.
- Because of pain I have less than 6 hours sleep.
- Because of pain I have less than 4 hours sleep.
- Because of pain I have less than 2 hours sleep.
- Pain prevents me from sleeping at all.

**Section 8 - Sex Life**

- My sex life is normal and causes no extra pain.
- My sex life is normal and causes some extra pain.
- My sex life is nearly normal but is very painful.
- My sex life is severely restricted by pain.
- My sex life is nearly absent because of pain.
- Pain prevents any sex life at all.

**Section 9 - Social Life**

- My social life is normal and causes me no extra pain.
- My social life is normal but increases the degree of pain.
- Pain has no significant effect on my social life apart from limiting my more energetic interests e.g. sport, etc.
- Pain has restricted my social life and I do not go out as often.
- Pain has restricted my social life to home.
- I have no social life because of pain.

**Section 10 - Travelling**

- I can travel anywhere without pain.
- I can travel anywhere but it gives me extra pain.
- Pain is bad but I manage journeys over two hours.
- Pain restricts me to journeys of less than 1 hour.
- Pain restricts me to short journeys under 30 minutes.
- Pain prevents me from travelling except to receive treatment.

## **Appendix B**

Visual Analogue Pain Scale

Visual Analogue Pain Scale

On the line below, please make a mark to indicate how much pain you feel at this time.



No Pain

Worst Imaginable

## **Appendix C**

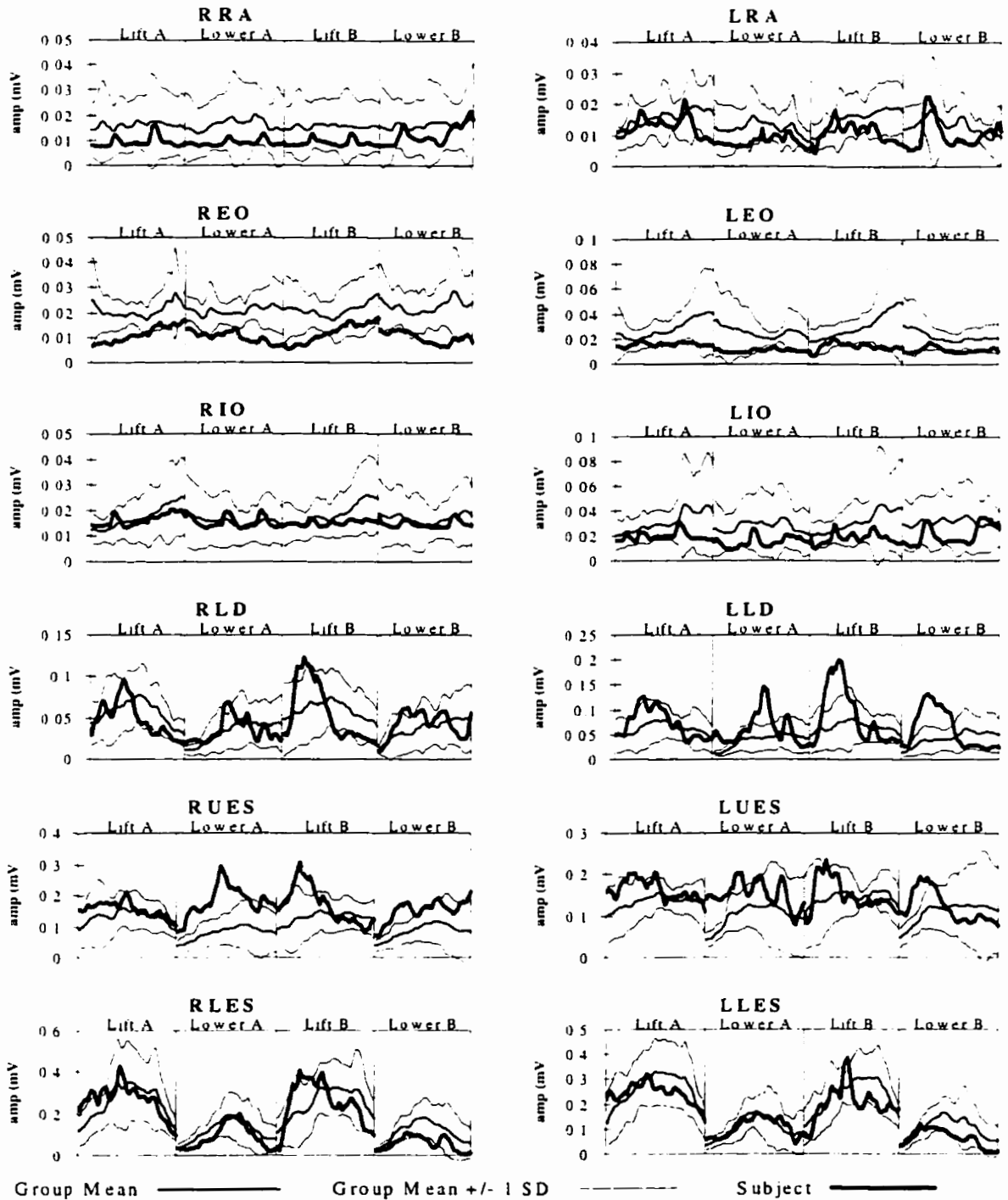
Case Study #1

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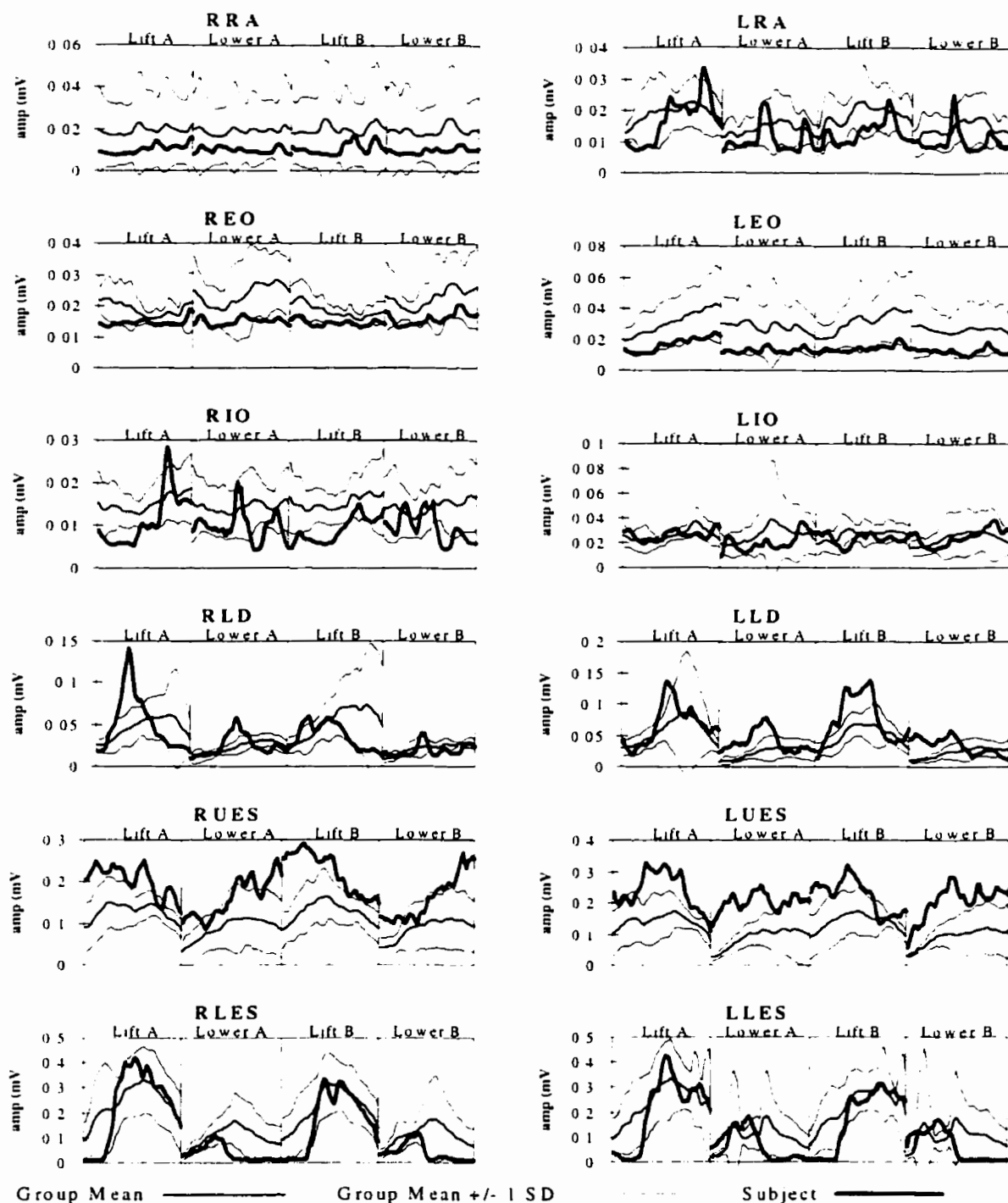
**Pages 238 & 239**

**UMI**

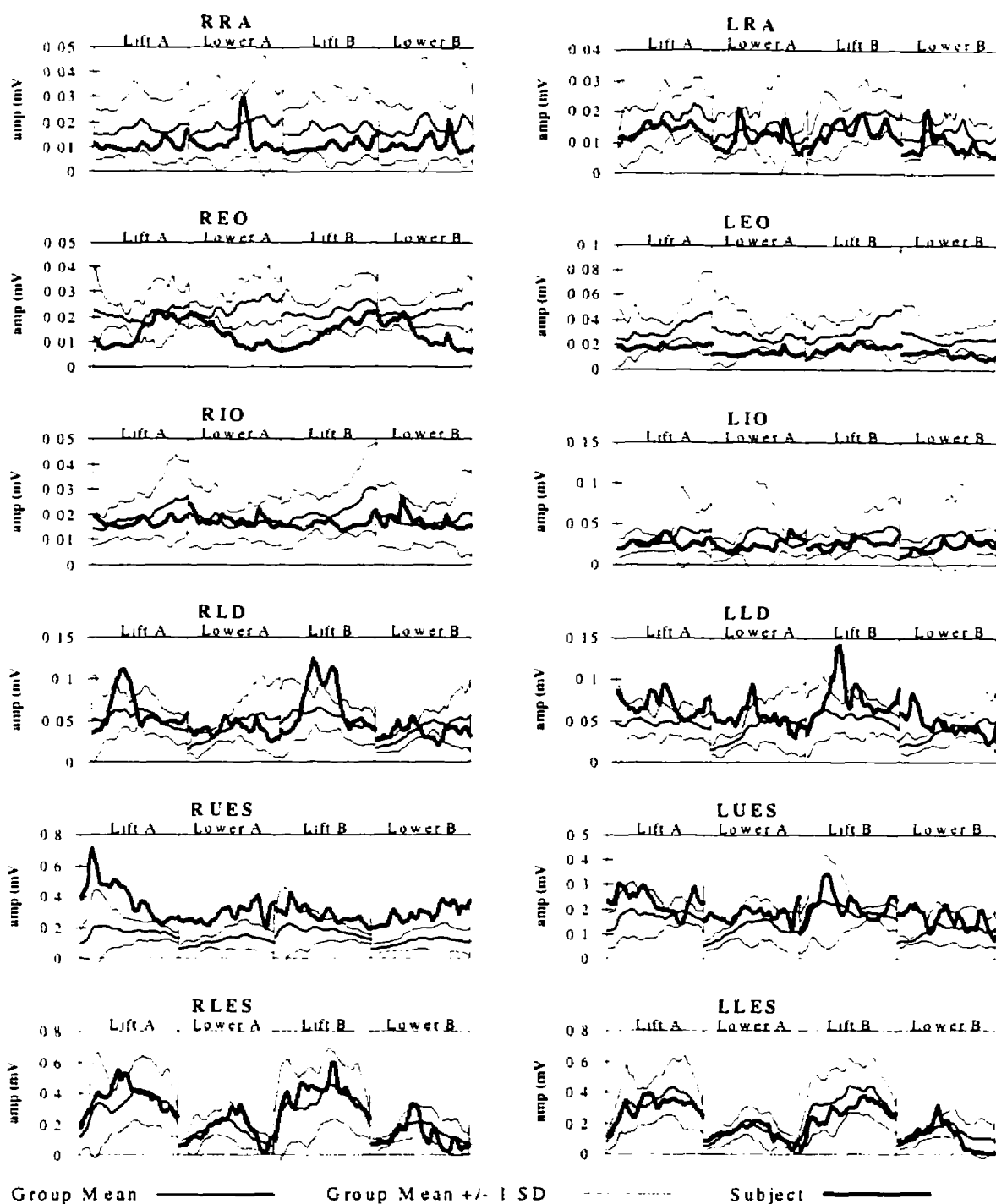


**Figure C1** Case Study #1 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (mV) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R = right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Bad Day.

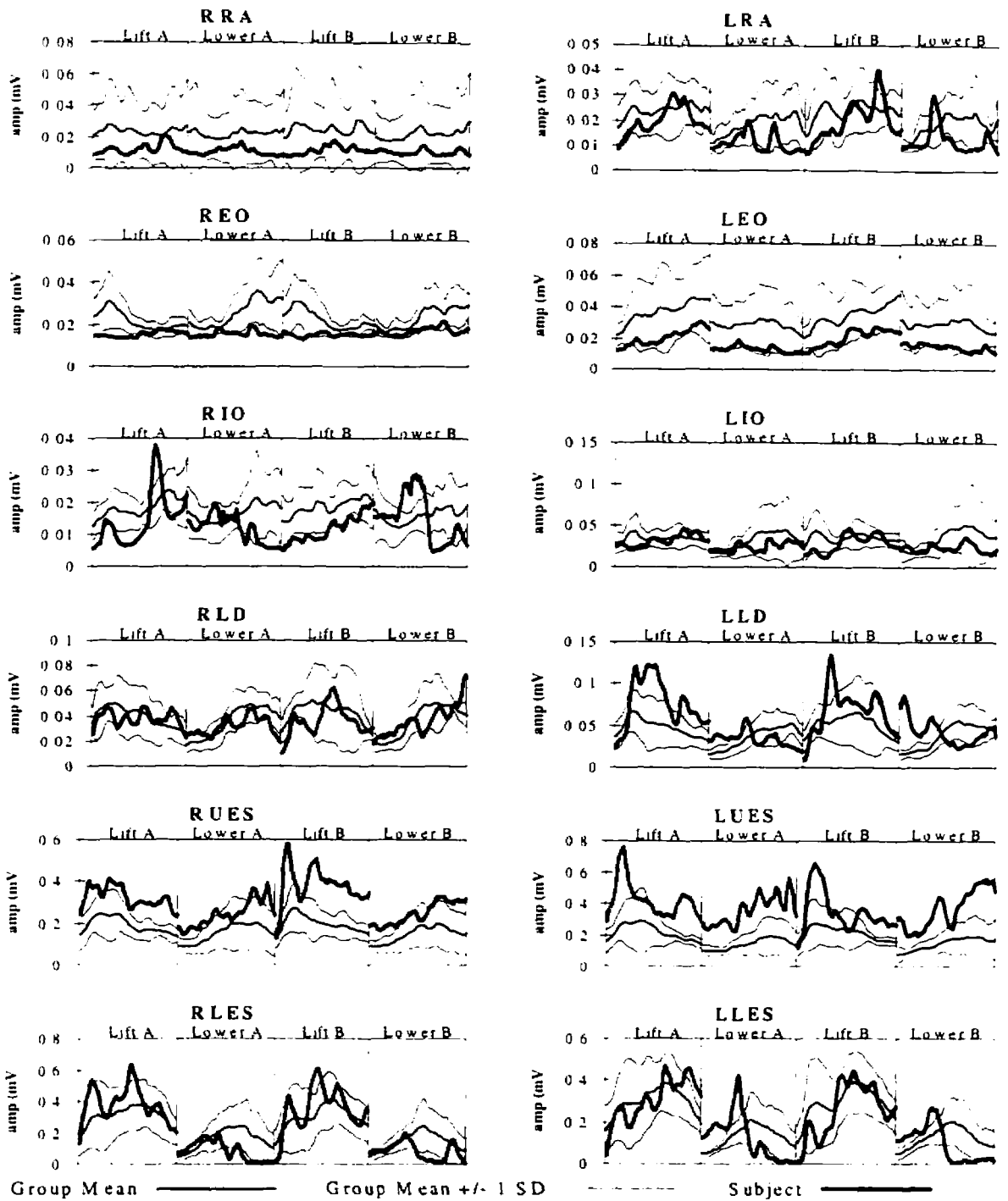




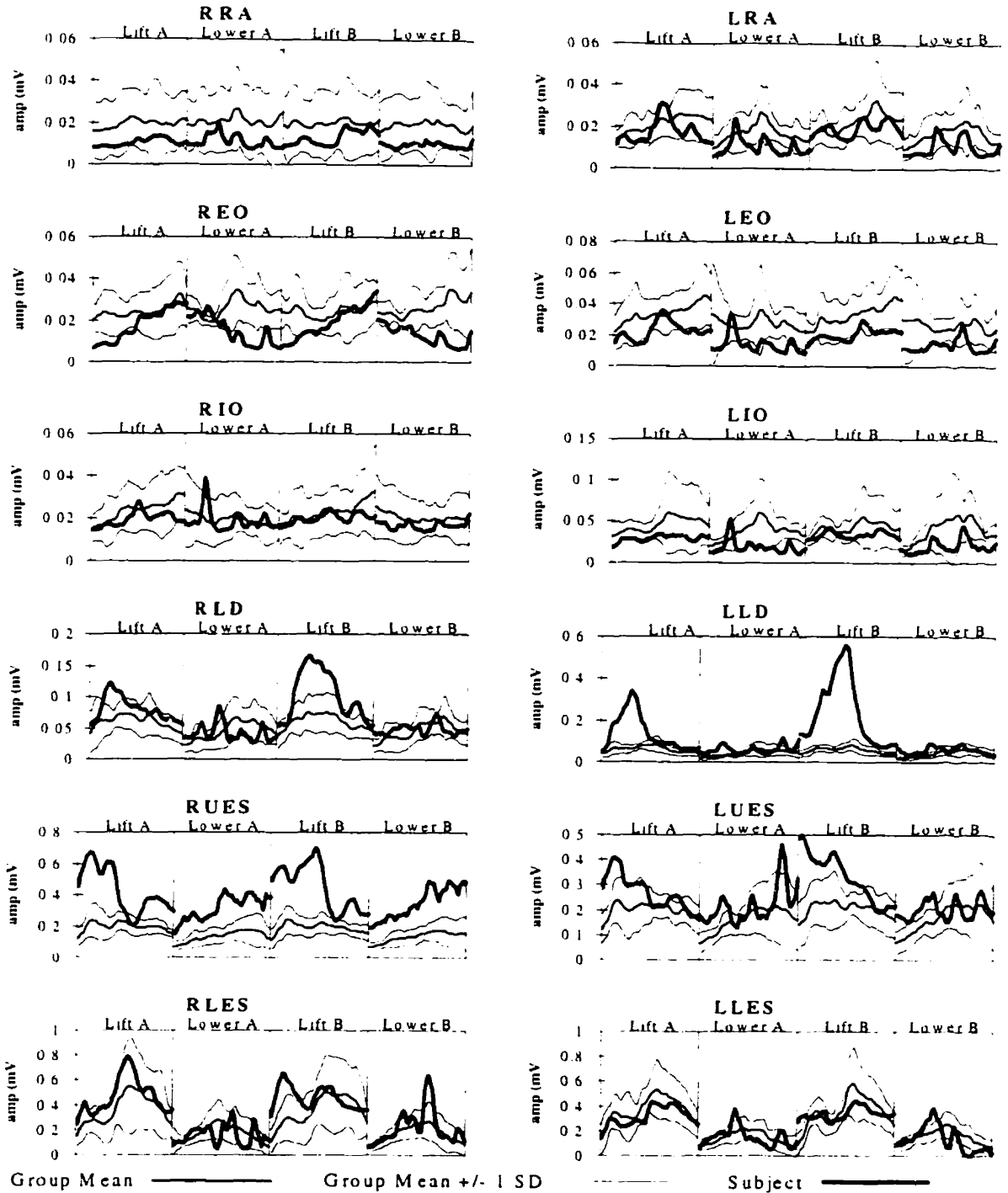
**Figure C2** Case Study #1 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (mV) for Day 2, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Good Day.



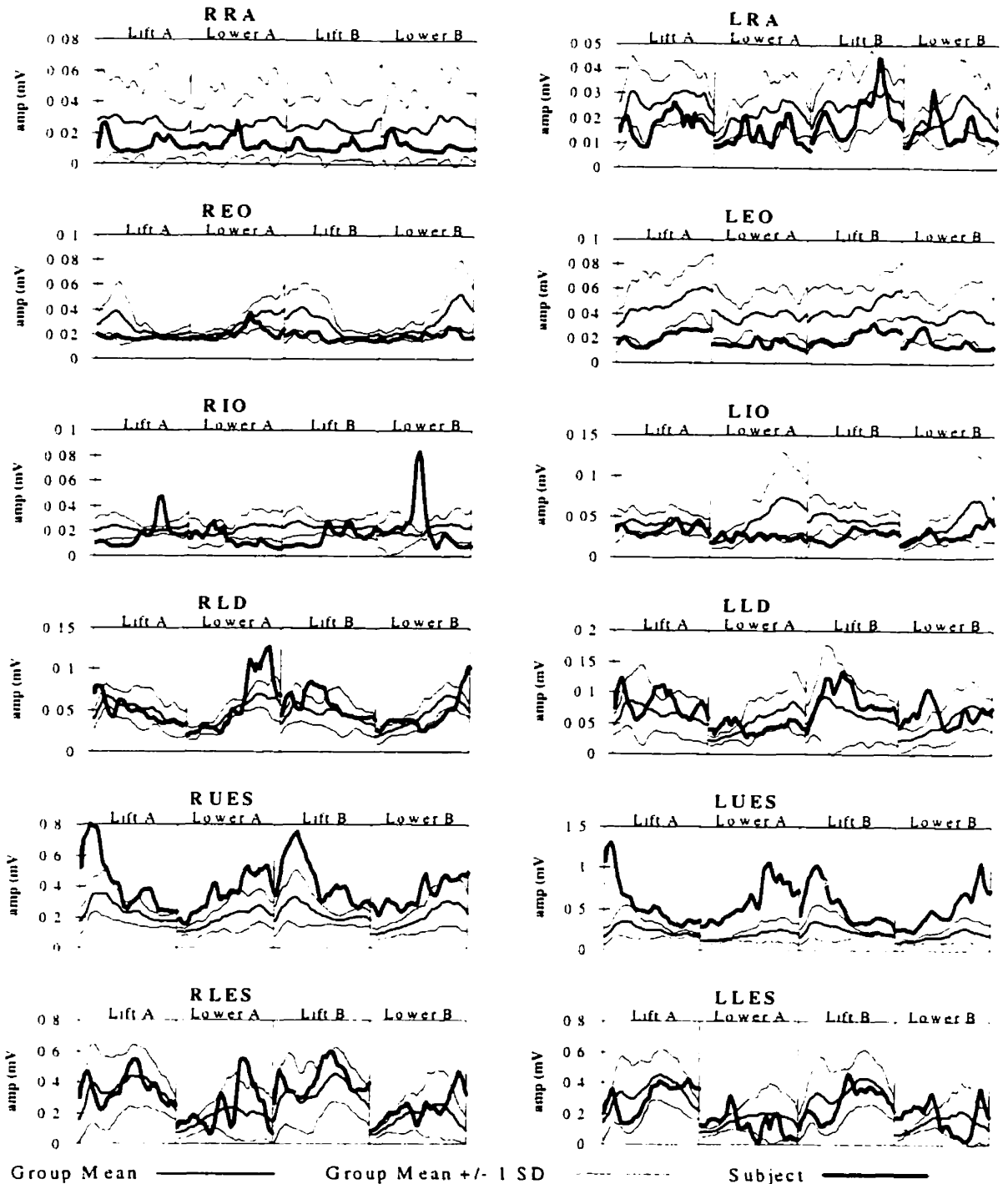
**Figure C3** Case Study #1 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (mV) for Day 1, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Bad Day.



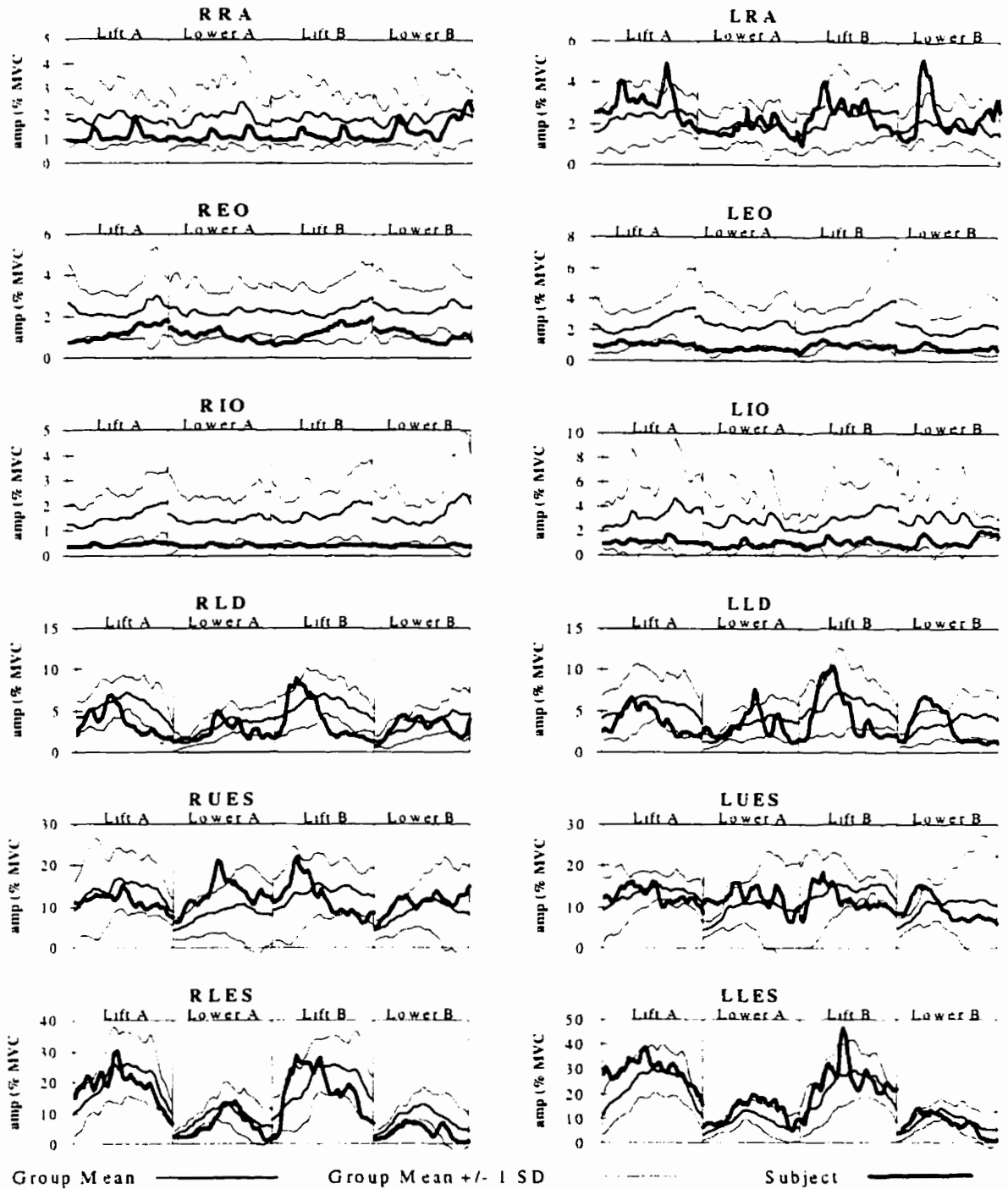
**Figure C4** Case Study #1 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (mV) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Good Day.



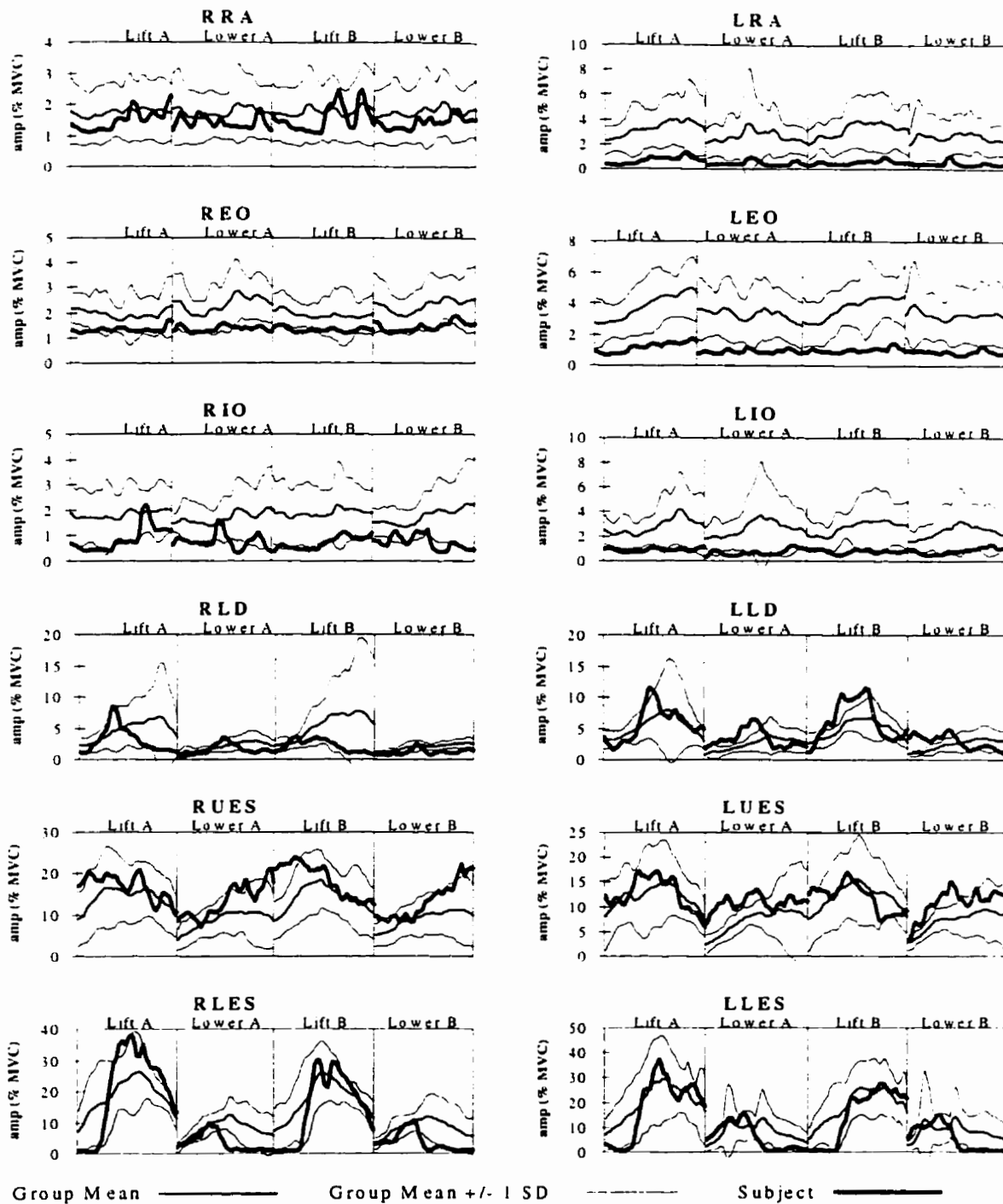
**Figure C5** Case Study #1 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (mV) for Day 1, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Bad Day.



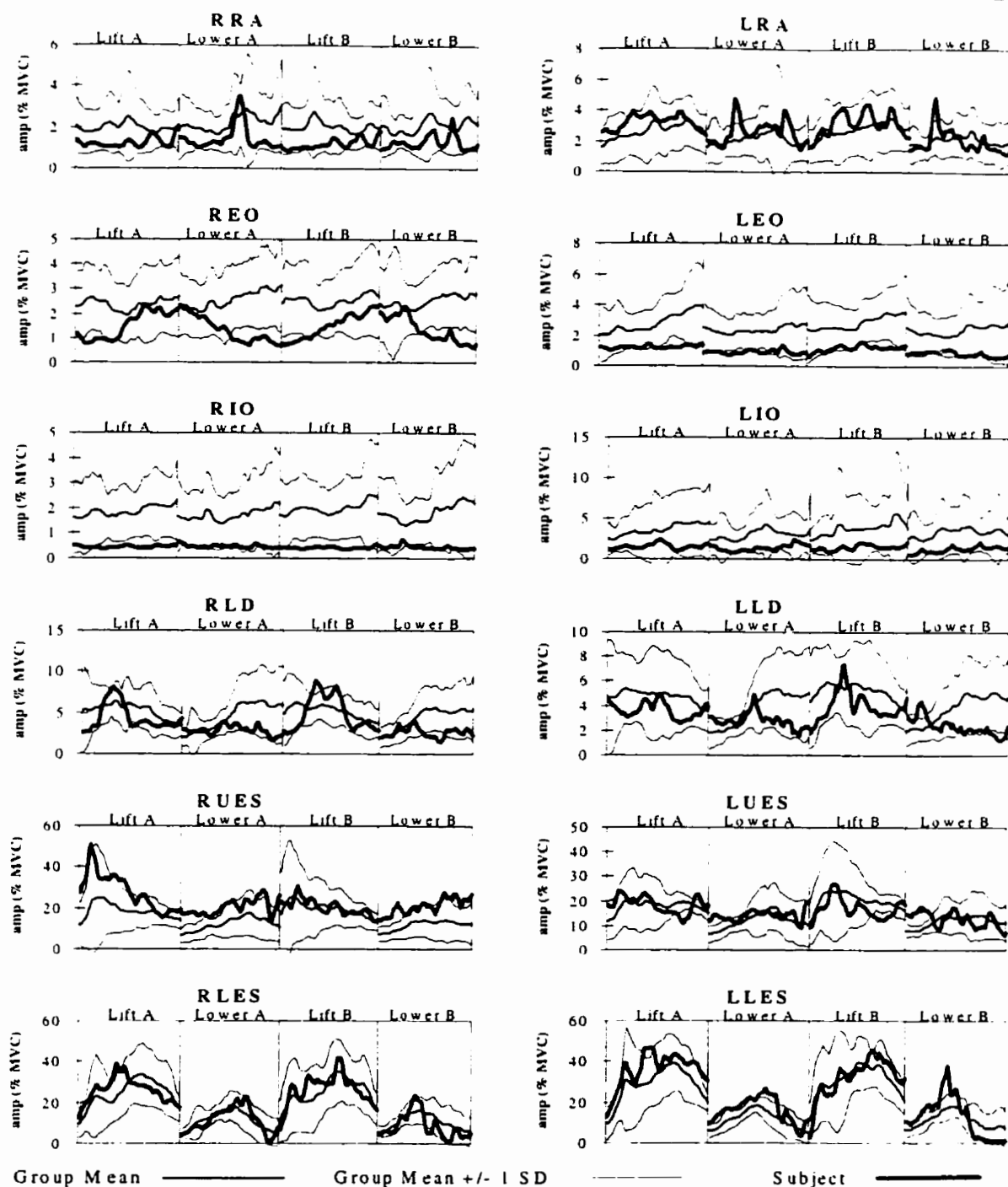
**Figure C6** Case Study #1 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (mV) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Good Day.



**Figure C7** Case Study #1 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (% MVC) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Bad Day.

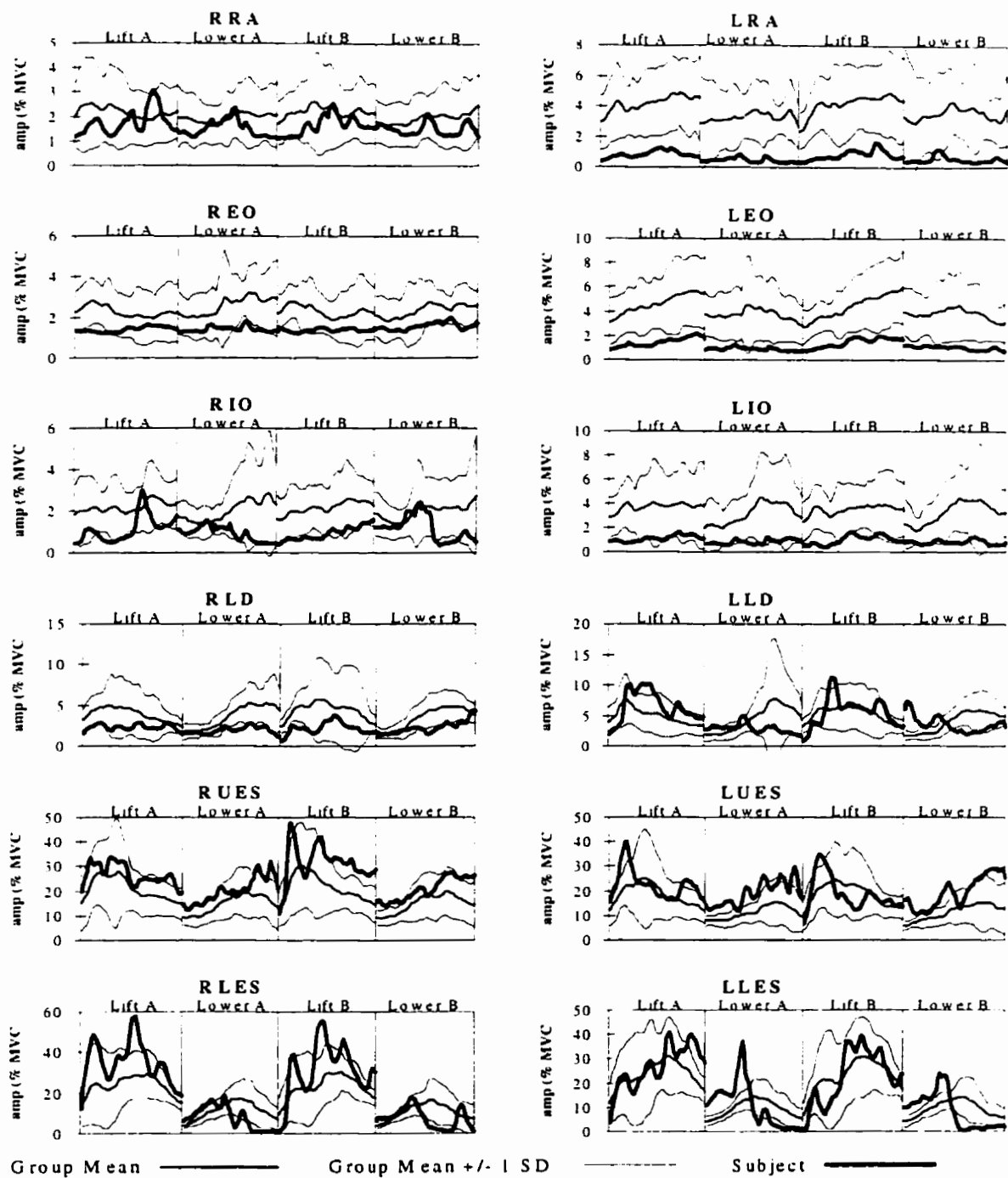


**Figure C8** Case Study #1 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 2, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Good Day.

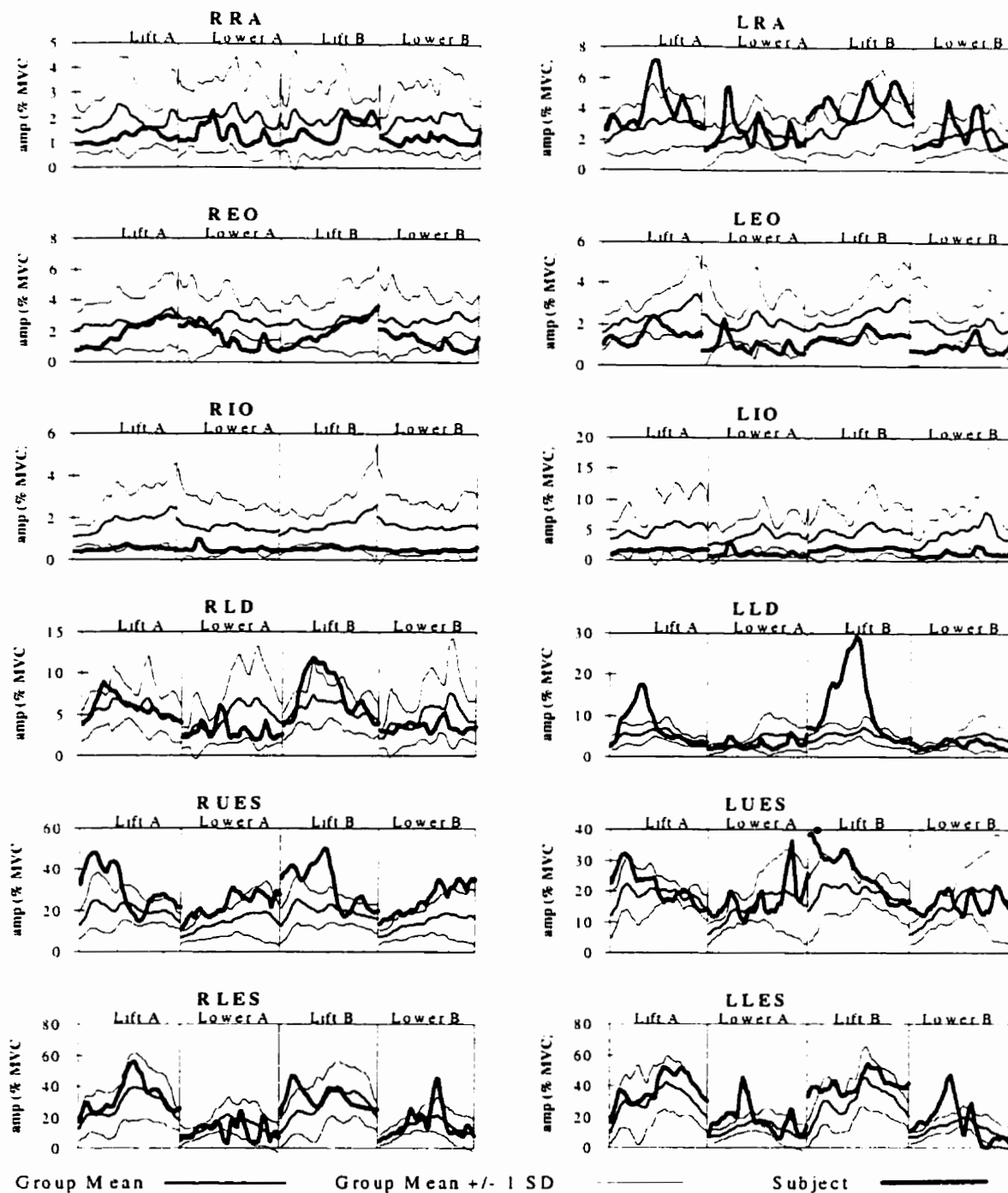


**Figure C9** Case Study #1 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 1, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Bad Day.

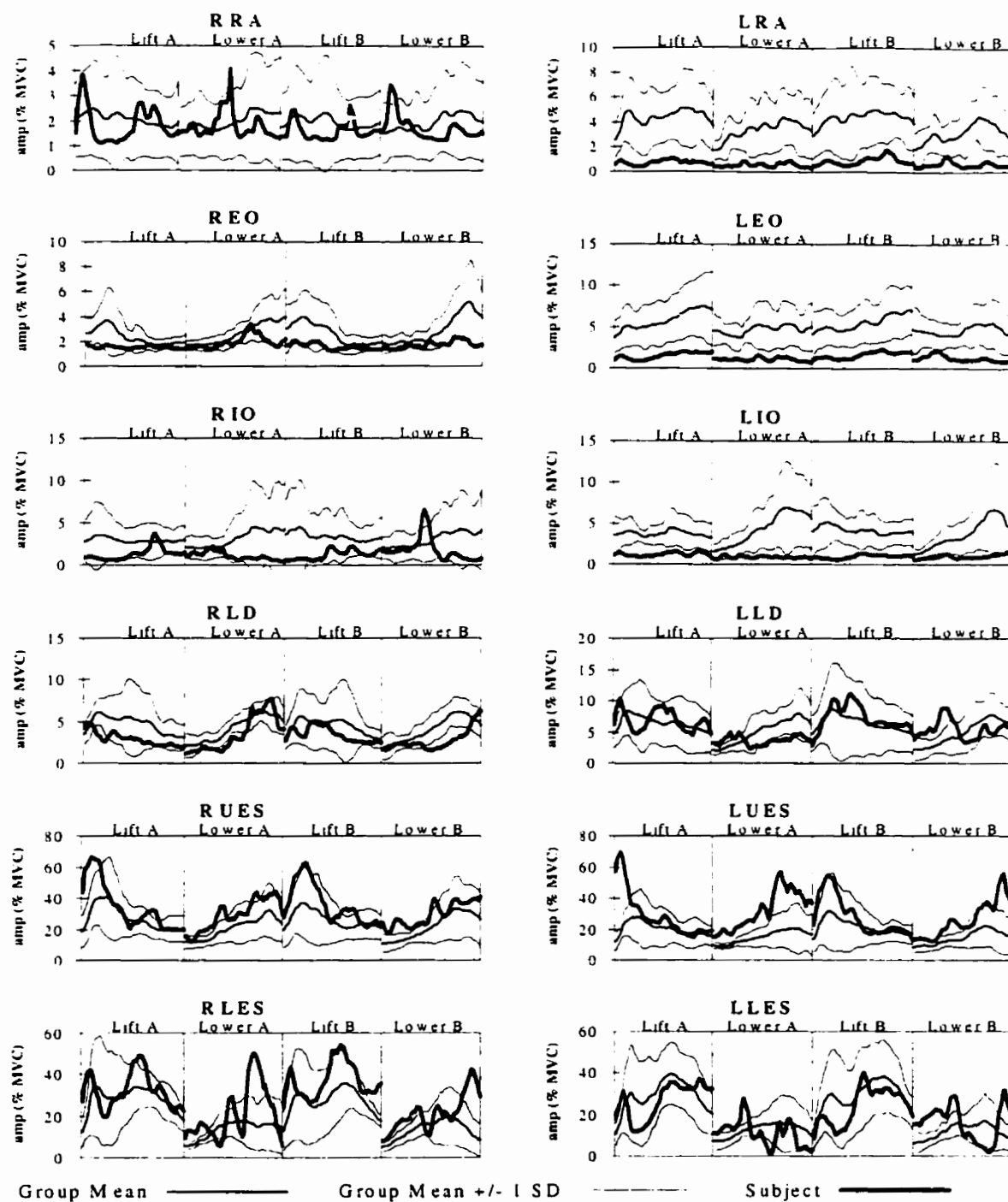




**Figure C10** Case Study #1 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Good Day.



**Figure C11 Case Study #1** graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (% MVC) for Day 1, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Bad Day.



**Figure C12** Case Study #1 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA= rectus abdominis, EO= external oblique, IO= internal oblique, LD= latissimus dorsi, UES= upper erector spinae, LES= lower erector spinae. Note: Day 2 = Good Day.

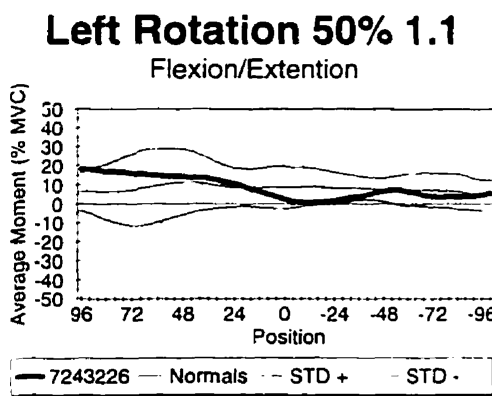
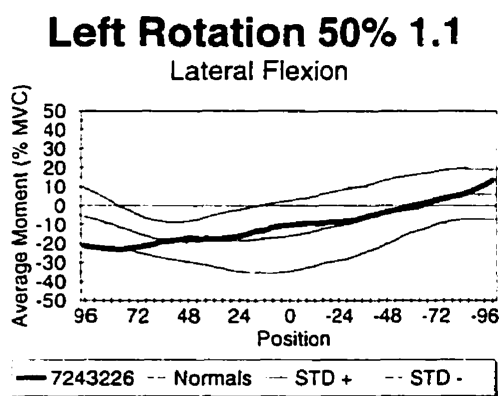
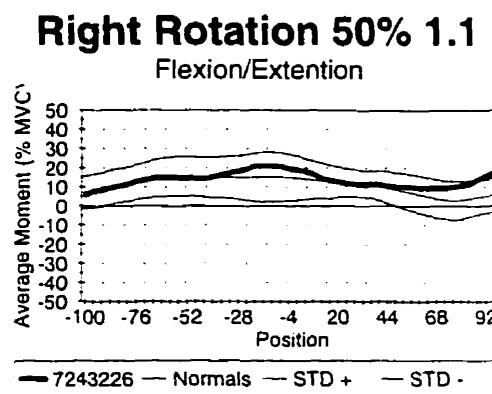
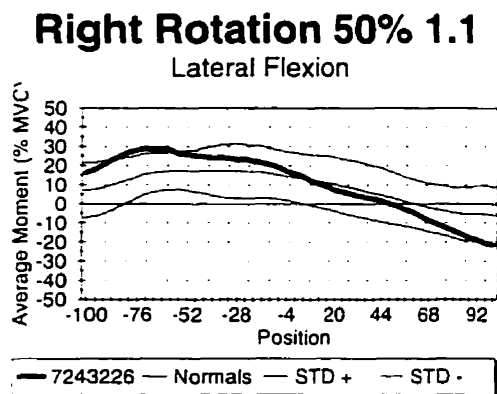
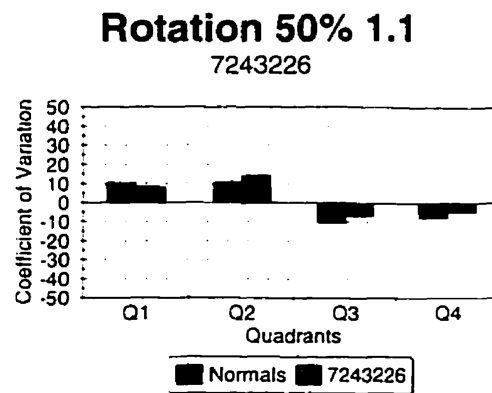
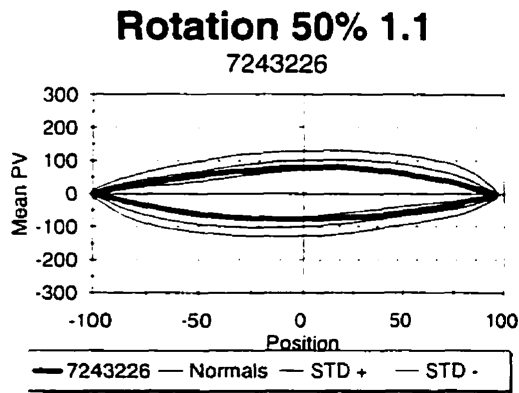


Figure C13B-200 summary performance for Case Study #1 (Bad day), for dynamic rotation at 50% resistance, day 1, test sequence #1.

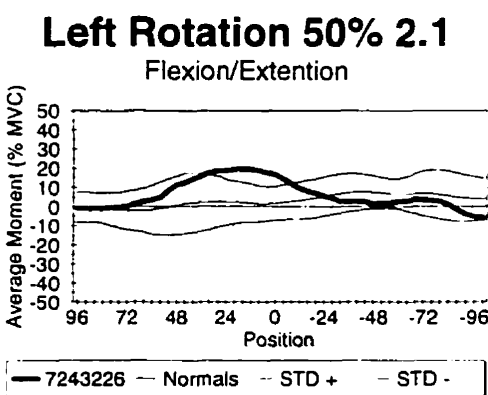
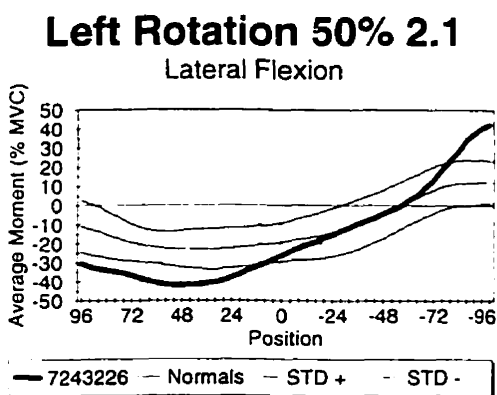
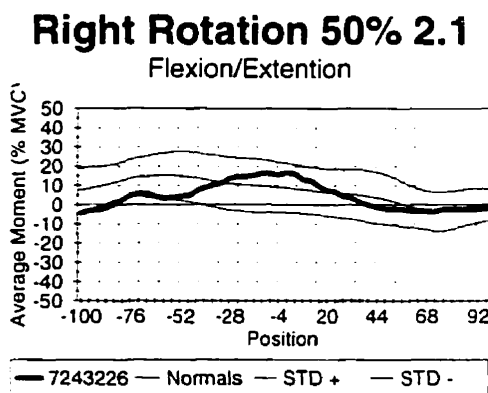
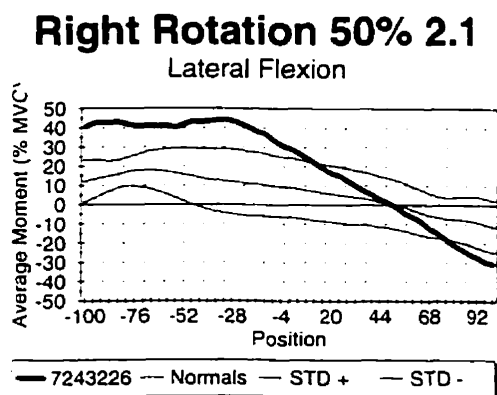
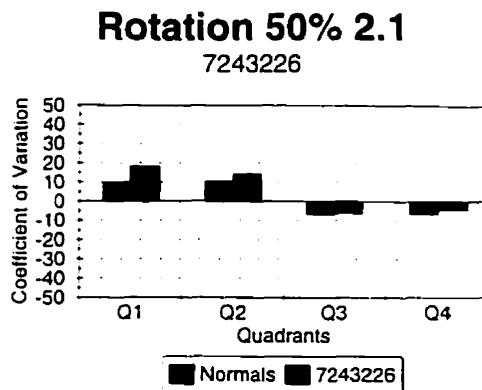
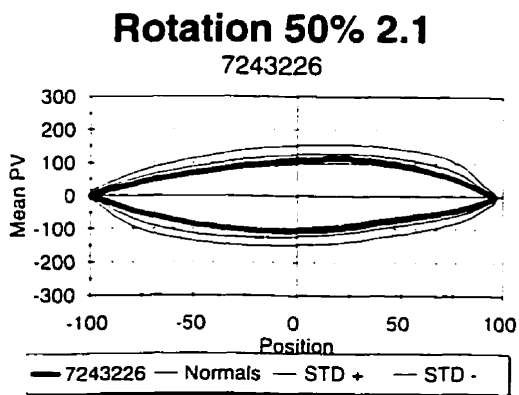


Figure C14B-200 summary performance for Case Study #1 (Good day), for dynamic rotation at 50% resistance, day 2, test sequence #1.

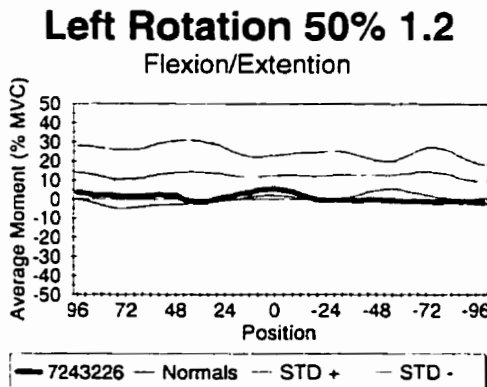
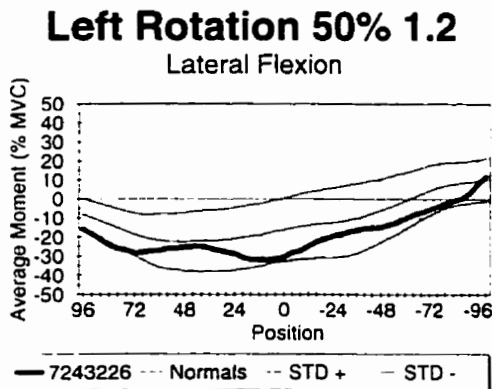
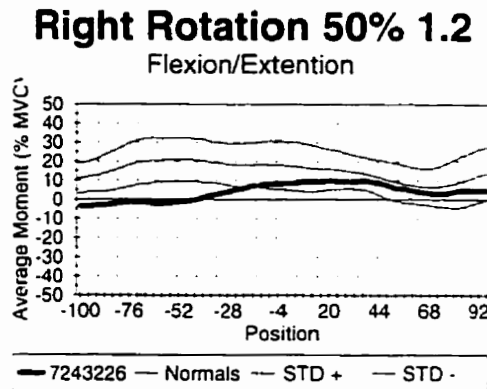
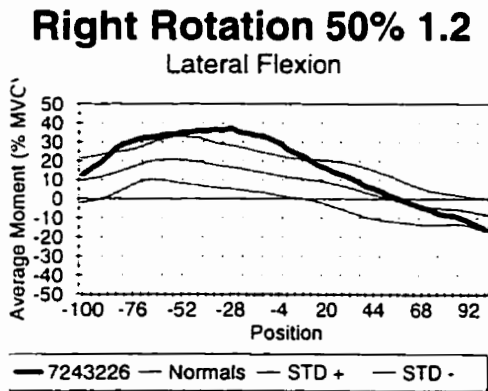
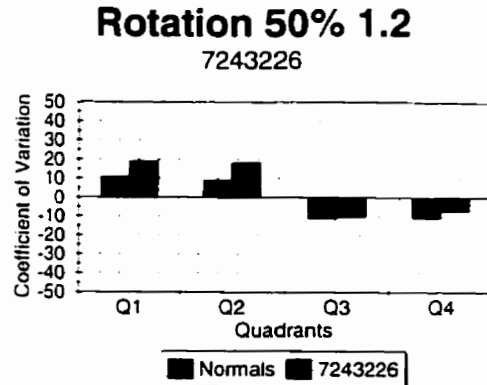
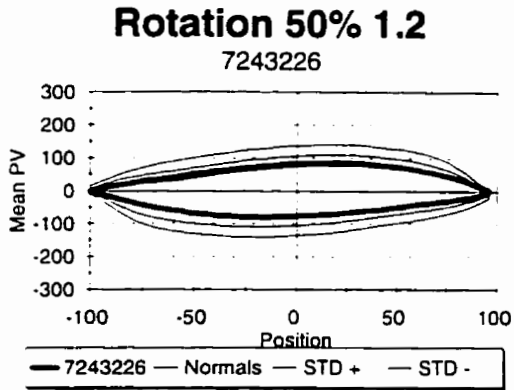
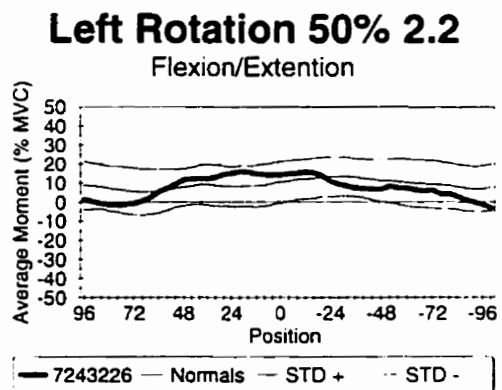
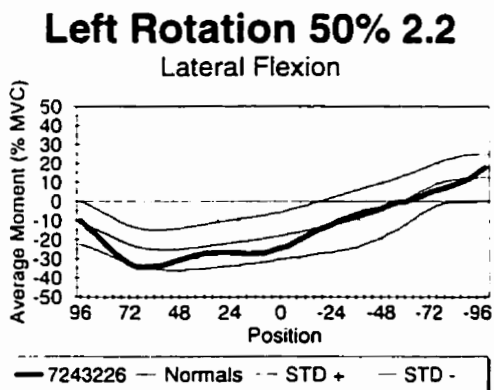
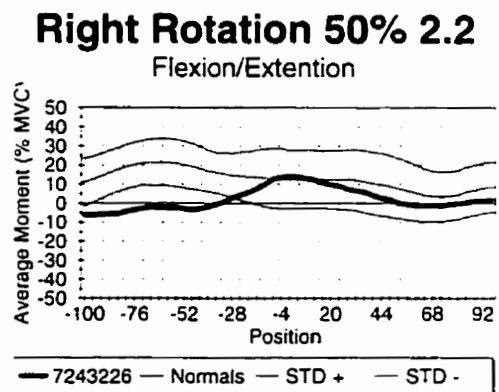
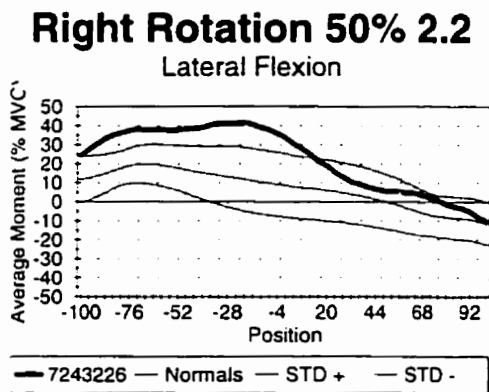
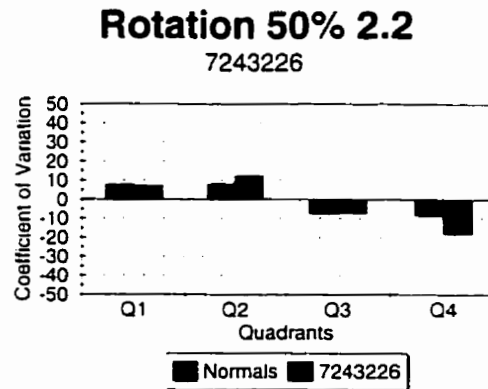
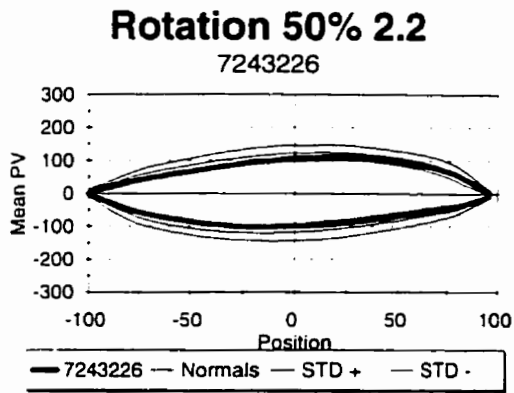


Figure C15B-200 summary performance for Case Study #1 (Bad day), for dynamic rotation at 50% resistance, day 1, test sequence #2.



**Figure C16B-200** summary performance for Case Study #1 (Good day), for dynamic rotation at 50% resistance, day 2, test sequence #2.

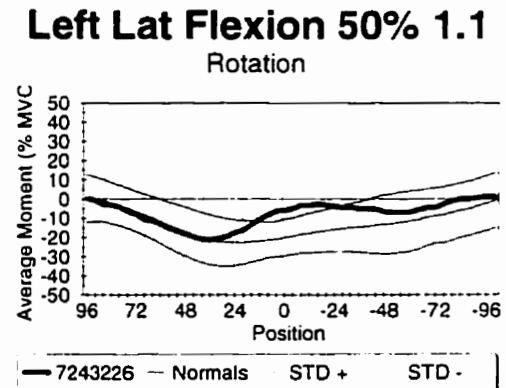
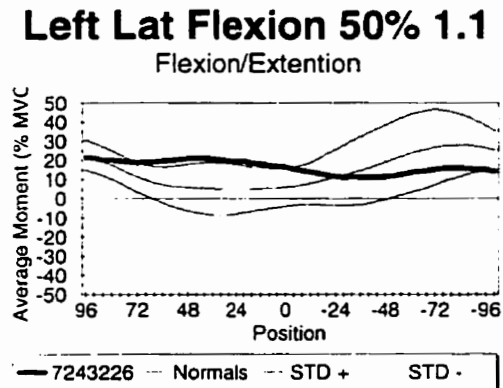
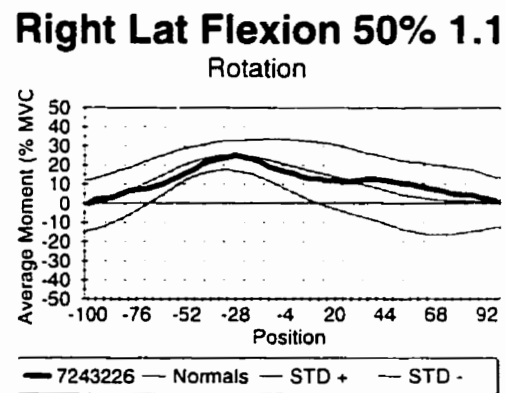
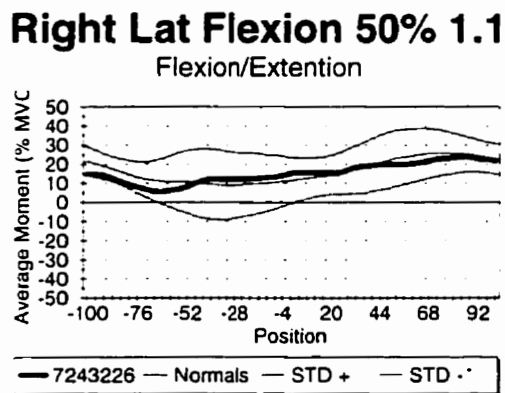
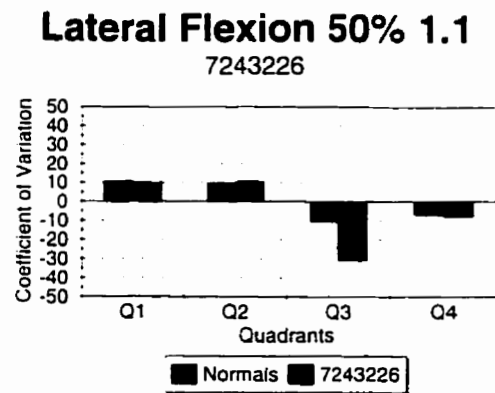
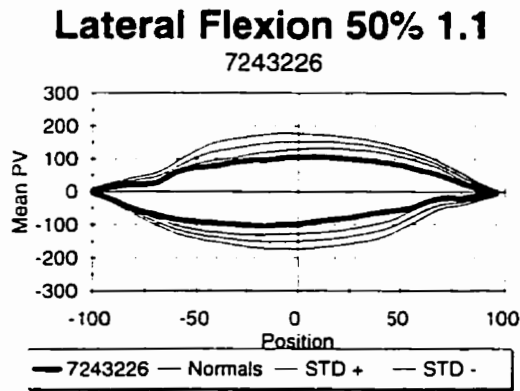
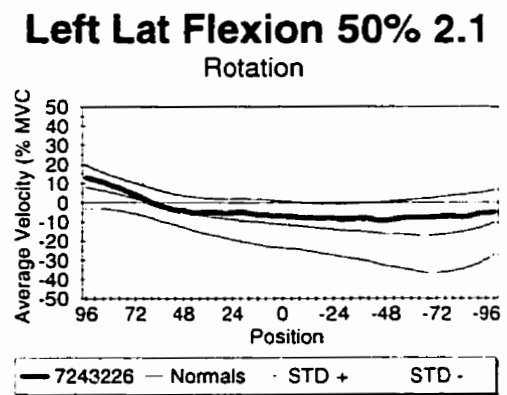
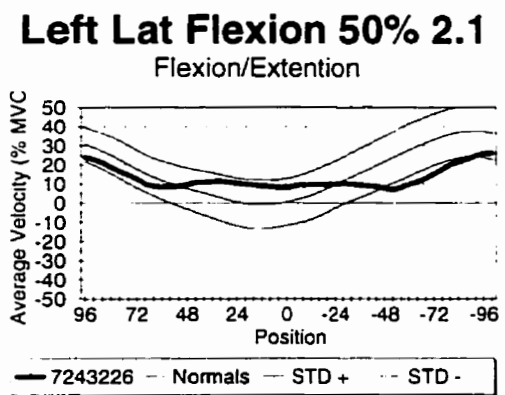
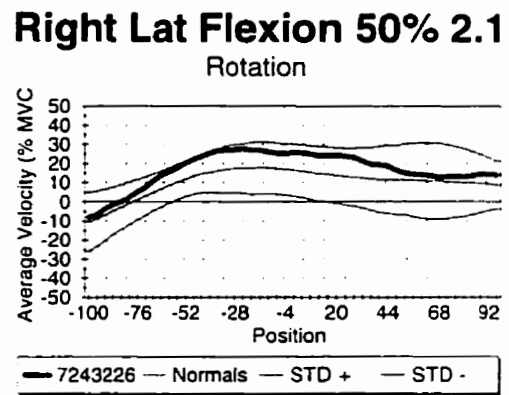
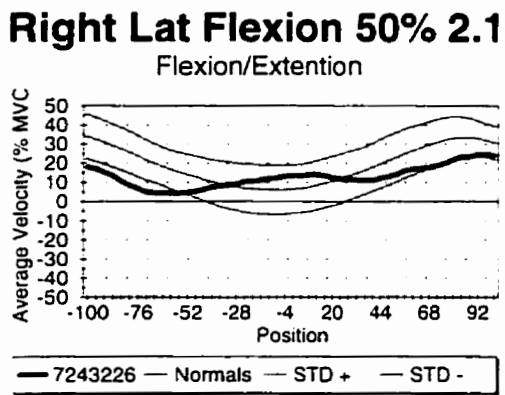
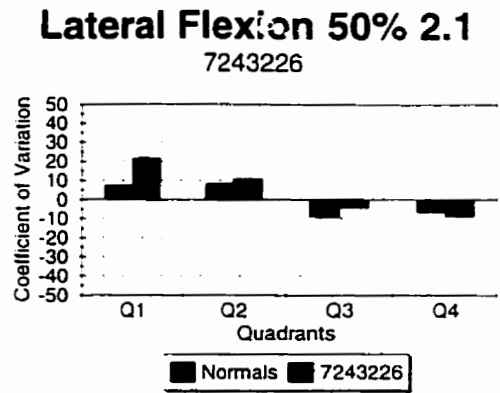
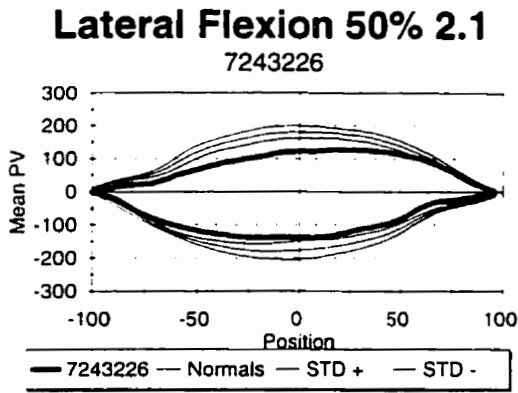


Figure C17B-200 summary performance for Case Study #1 (Bad day), for dynamic lateral flexion at 50% resistance, day 1, test sequence #1.





**Figure C18B-200** summary performance for Case Study #1 (Good day), for dynamic lateral flexion at 50% resistance, day 2, test sequence #1.

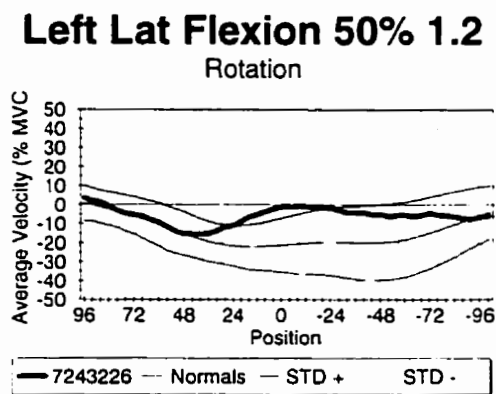
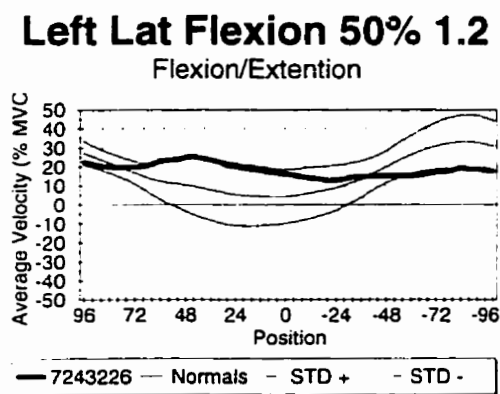
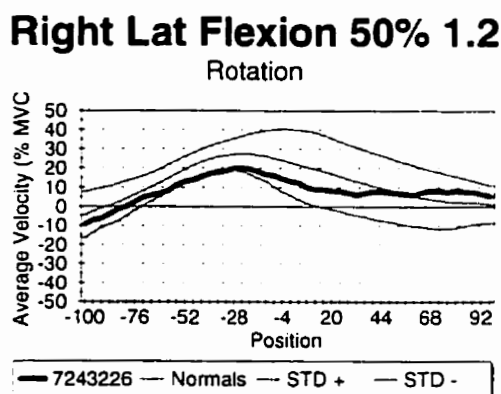
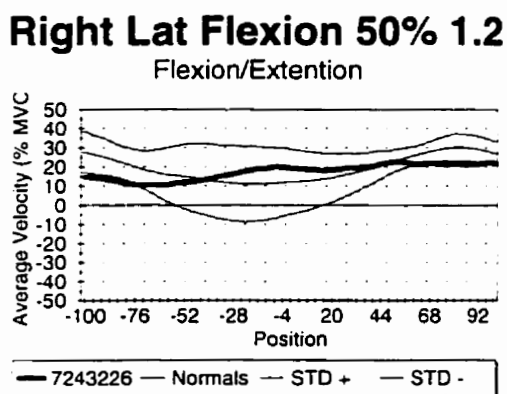
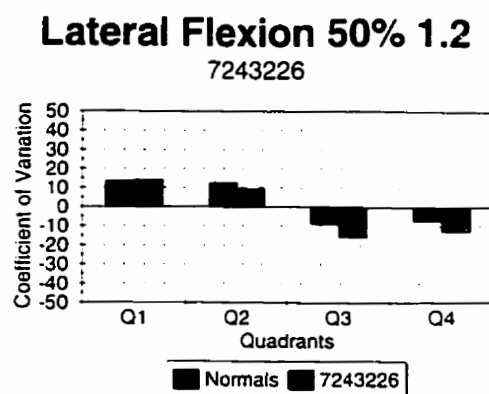
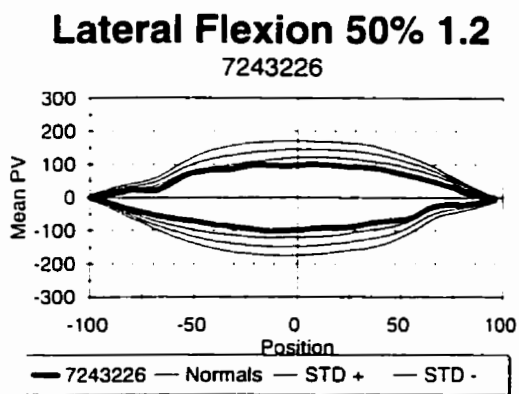
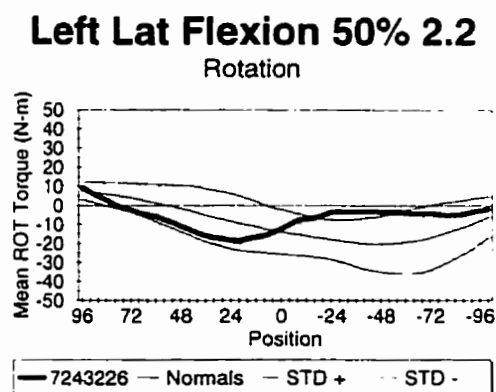
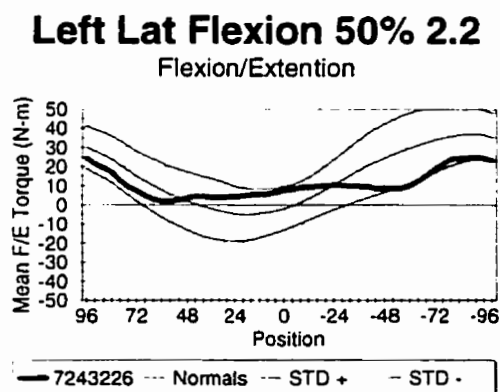
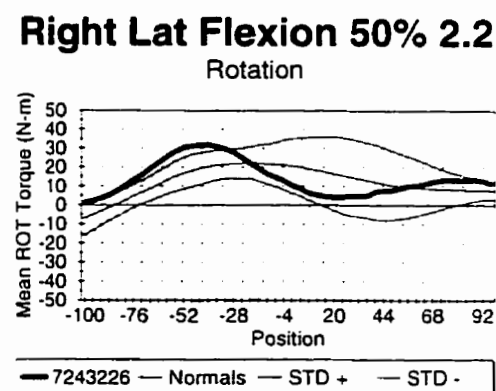
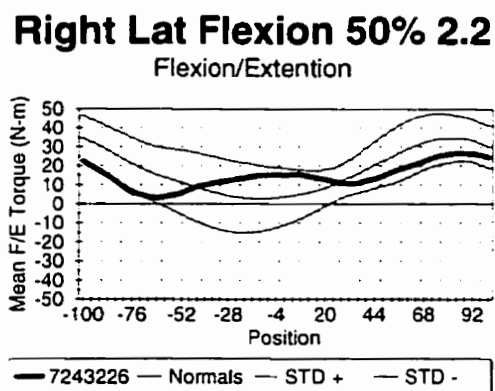
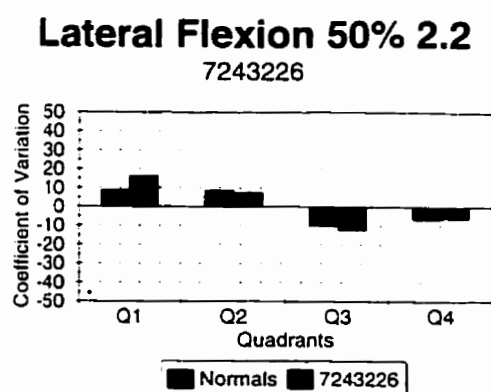
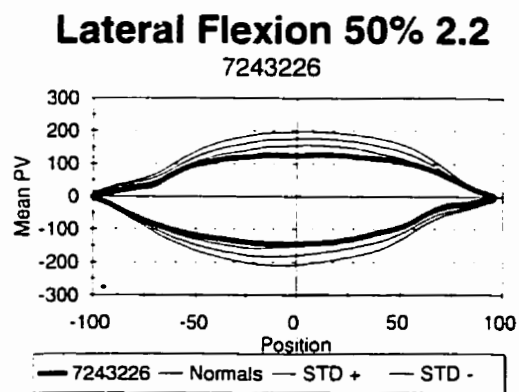
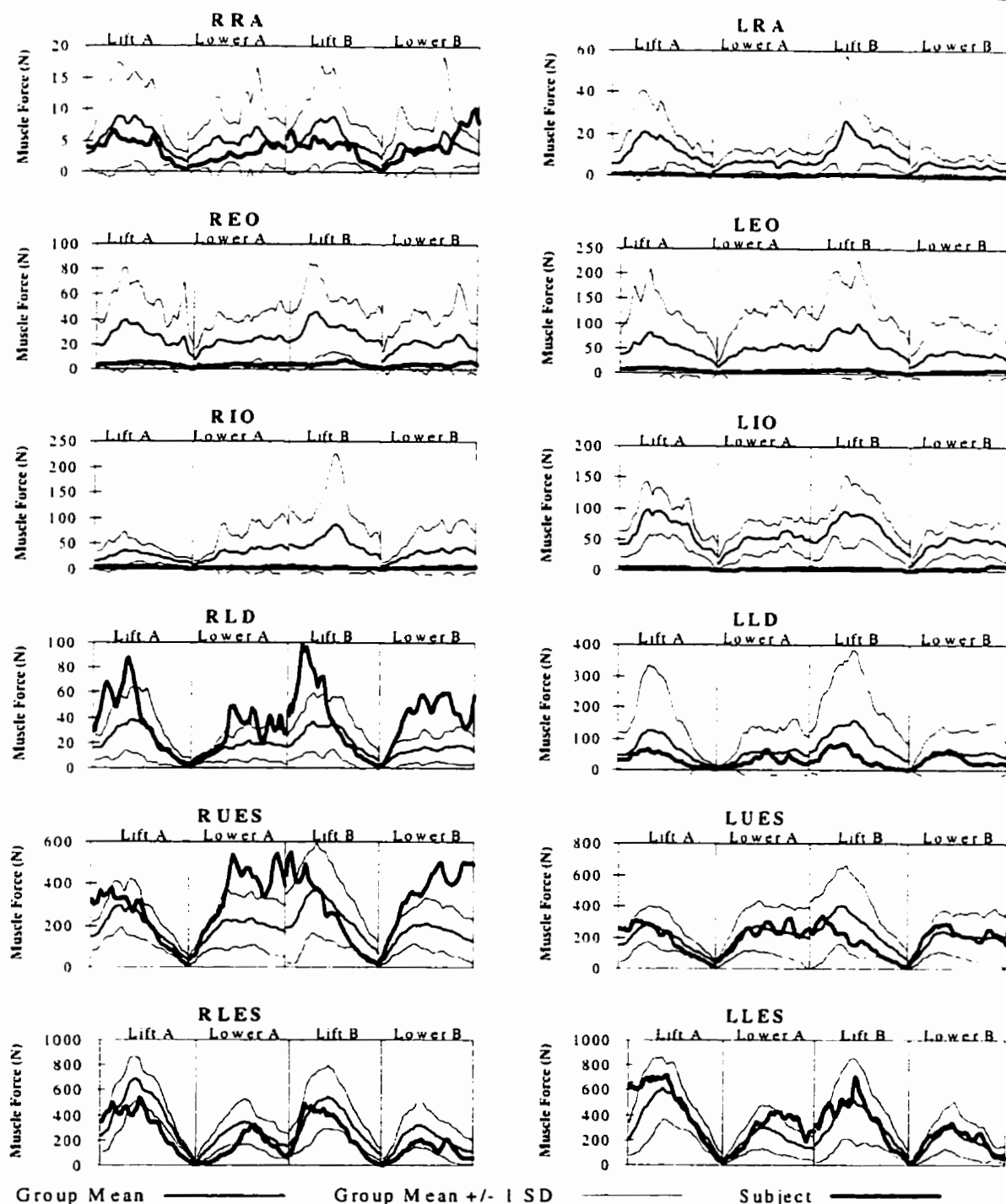


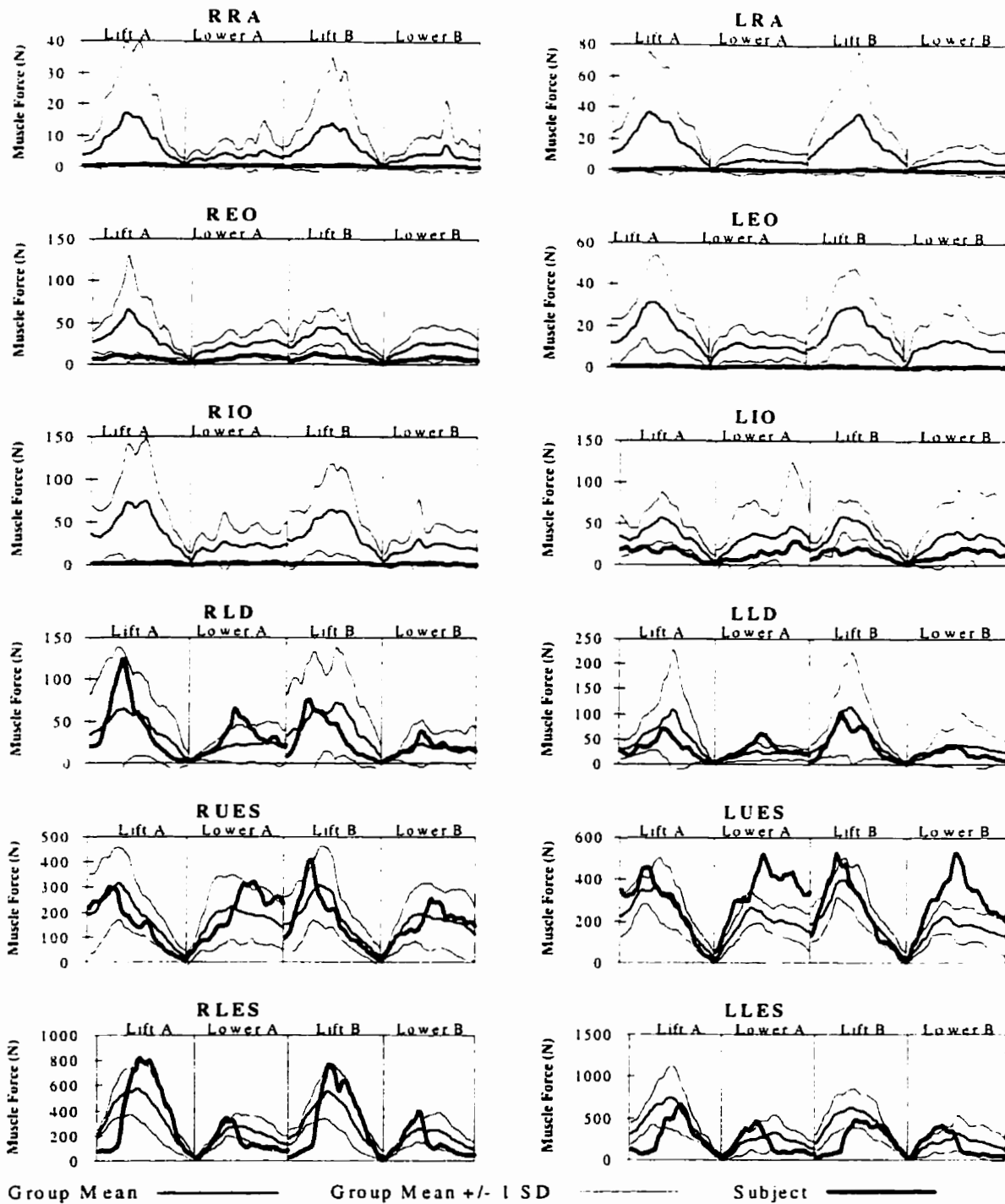
Figure C19B-200 summary performance for Case Study #1 (Bad day), for dynamic lateral flexion at 50% resistance, day 1, test sequence #2.



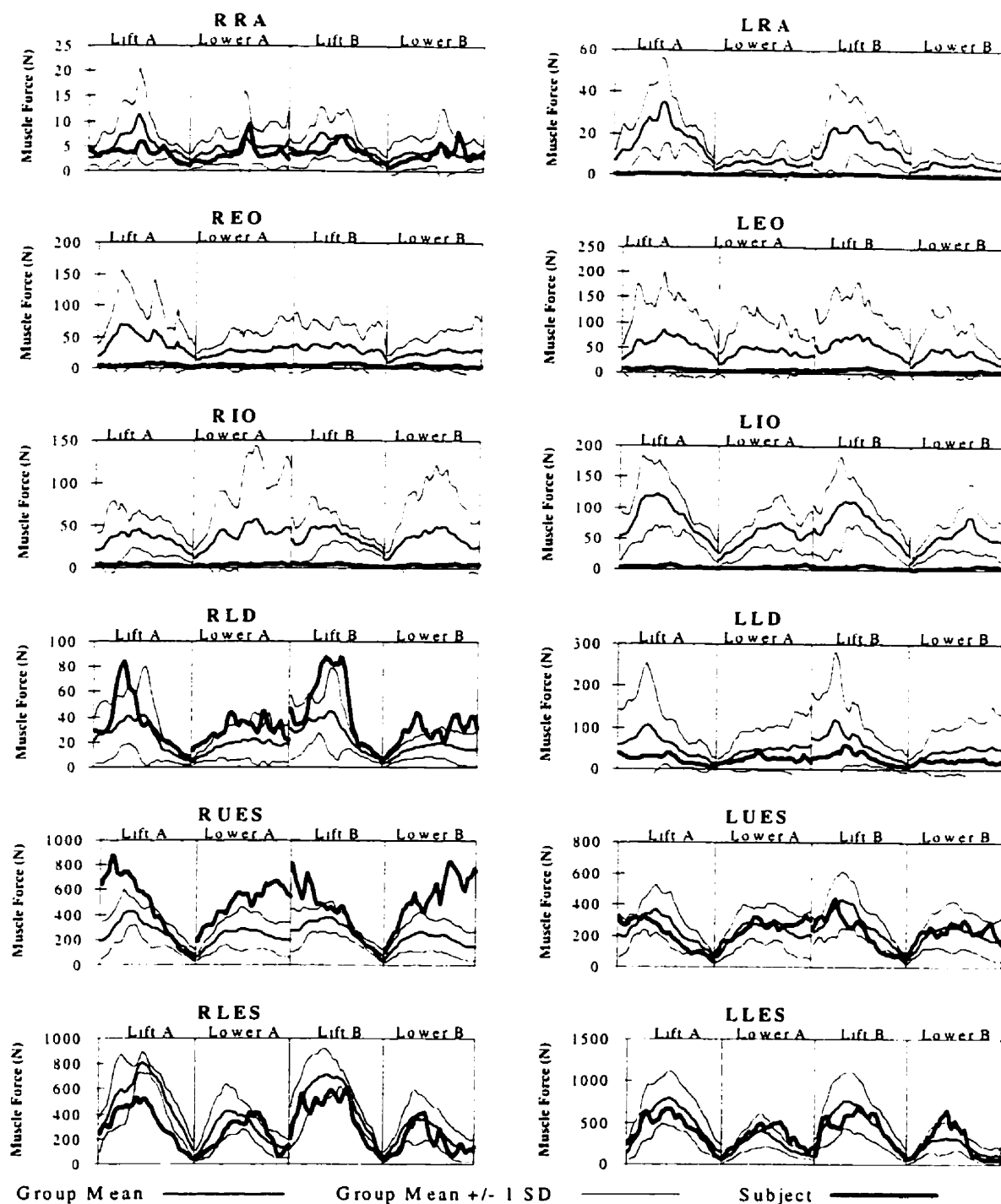
**Figure C20B-200** summary performance for Case Study #1 (Good day), for dynamic lateral flexion at 50% resistance, day 2, test sequence #2.



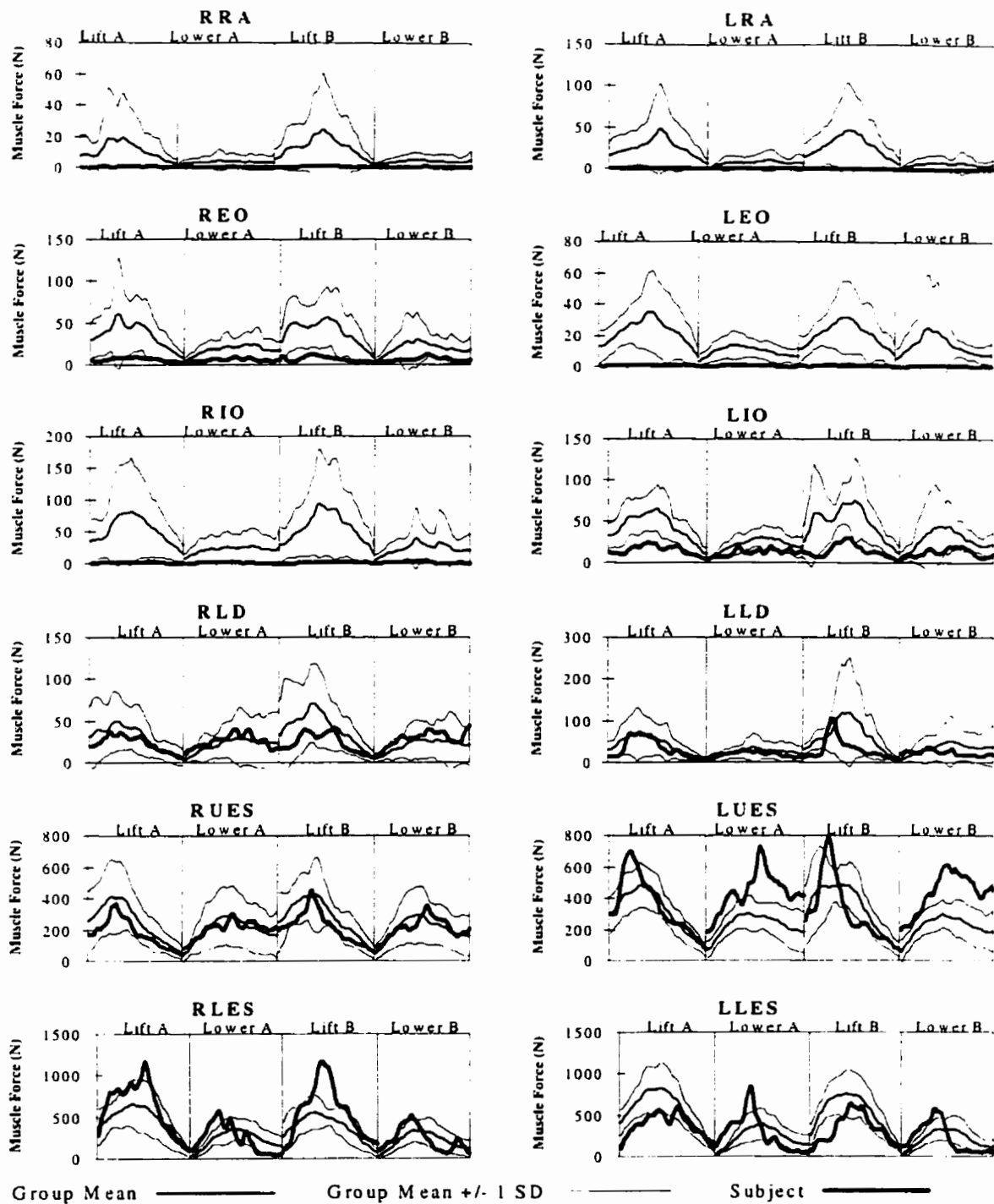
**Figure C21** Case Study #1 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) muscle force (N) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R=right, L=left, RA=rectus abdominis, EO=external oblique, IO=internal oblique, LD=latissimus dorsi, UES=upper erector spinae, LES=lower erector spinae. Note: Day 1 = Bad Day.



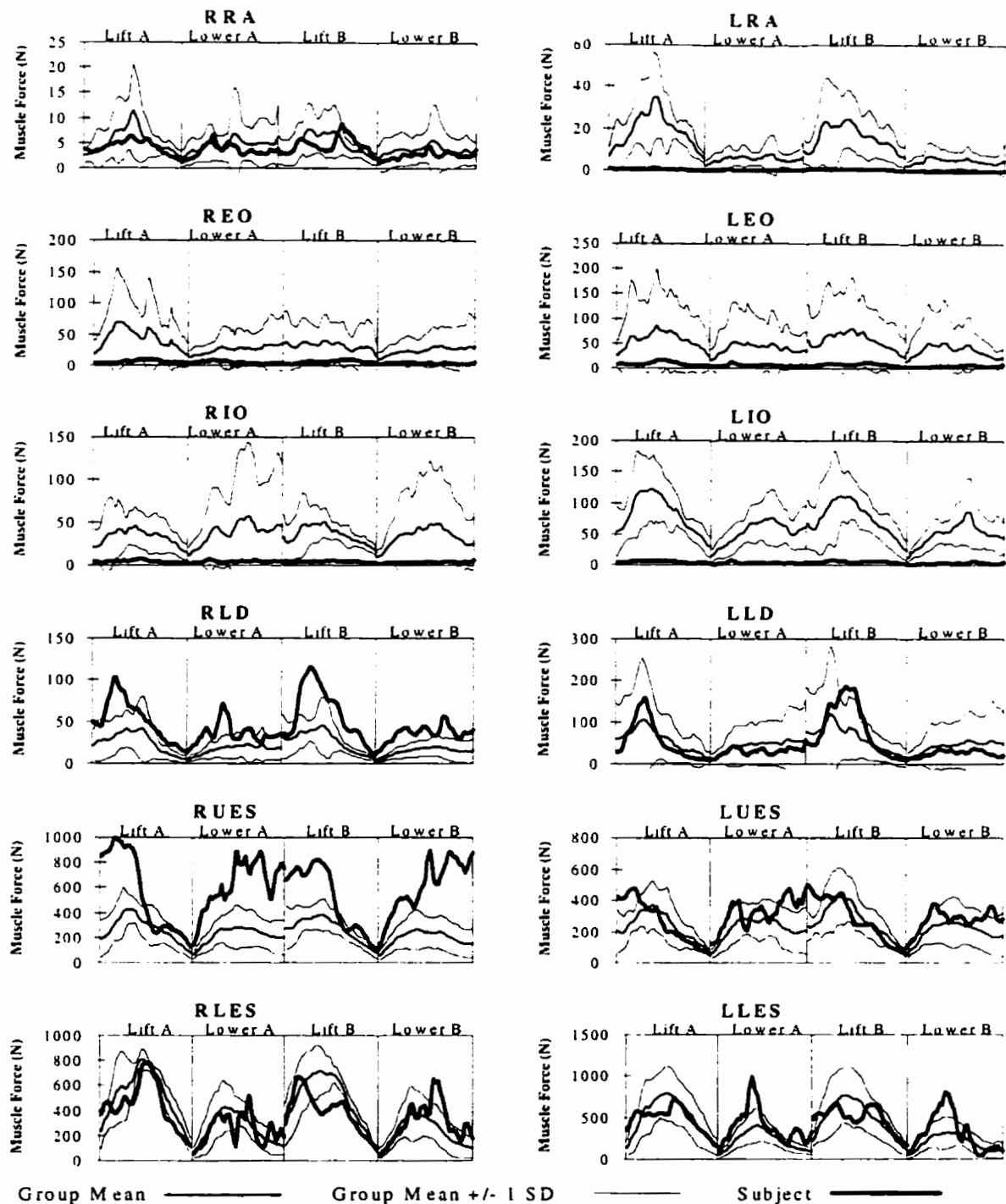
**Figure C22 Case Study #1** graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) muscle force (N) for Day 2, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Good Day.



**Figure C23** Case Study #1 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) muscle force (N) for Day 1, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R = right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Bad Day.

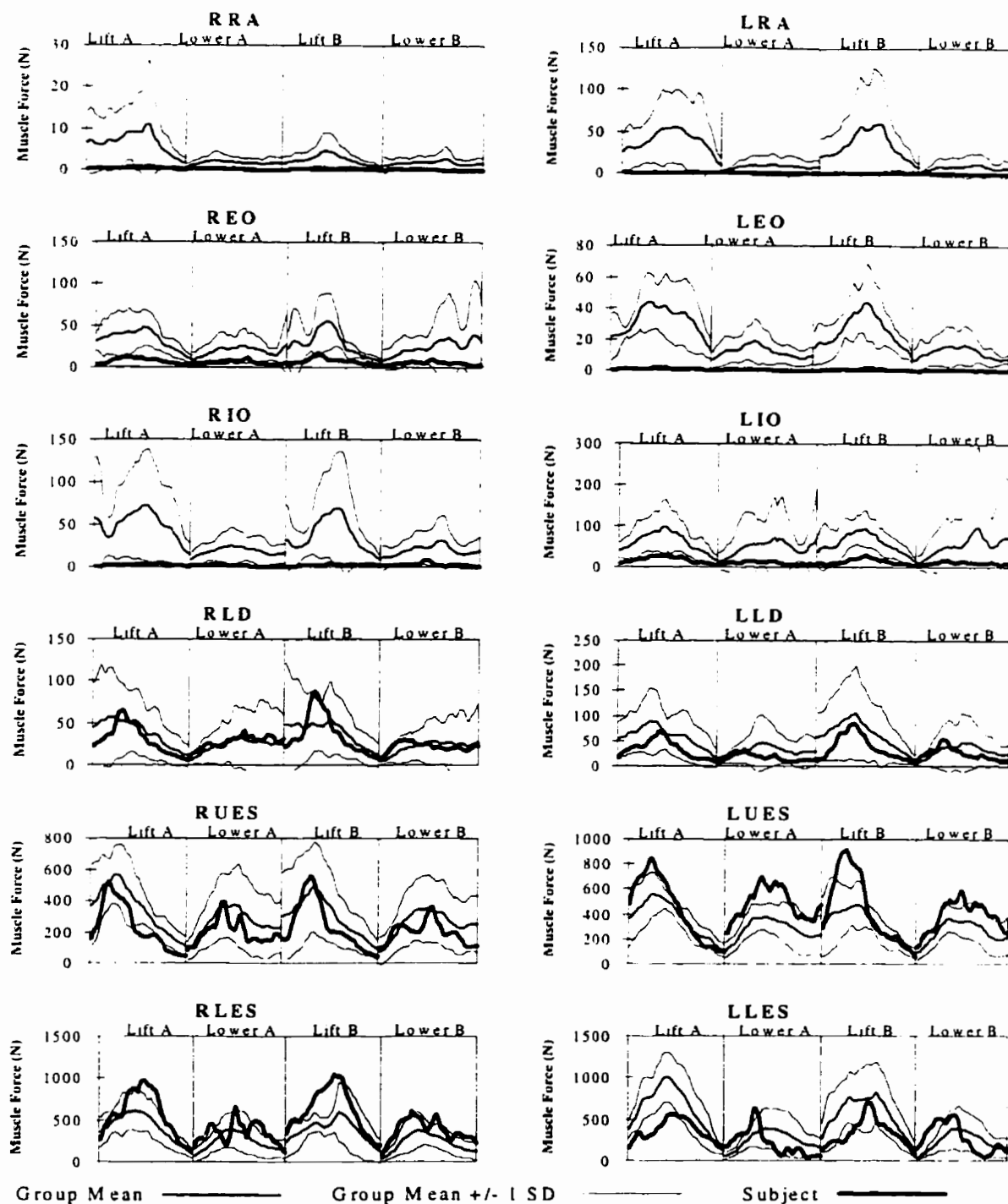


**Figure C24 Case Study #1** graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) muscle force (N) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R = right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Good Day.

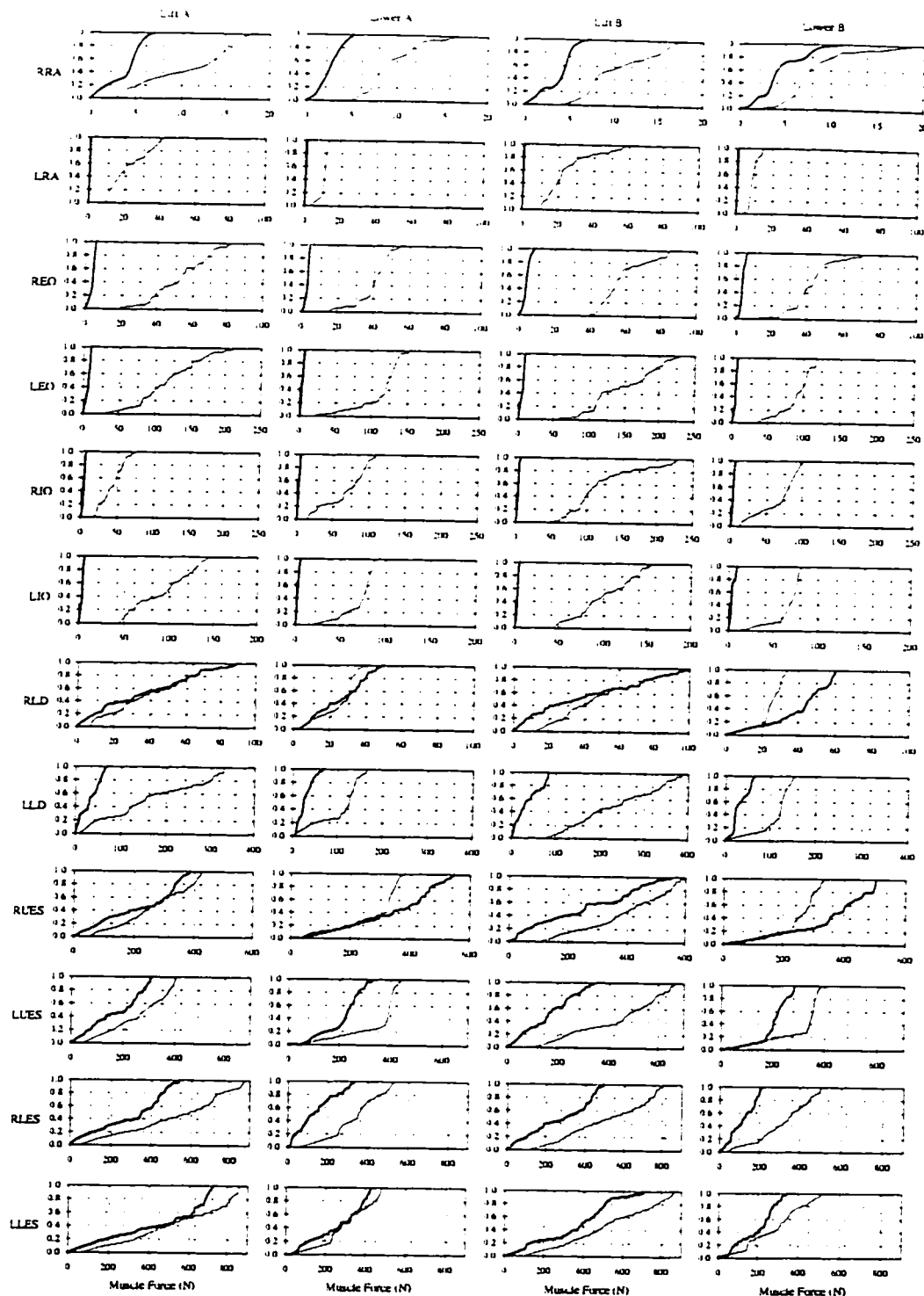


**Figure C25** Case Study #1 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (8 subjects) muscle force (N) for Day 1, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Bad Day.

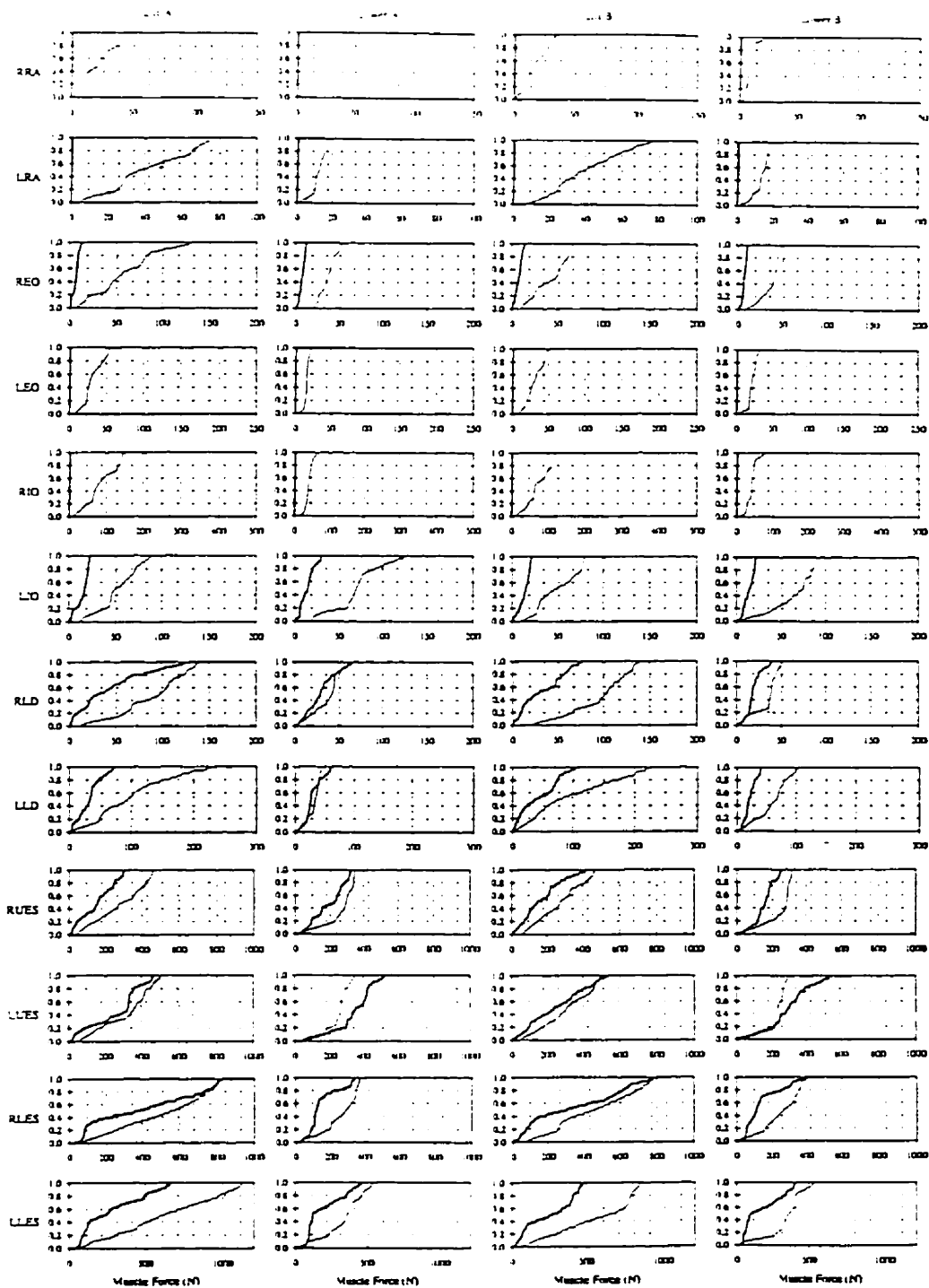




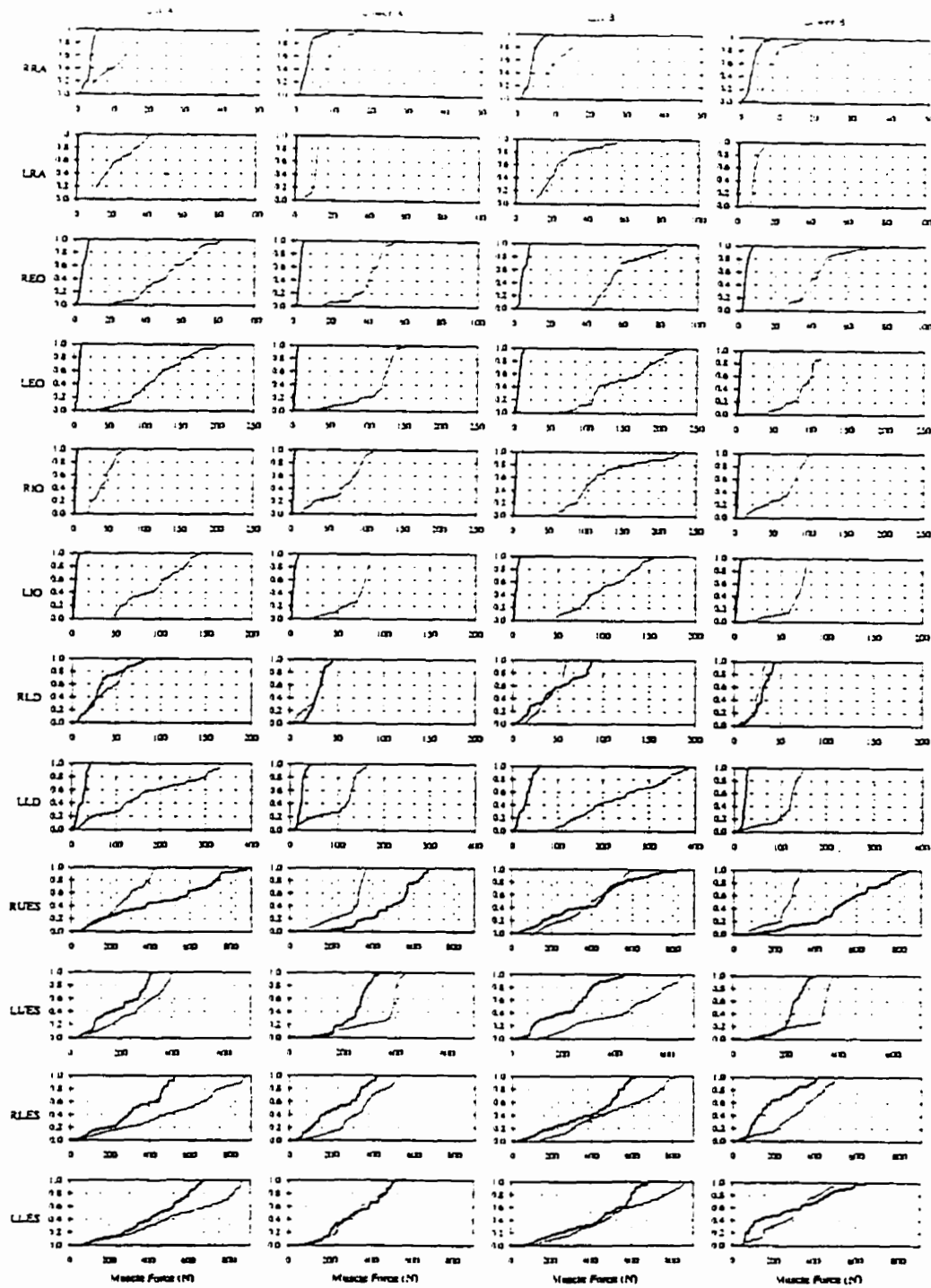
**Figure C26** Case Study #1 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (8 subjects) muscle force (N) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Good Day.



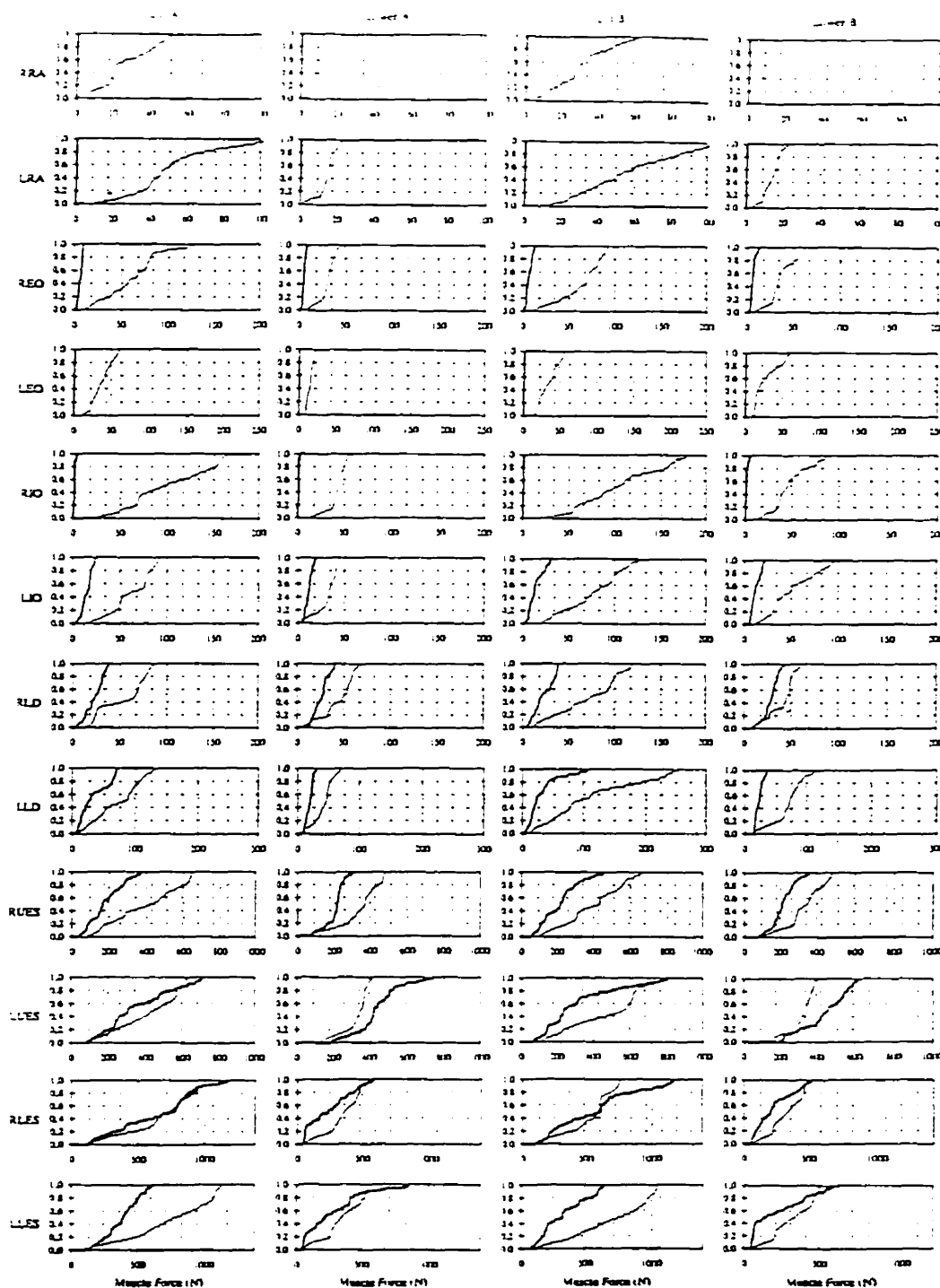
**Figure C27** Summary APDF profile of the muscle forces (N) by each electrode site for Day 1, 0 kg load and Case Study #1. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 9$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 1 = Bad Day.



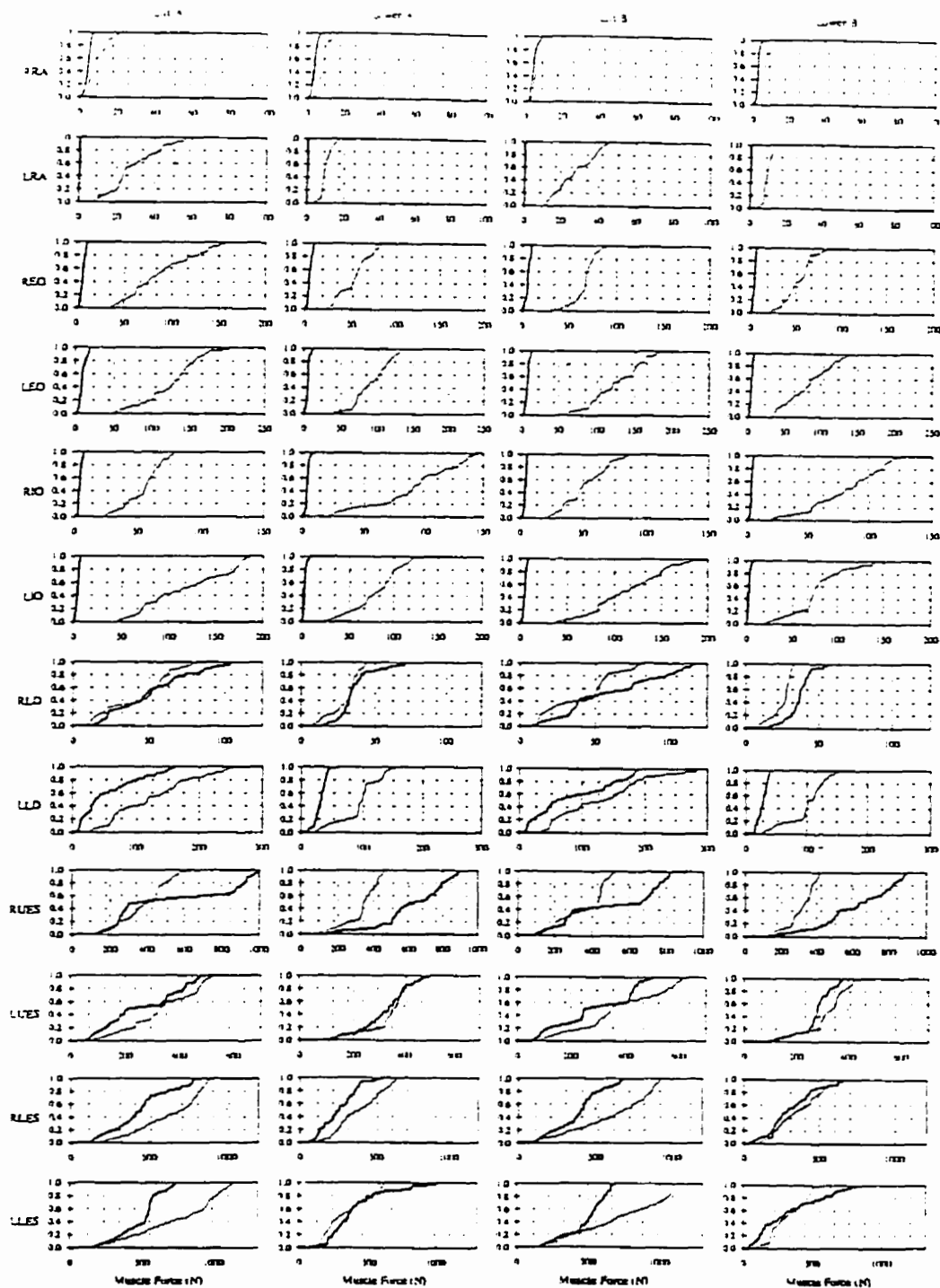
**Figure C28** Summary APDF profile of the muscle forces (N) by each electrode site for Day 2, 0 kg load and Case Study #1. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP (n = 10 subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 2 = Good Day.



**Figure C29** Summary APDF profile of the muscle forces (N) by each electrode site for Day 1, 5 kg load and Case Study #1. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 9$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 1 = Bad Day.

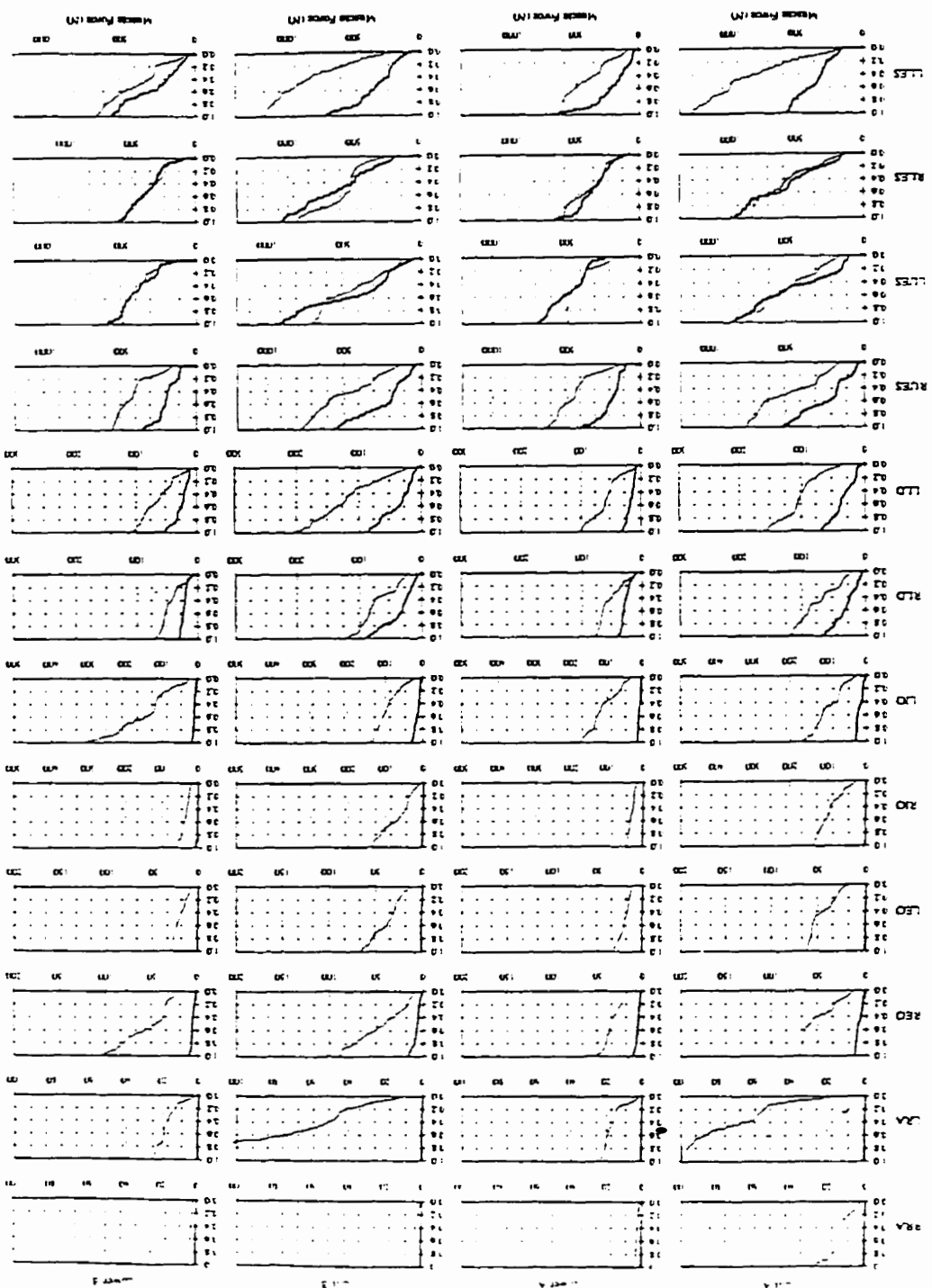


**Figure C30** Summary APDF profile of the muscle forces (N) by each electrode site for Day 2, 5 kg load and Case Study #1. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 9$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 2 = Good Day.



**Figure C31** Summary APDF profile of the muscle forces (N) by each electrode site for Day 1, 10 kg load and Case Study #1. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 8$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 1 = Bad Day.

**Figure C32 Summary APDF profile of the muscle forces (N) by each electrode site for Day 2.** 10 kg load and Case Study #1. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP (n = 8 subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 2 = Good Day.



QOC Evaluation Results for

#7243226  
25-OCT-94

Demographic Data

Resistance Settings

Age : 14	Sex : M	Rotation 25% : 20 N-m
Height : 180.3 cm	Weight : 35.4 kg	Rotation 50% : 42 N-m
Diagnosis :		Flex/Ext 25% : 43 N-m
Surgical Category :		Flex/Ext 50% : 98 N-m
Activity Level Category :		Lat Flex 25% : 34 N-m
		Lat Flex 50% : 66 N-m

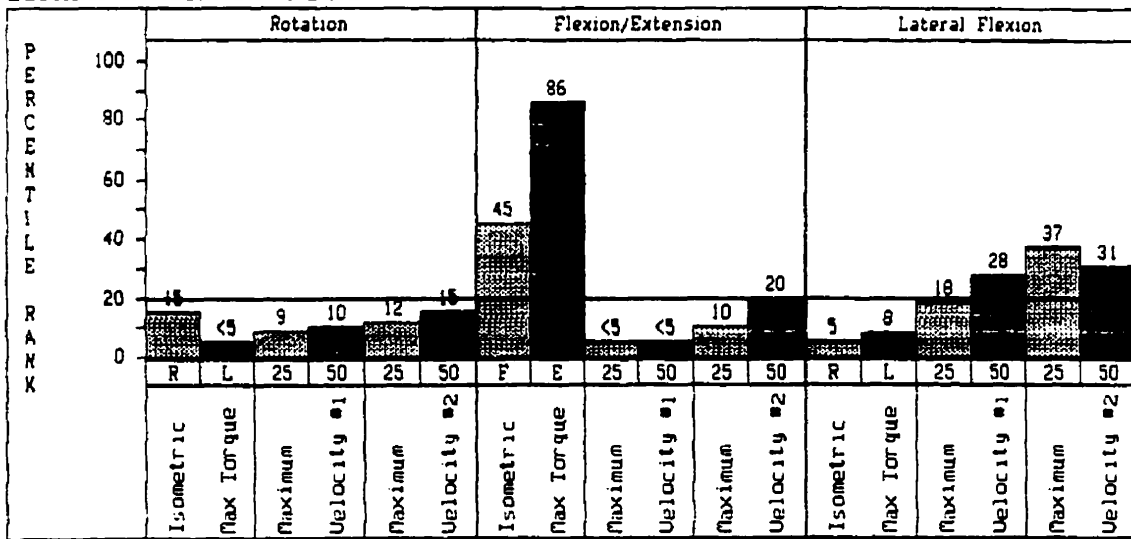
Abnormal Indicators 8

	Rotation		Flex/Ext		Lat Flex	
	25%	50%	25%	50%	25%	50%
Isometric Max Torque						
Max Velocity						
Rotation Sec Max Torque						
Flex/Ext Sec Max Torque						
Lat Flex Sec Max Torque						

Non-physiological Indicators 2

- 1) not observed
- 2) Max velocity 50% greater than or equal 25%, seq 2 : F/E
- 3) not observed
- 4) not observed
- 5) not observed
- 6) ↓ range of motion ratio : F/E

Baseline Rehabilitation Data



Test Administered By : MBF

Signed : \_\_\_\_\_ Date : \_\_\_\_\_

Figure C33 Isostation B-200 report (page 1) for Case Study #1 on Day 1 (Bad Day).



## OOC Evaluation Results for

#7243226

25-OCT-94

OOC Test	Rotation	Flex/Ext	Lat Flex
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## Range of Motion

ROM (deg) #1	84.3	96.9	69.1
#2	78.8	74.8	64.8
ROM #2 (Δ%)			
ROM #1	-6.52	-13.82↓	-6.22

## Isometric

Max Torque (N-m)	89.1	175.3	299.4	133.5
------------------	------	-------	-------	-------

## Dynamic

		25%	50%	25%	50%	25%	50%
Resisted ROM (Δ%)	#1	-7.89	-14.04	-13.81	-25.97	5.06	4.43
	#2	13.62↑	8.92	12.18	4.49	22.97↑	-5.41
Avg Vel 50% (Δ%)	#1	-9.23		-4.27		2.77	
	#2	-23.15		-20.94		-31.84	
Max Velocity (deg/sec)	#1	96.9	88.9	97.5↓	100.2↓	112.9	123.1
	#2	119.7	94.9	127.3	127.3	156.5	115.4

## Secondary Axes

Rot Secondary	#1	--	--	19.2	27.7	19.2	32.0
Max Torq (N-m)	#2	--	--	10.7	21.3	14.9↓	32.0
F/E Secondary	#1	49.2	76.9	--	--	52.3	78.7
Max Torq (N-m)	#2	40.0	36.9	--	--	40.0	110.7
L F Secondary	#1	47.4	64.6	21.5	21.5	--	--
Max Torq (N-m)	#2	71.1	75.4	12.9	21.5	--	--

## Key

↓ Subnormal, less than critical level  
 ↑ Supernormal, greater than critical level  
 (see Statistical Review in OOC Back Evaluation System)

Version 3.0

Figure C34 Isostation B-200 report (page 2) for Case Study #1 on Day 1 (Bad Day).

OOE Evaluation Results for

#7243226  
05-JAN-95

Demographic Data

Resistance Settings

Age : 14	Sex : M	Rotation 25% : 20 N-m
Height : 180.3 cm	Weight : 35.4 kg	Rotation 50% : 42 N-m
Diagnosis :		Flex/Ext 25% : 31 N-m
Surgical Category :		Flex/Ext 50% : 61 N-m
Activity Level Category :		Lat Flex 25% : 31 N-m
		Lat Flex 50% : 62 N-m

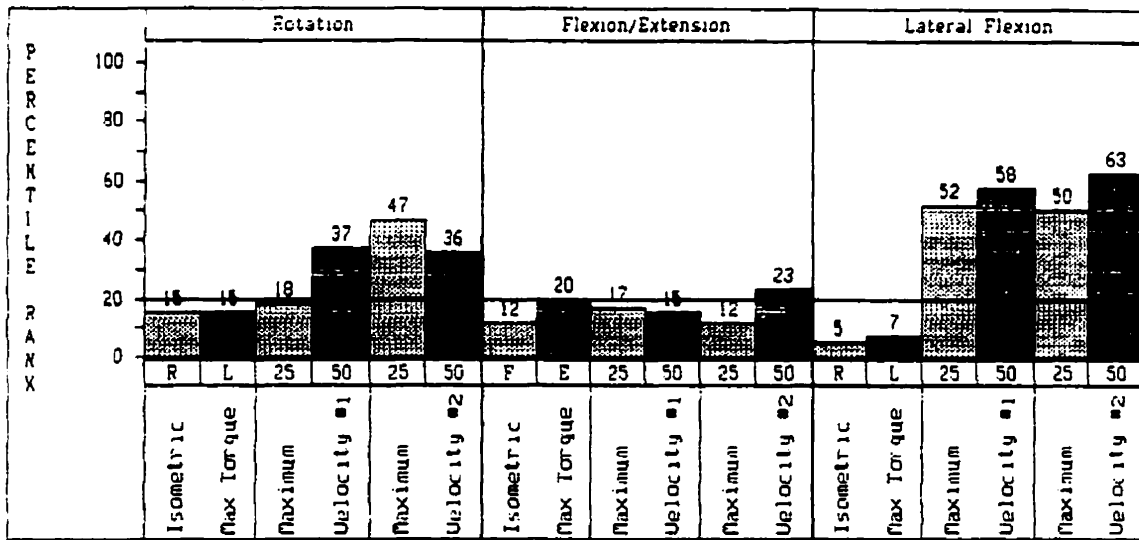
Abnormal Indicators

	Rotation		Flex/Ext		Lat Flex	
	25%	50%	25%	50%	25%	50%
Isometric Max Torque						
Max Velocity						
Rotation Sec Max Torque						
Flex/Ext Sec Max Torque						
Lat Flex Sec Max Torque						

Non-physiological Indicators

- 1) not observed
- 2) not observed
- 3) not observed
- 4) not observed
- 5) not observed
- 6) not observed

Baseline Rehabilitation Data



Test Administered By : KBF

Signed : \_\_\_\_\_ Date : \_\_\_\_\_

Figure C35 Isostation B-200 report (page 1) for Case Study #1 on Day 2 (Good Day).

## OOE Evaluation Results for

■7243226  
05-JAN-95

OOE Test	Rotation	Flex/Ext	Lat Flex
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## Range of Motion

ROM (deg) #1	66.2	72.9	53.8
#2	79.5	82.0	71.8
ROM #2 (Δ%)	20.09	12.48	33.46†

## Isometric

Max Torque (N-m)	93.1	123.0	193.7	124.9
------------------	------	-------	-------	-------

## Dynamic

	25%	50%	25%	50%	25%	50%
Resisted ROM (Δ%) #1	36.87	33.52	0.00	1.97	35.77†	23.58†
ROM #2	18.60†	17.21†	8.19	5.26	14.02	0.00
Avg Vel 50% (Δ%) #1	-14.01		-2.79		-12.93	
Avg Vel 25% #2	-21.82		-20.05		-19.35	
Max Velocity (deg/sec) #1	122.5	124.5	135.4	127.3	164.2	148.6
#2	150.2	118.6	138.1	135.4	177.0	156.5

## Secondary Axes

Rot Secondary #1	--	--	10.7	23.5	32.0	55.4
Max Torq (N-m) #2	--	--	14.9	19.2	46.9	49.0
F/E Secondary #1	33.8	40.0	--	--	33.8	58.4
Max Torq (N-m) #2	49.2	46.1	--	--	70.7	46.1†
L F Secondary #1	51.7	84.0	15.1	38.8†	--	--
Max Torq (N-m) #2	66.8	73.2	17.2	12.9	--	--

## Key

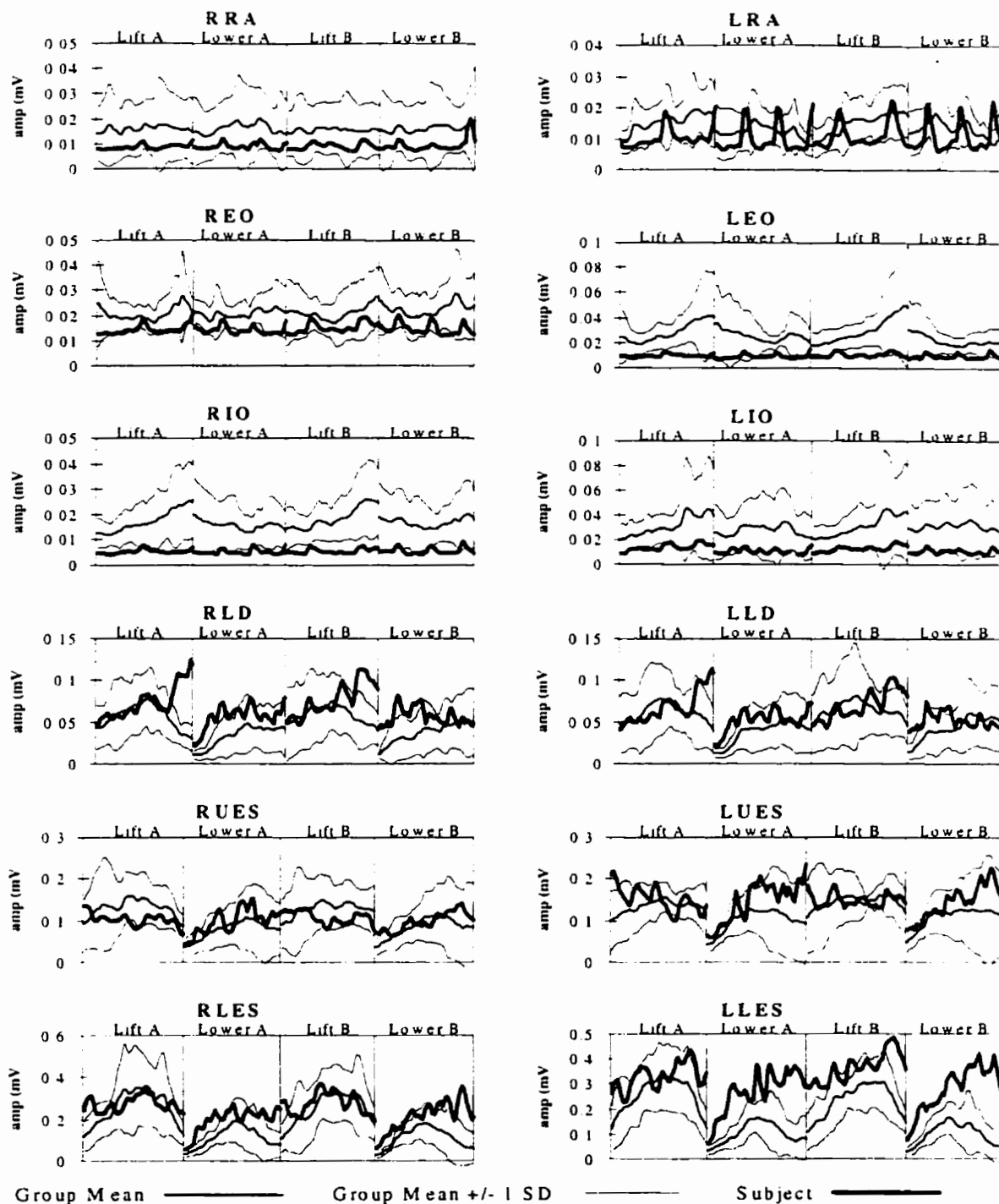
- † Subnormal, less than critical level  
 ‡ Supernormal, greater than critical level  
 (see Statistical Review in OOE Back Evaluation System)

Version 3.0

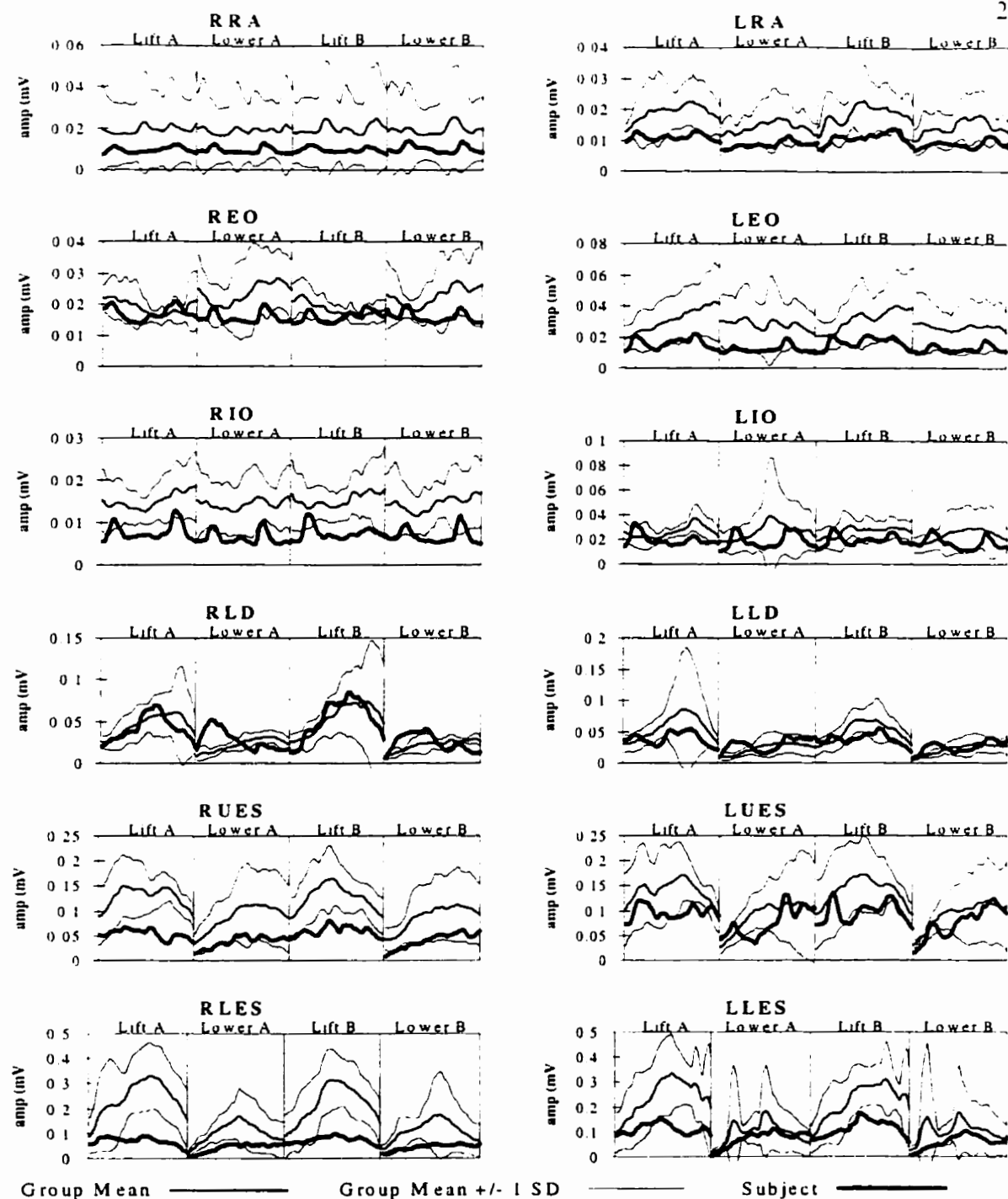
Figure C36 Isostation B-200 report (page 2) for Case Study #1 on Day 2 (Good Day).

## **Appendix D**

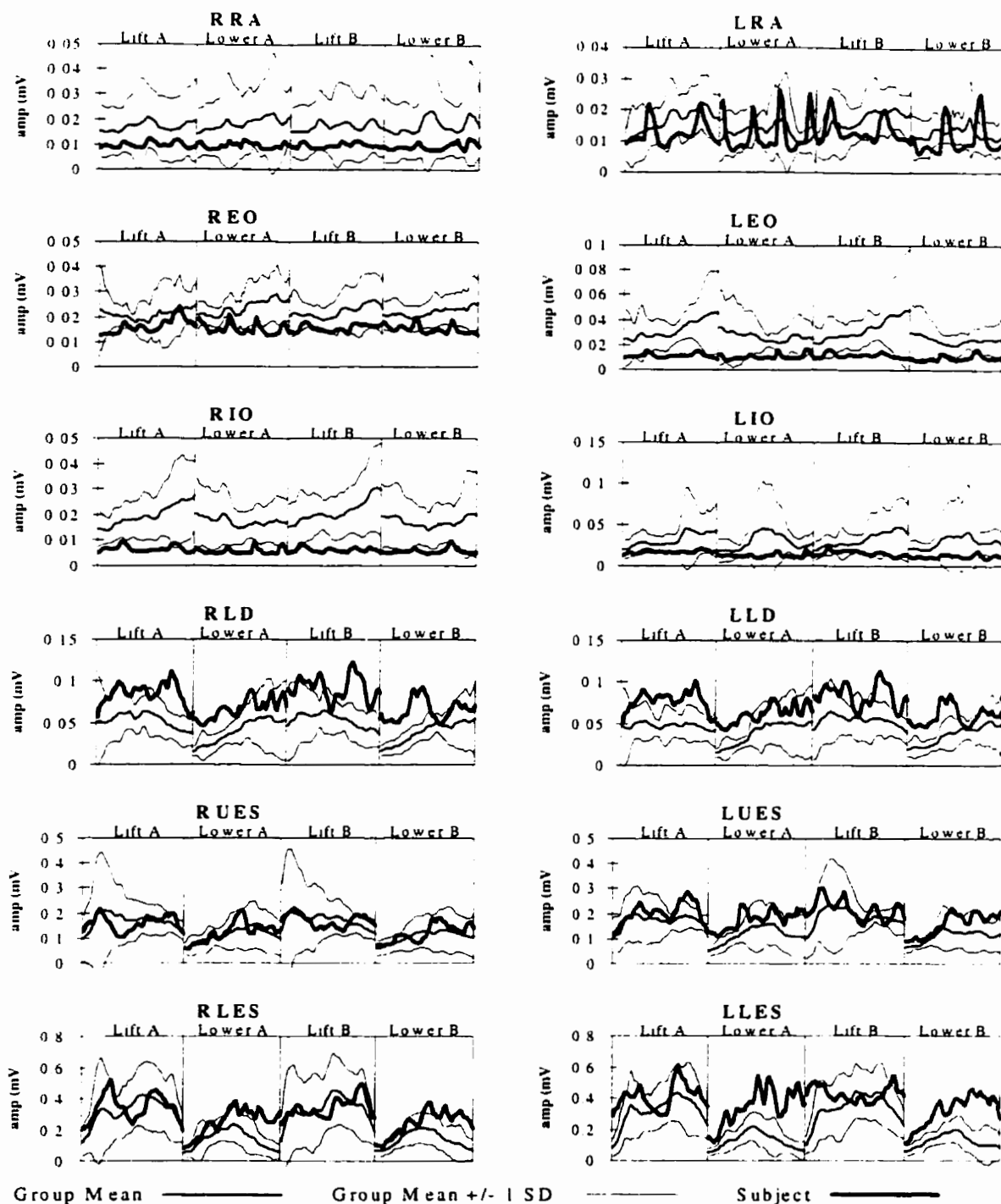
Case Study #2



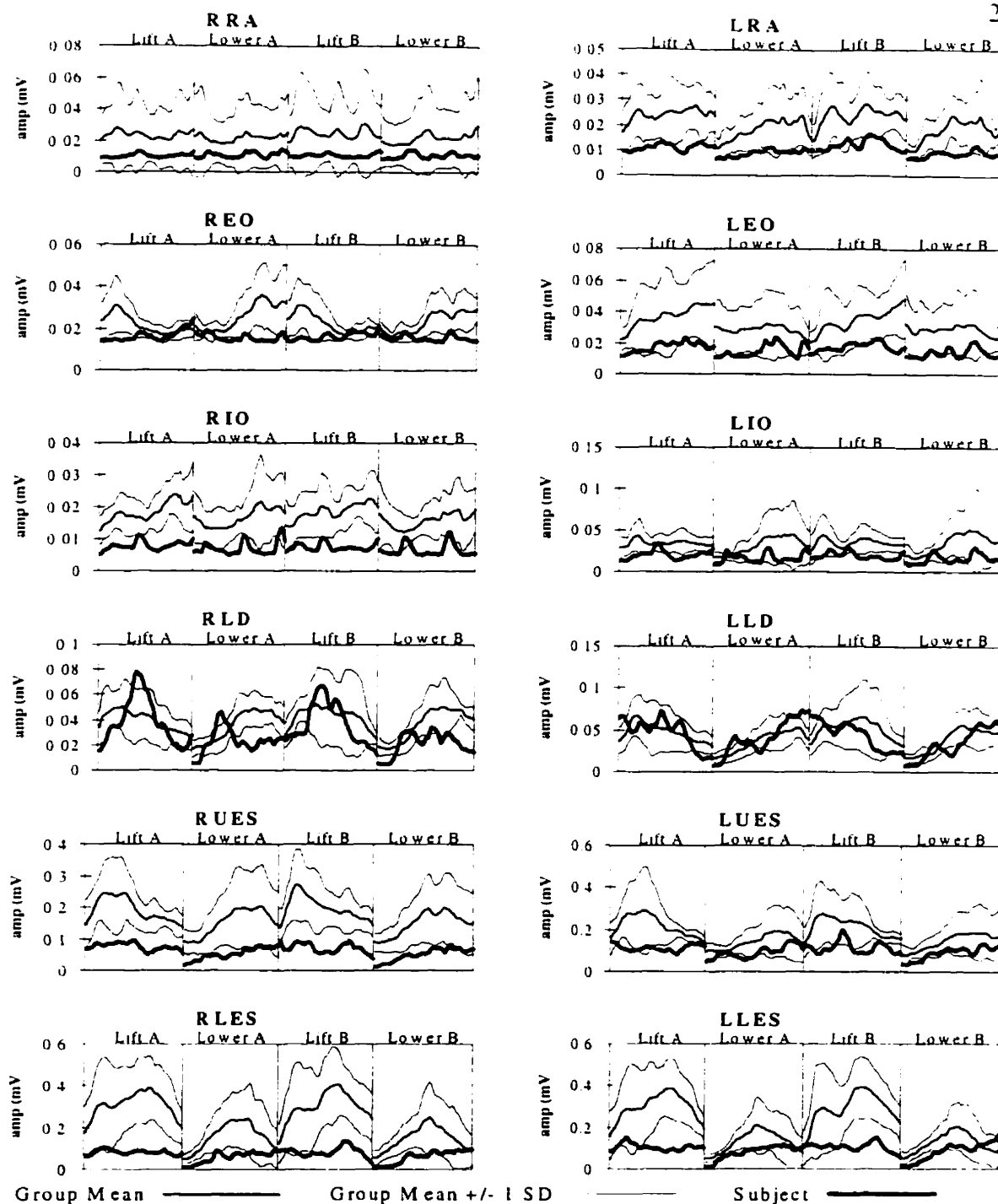
**Figure D1** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (mV) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Good Day.



**Figure D2** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (mV) for Day 2, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.

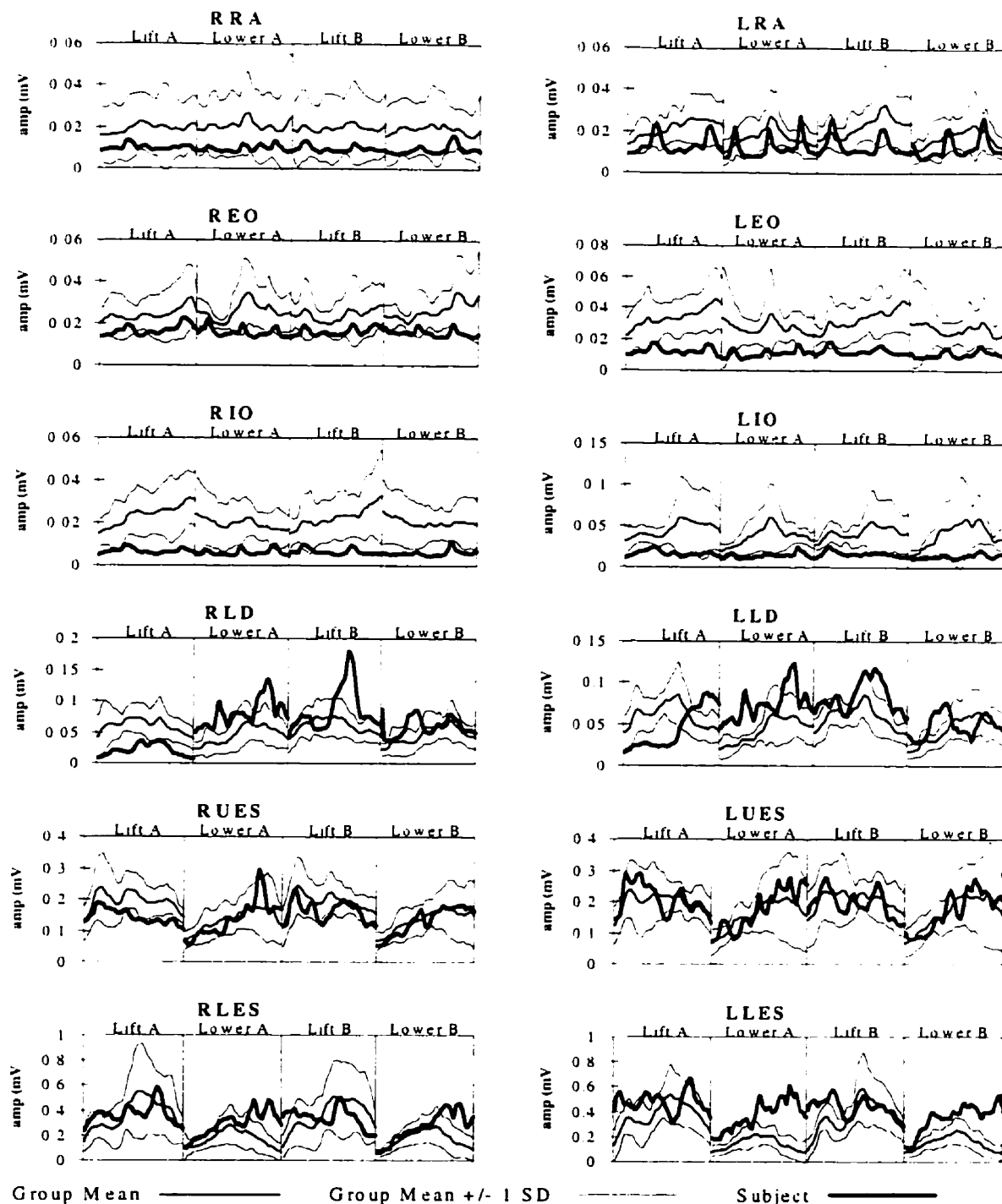


**Figure D3** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (mV) for Day 1, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Good Day.

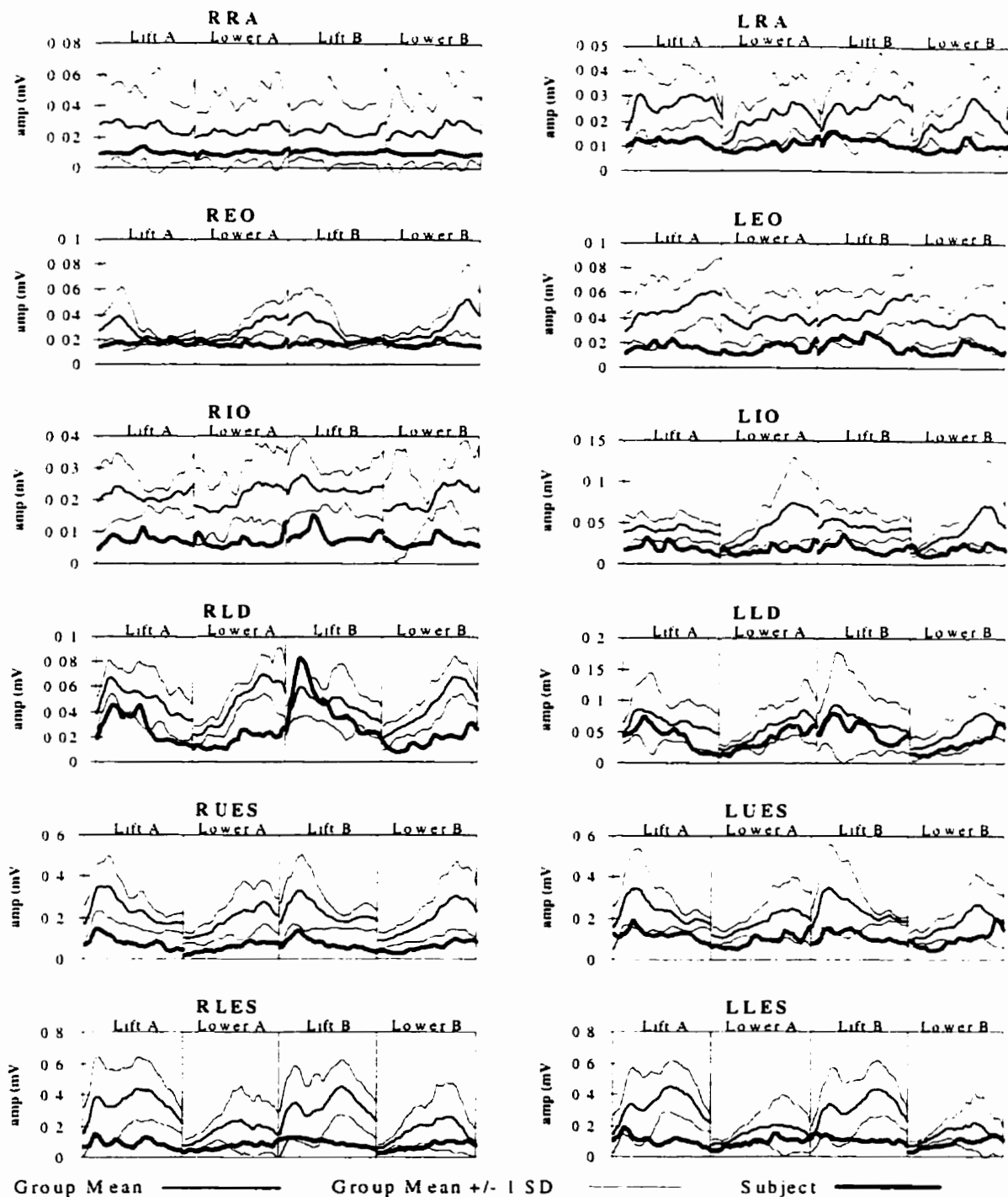


**Figure D4** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (mV) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.

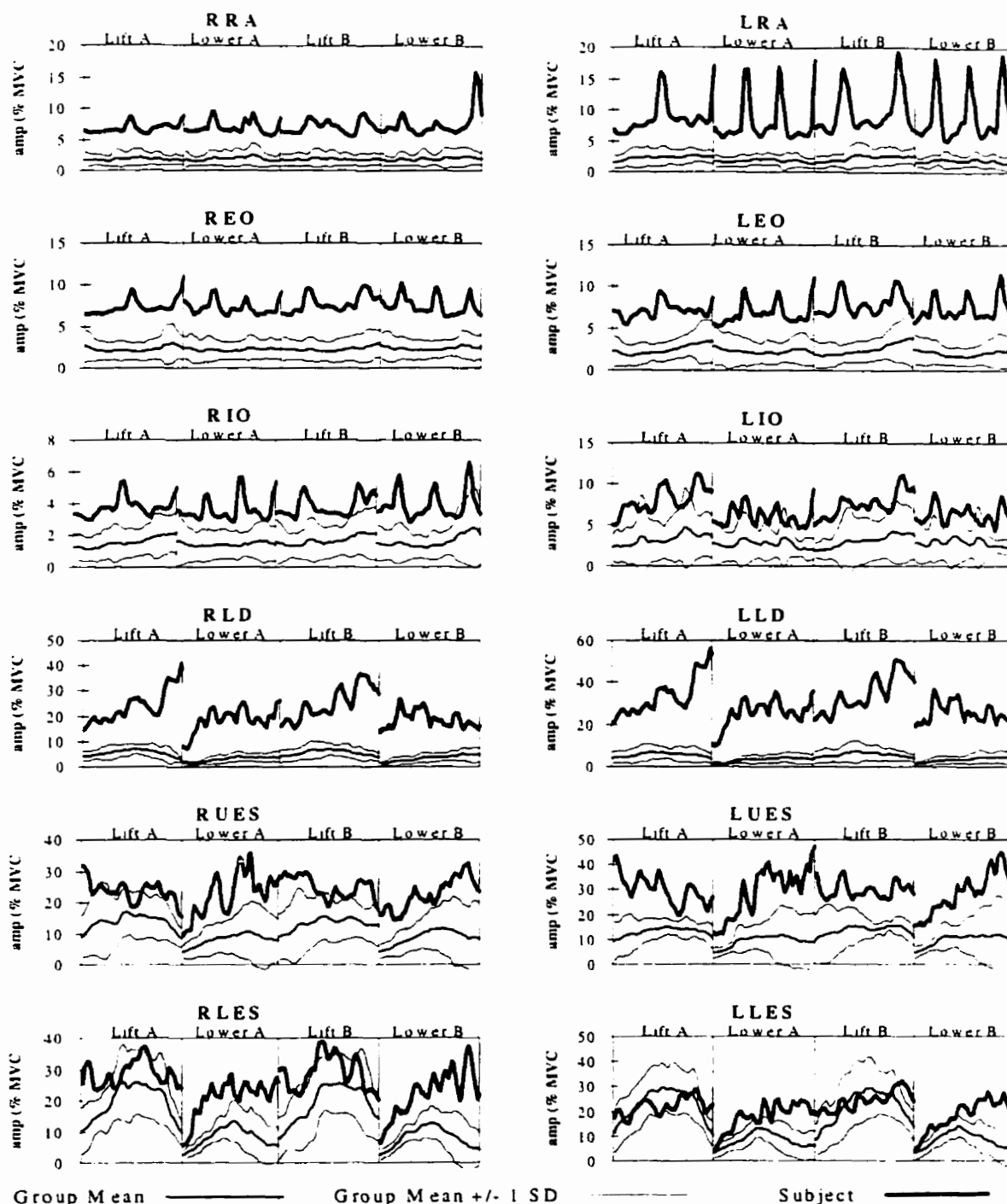




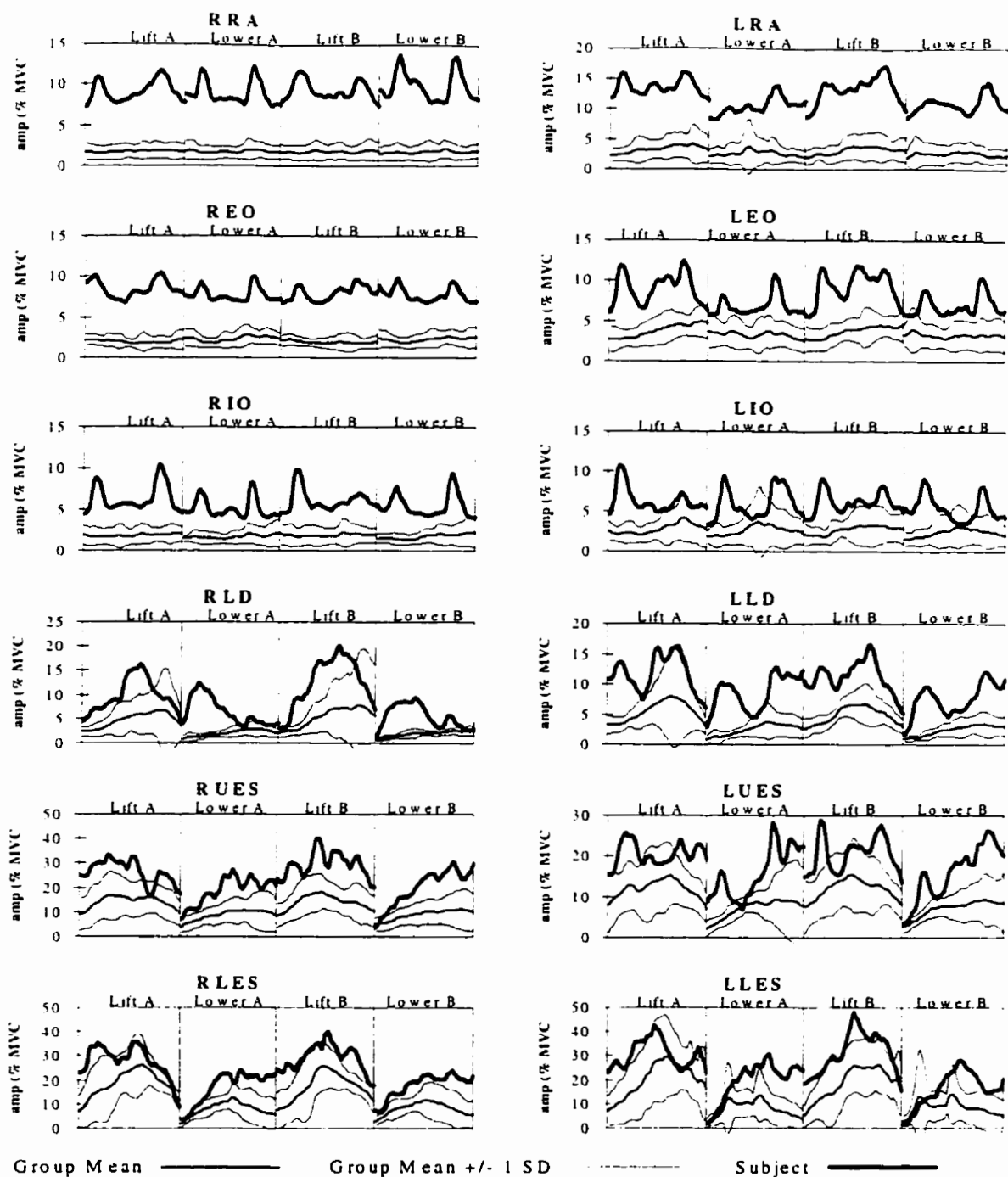
**Figure D5** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (mV) for Day 1, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Good Day.



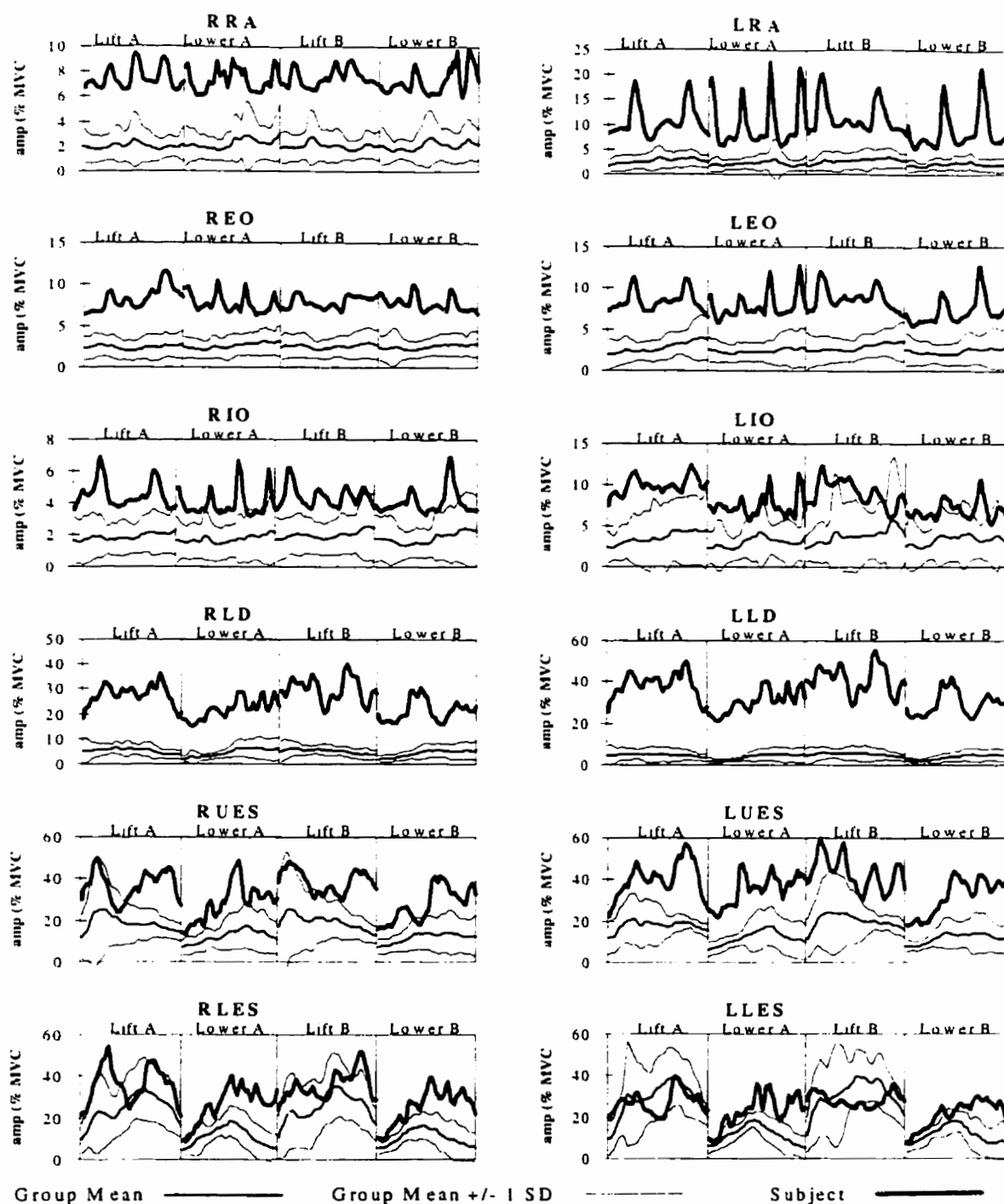
**Figure D6** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (mV) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.



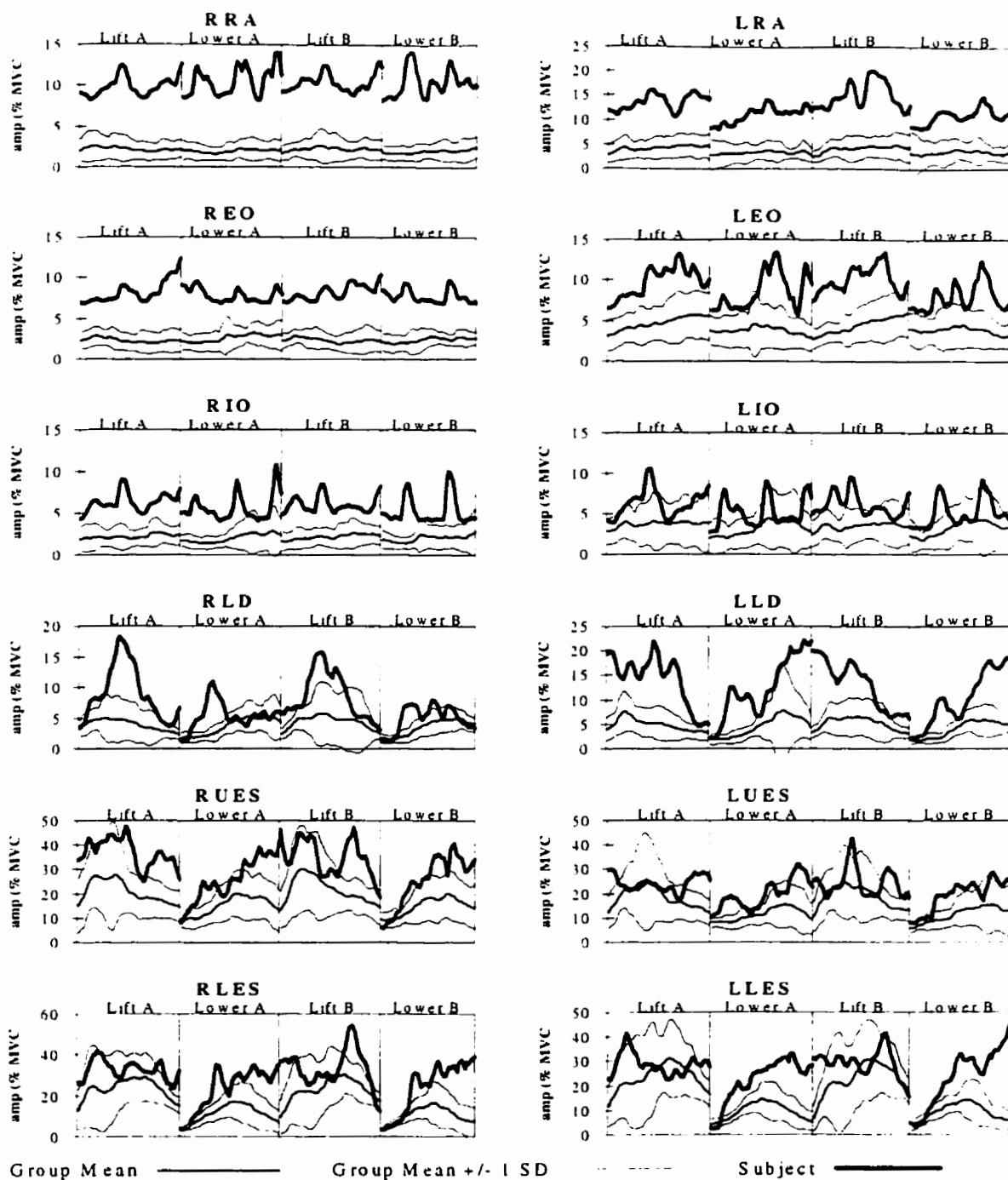
**Figure D7** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (% MVC) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Good Day.



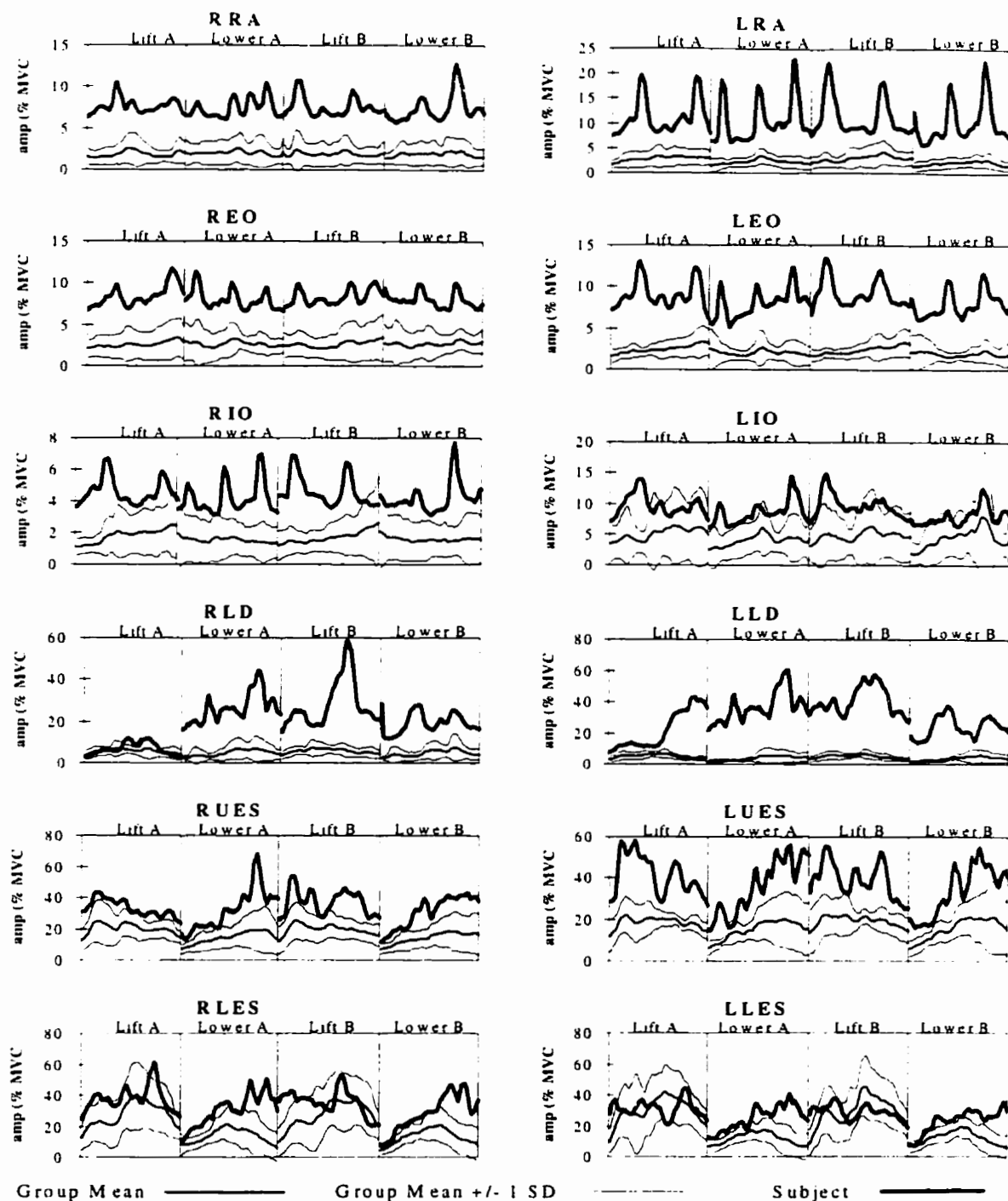
**Figure D8** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 2, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.



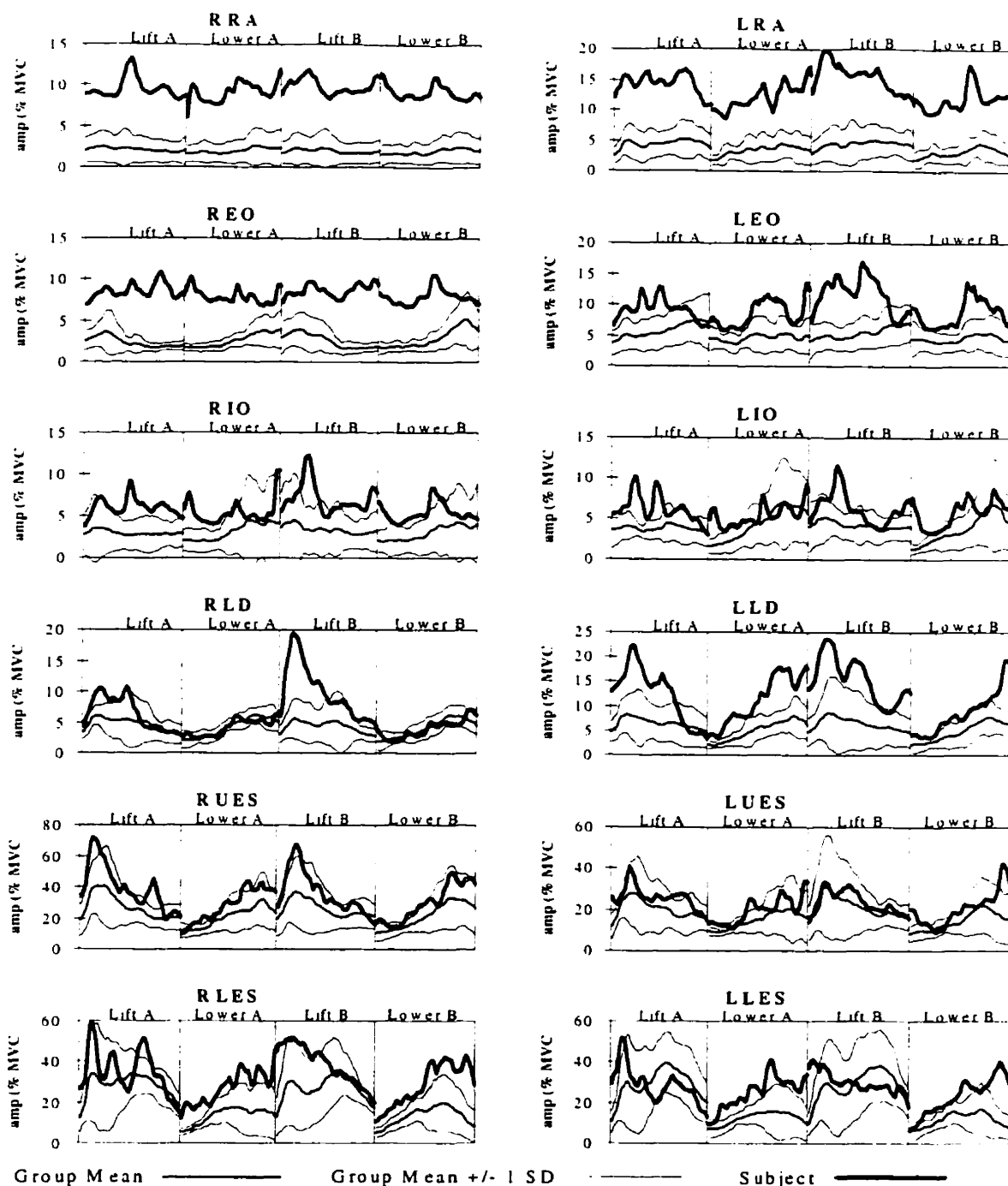
**Figure D9** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (% MVC) for Day 1, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R = right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Good Day.



**Figure D10** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.



**Figure D11** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (% MVC) for Day 1, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Good Day.



**Figure D12** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.



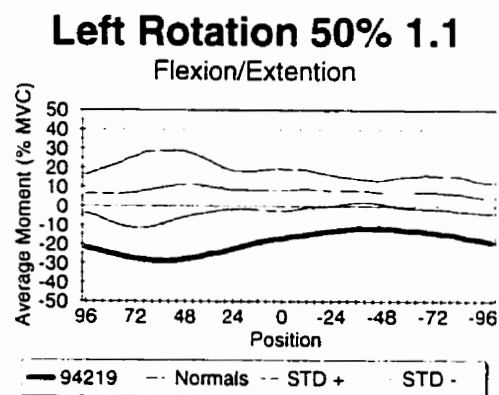
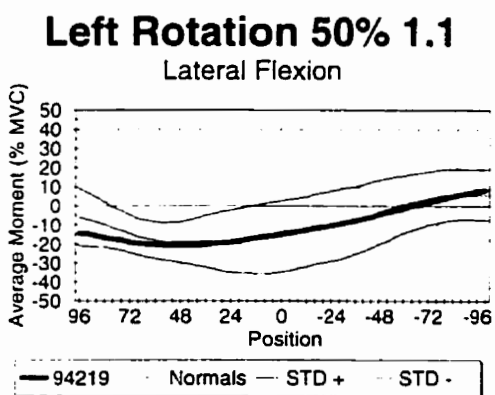
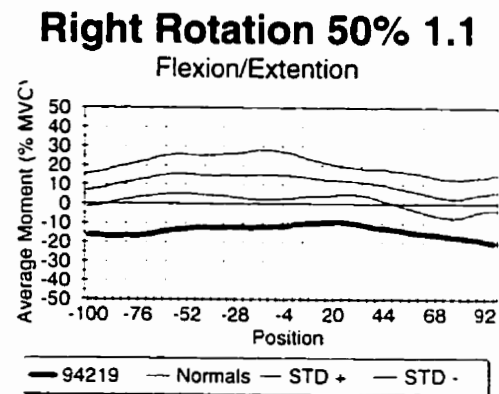
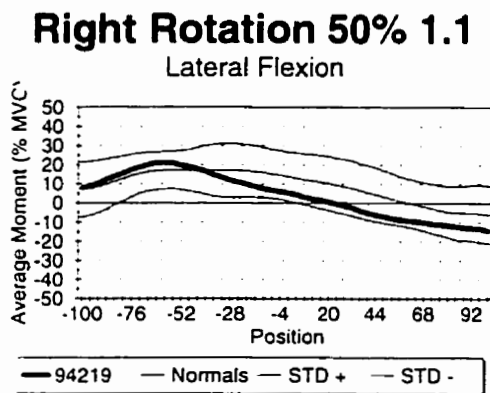
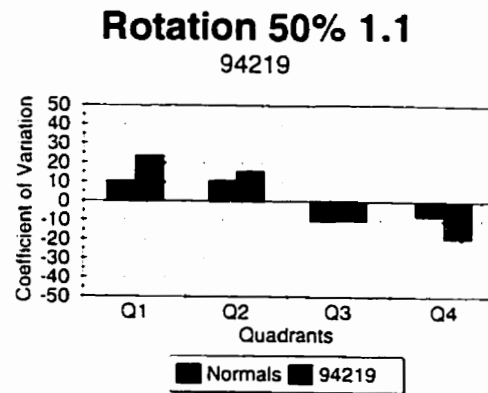
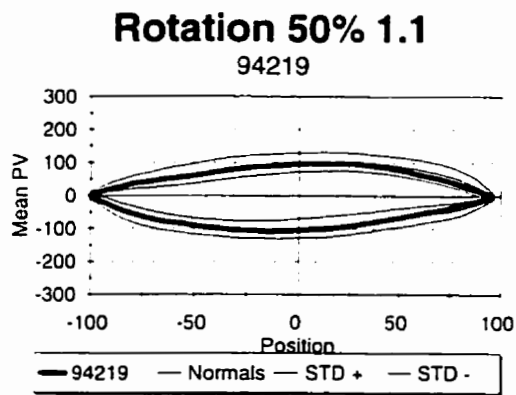
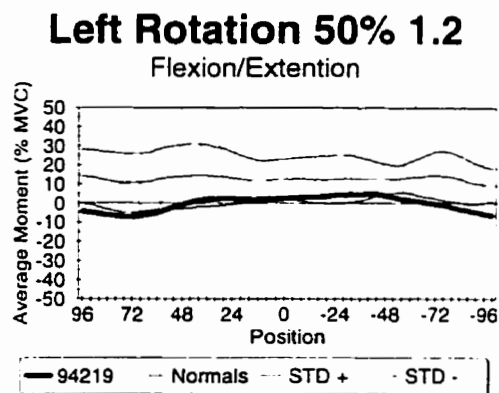
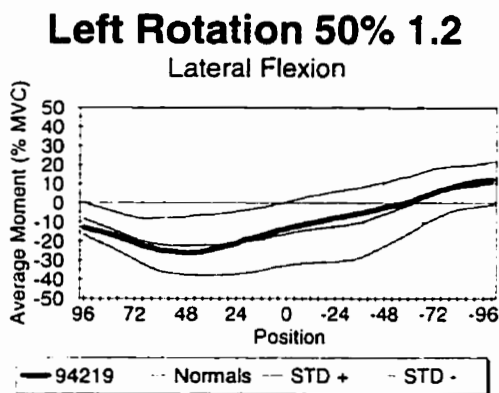
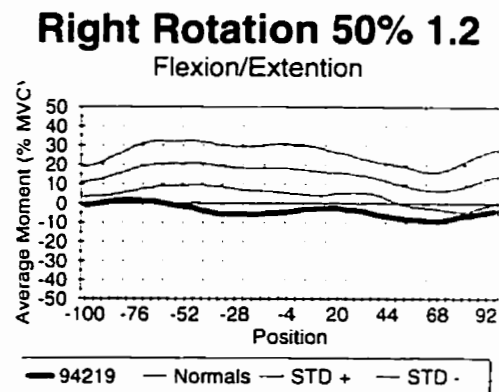
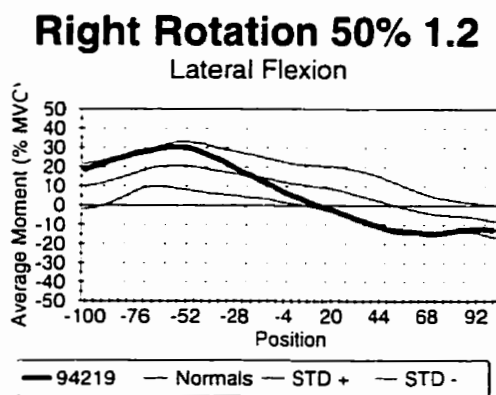
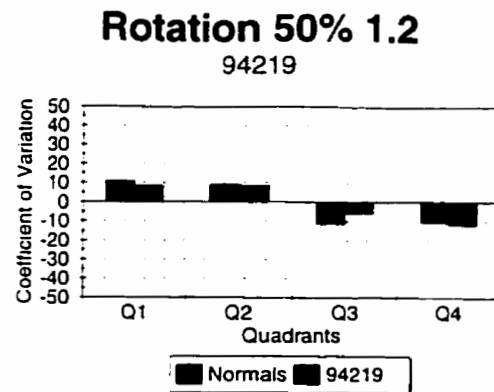
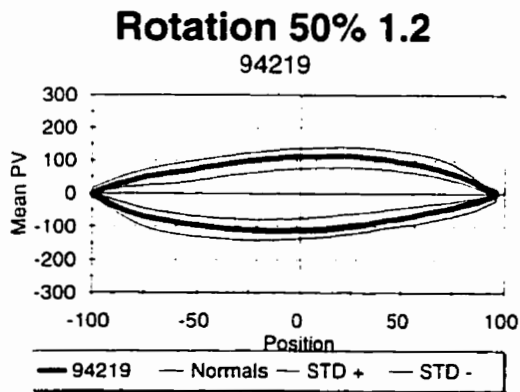


Figure D13 B-200 summary performance for Case Study #2 (Good day), for dynamic rotation at 50% resistance, day 1, test sequence #1.



**Figure D14 B-200** summary performance for Case Study #2 (Good day), for dynamic rotation at 50% resistance, day 1, test sequence #2.

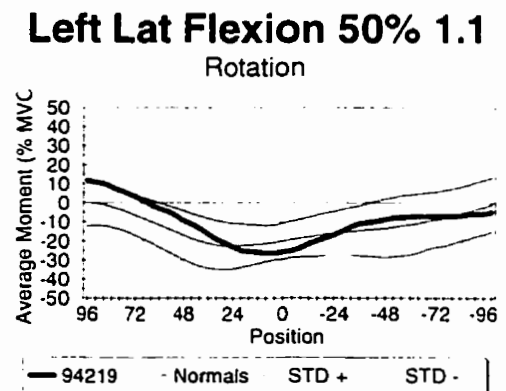
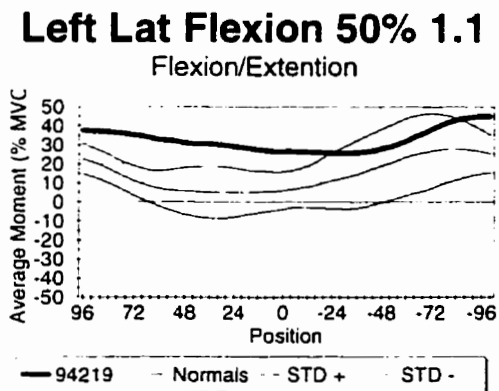
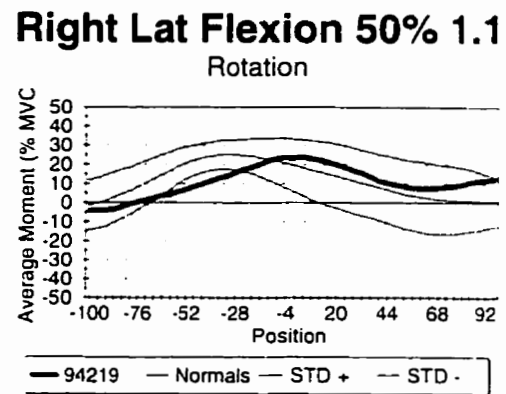
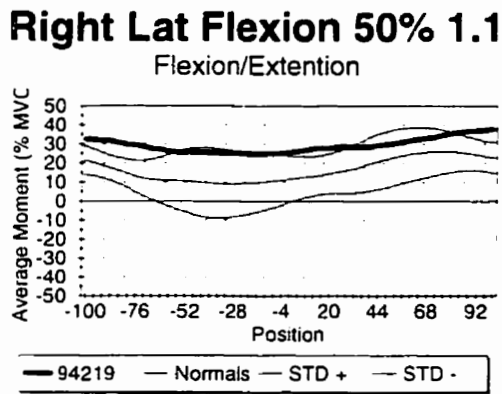
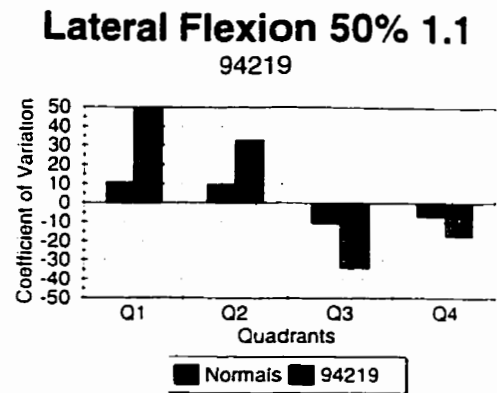
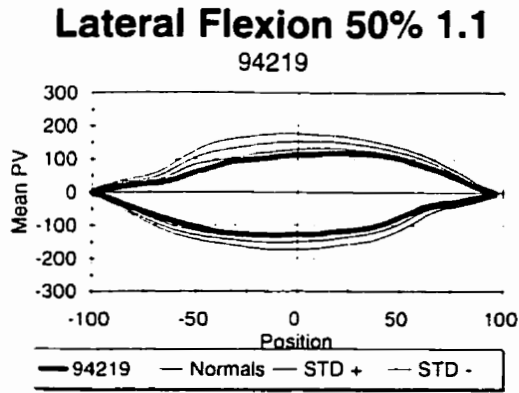


Figure D15 B-200 summary performance for Case Study #2 (Good day), for dynamic lateral flexion at 50% resistance, day 1, test sequence #1.

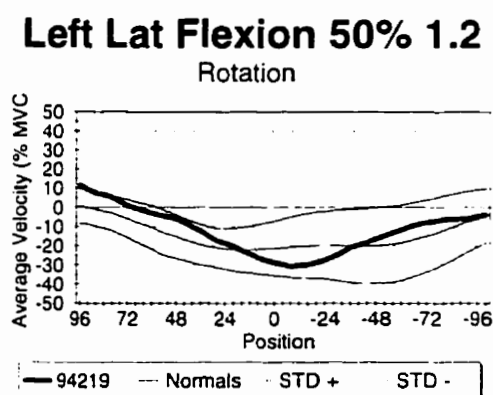
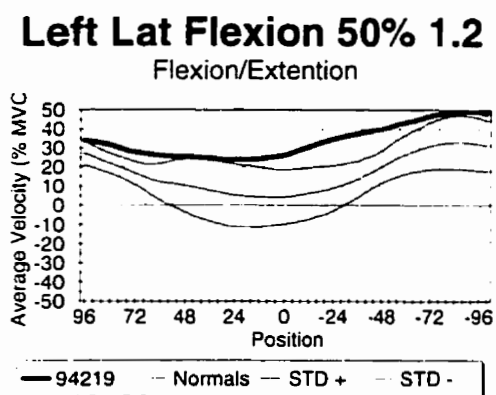
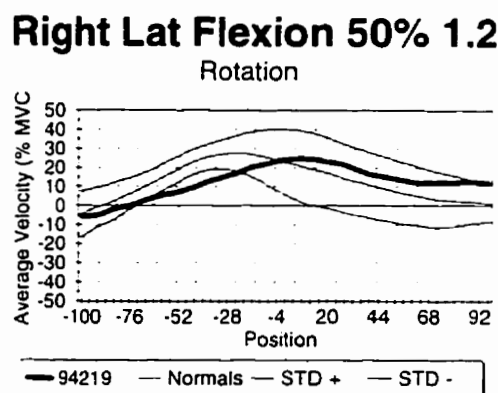
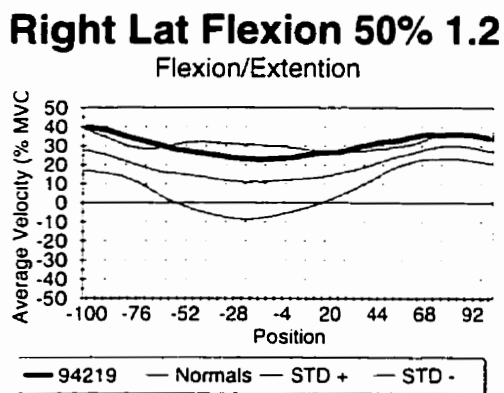
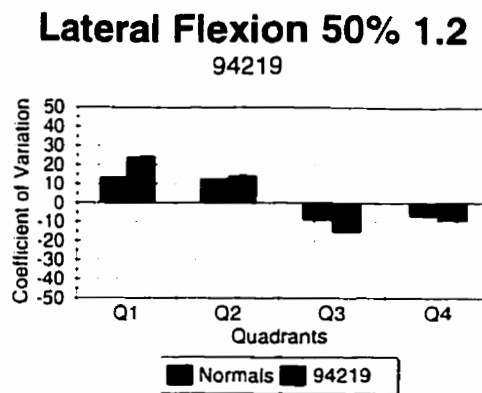
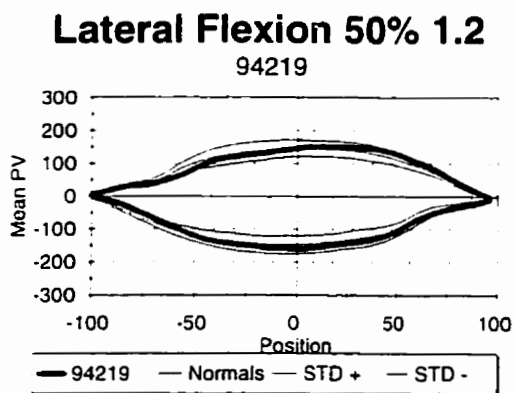
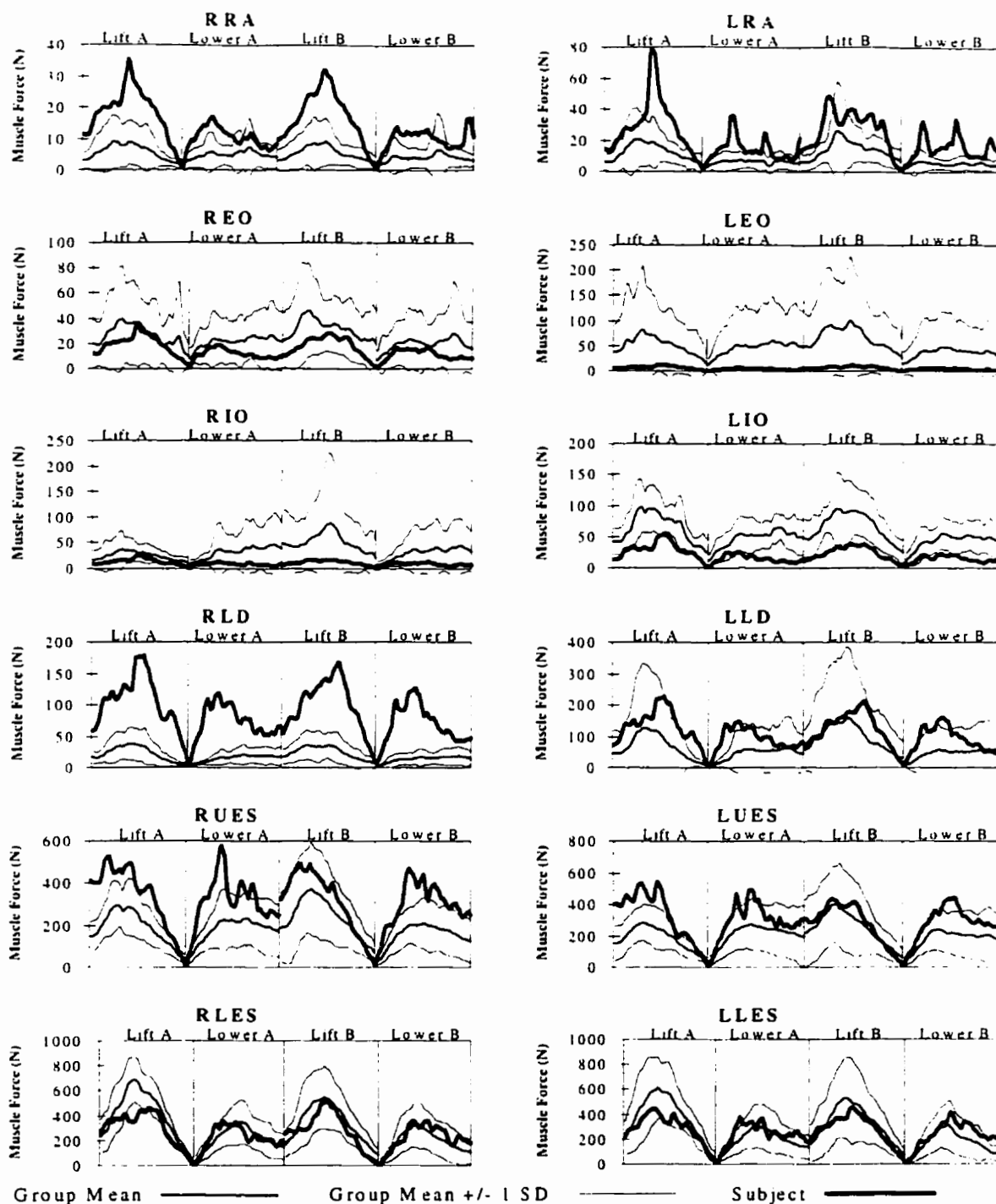
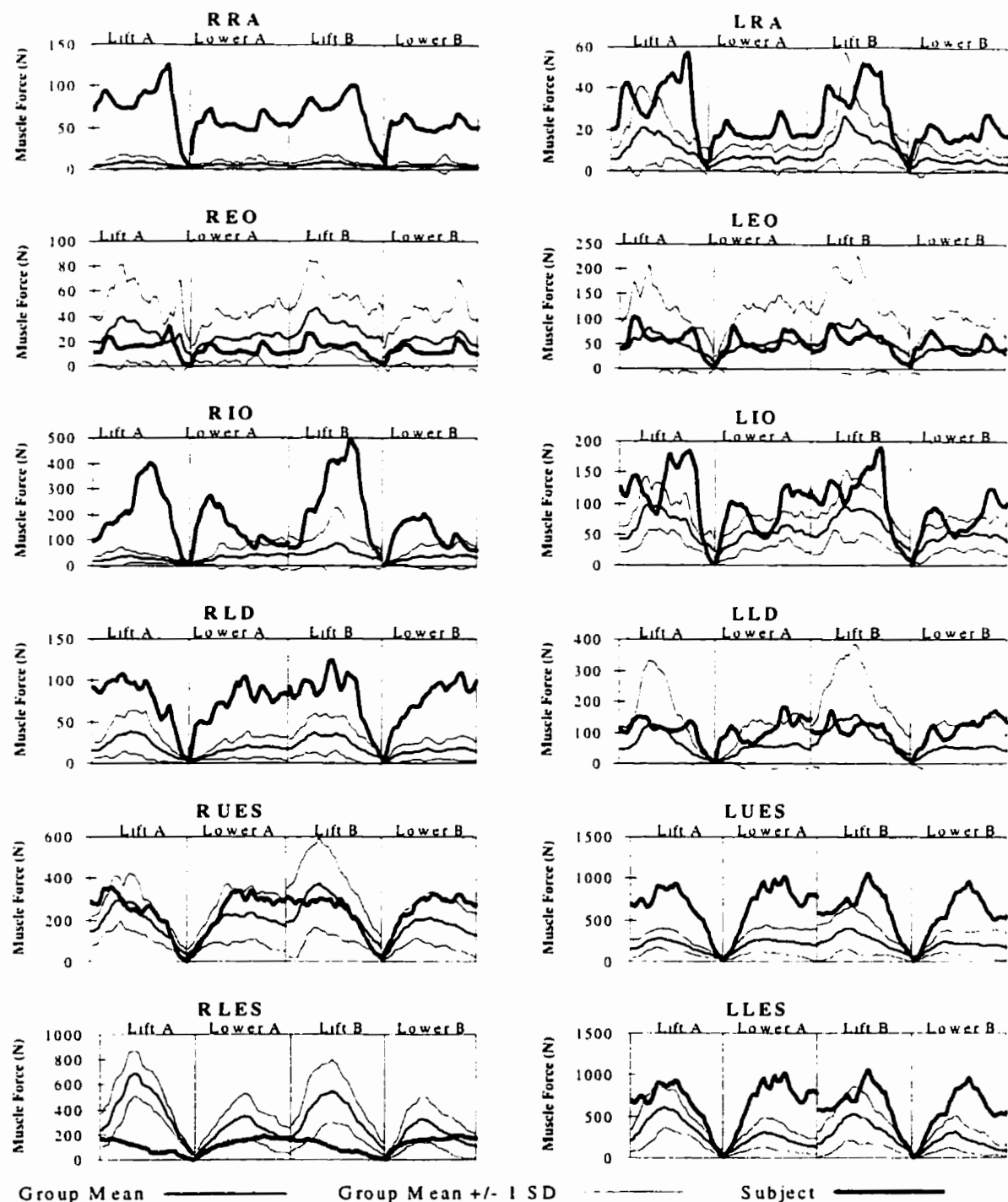


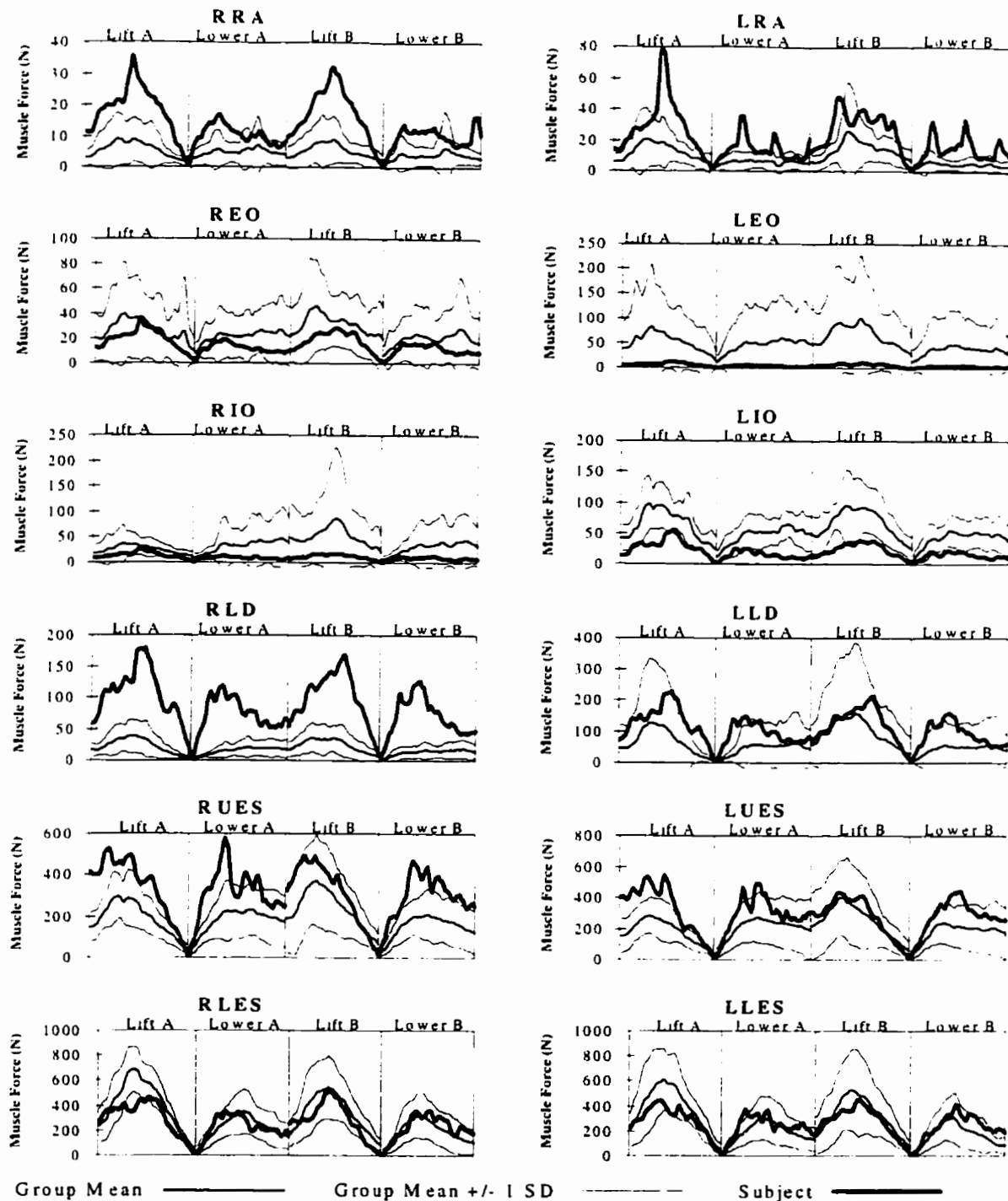
Figure D16 B-200 summary performance for Case Study #2 (Good day), for dynamic lateral flexion at 50% resistance, day 1, test sequence #2.



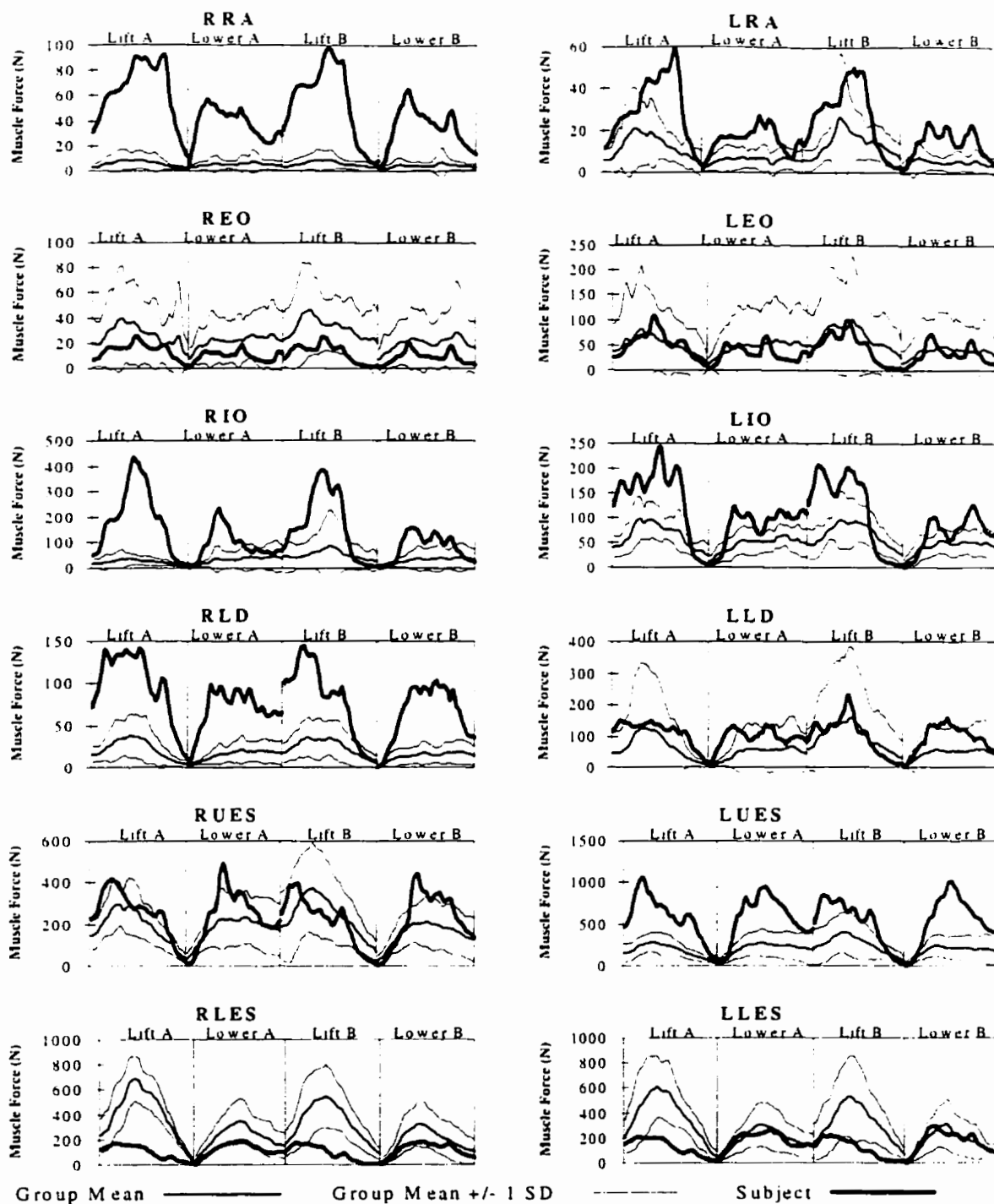
**Figure D17** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) muscle force (N) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Good Day.



**Figure D18** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) muscle force (N) for Day 2, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.

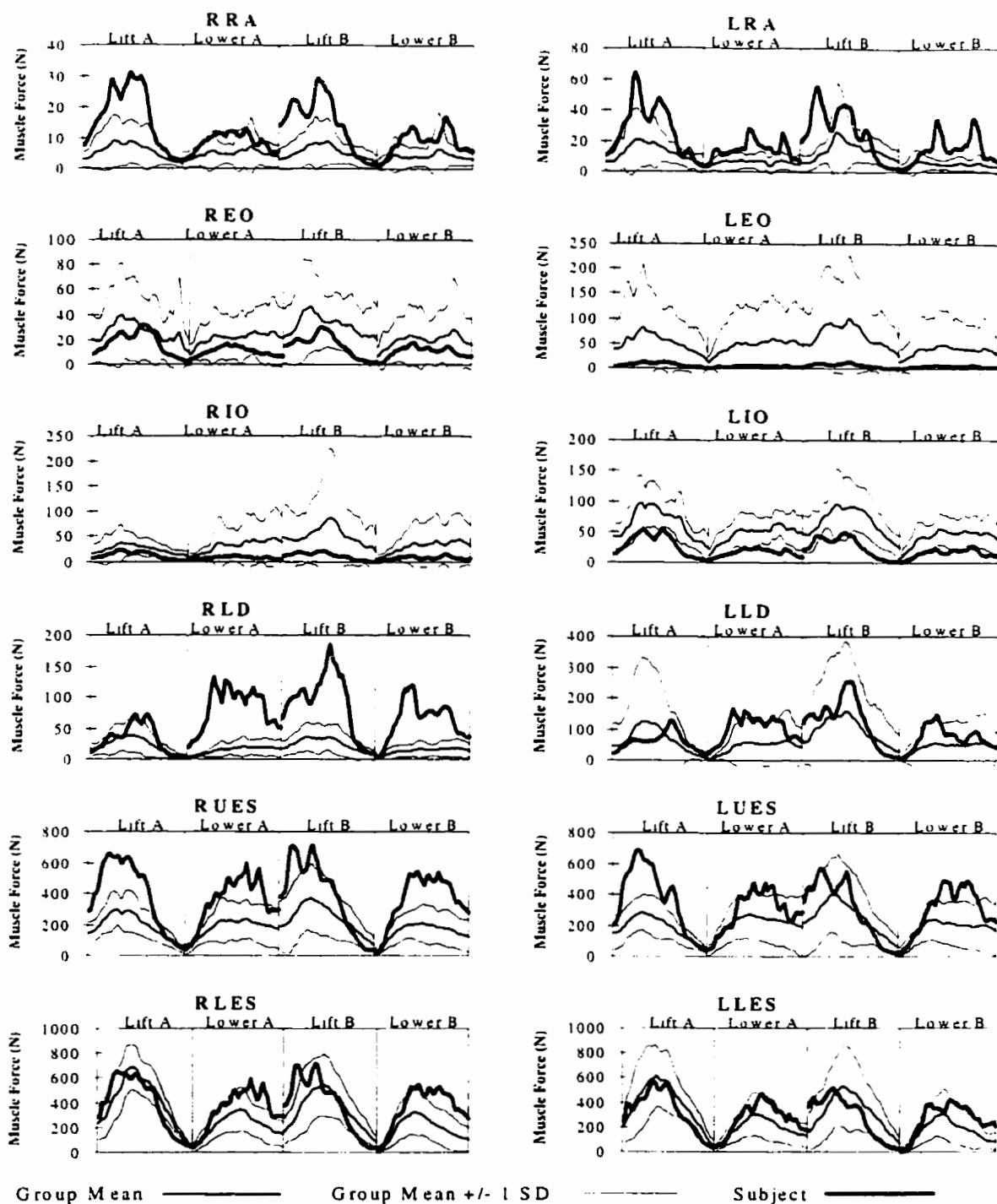


**Figure D19** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) muscle force (N) for Day 1, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA= rectus abdominis, EO= external oblique, IO= internal oblique, LD= latissimus dorsi, UES= upper erector spinae, LES= lower erector spinae. Note: Day 1 = Good Day.

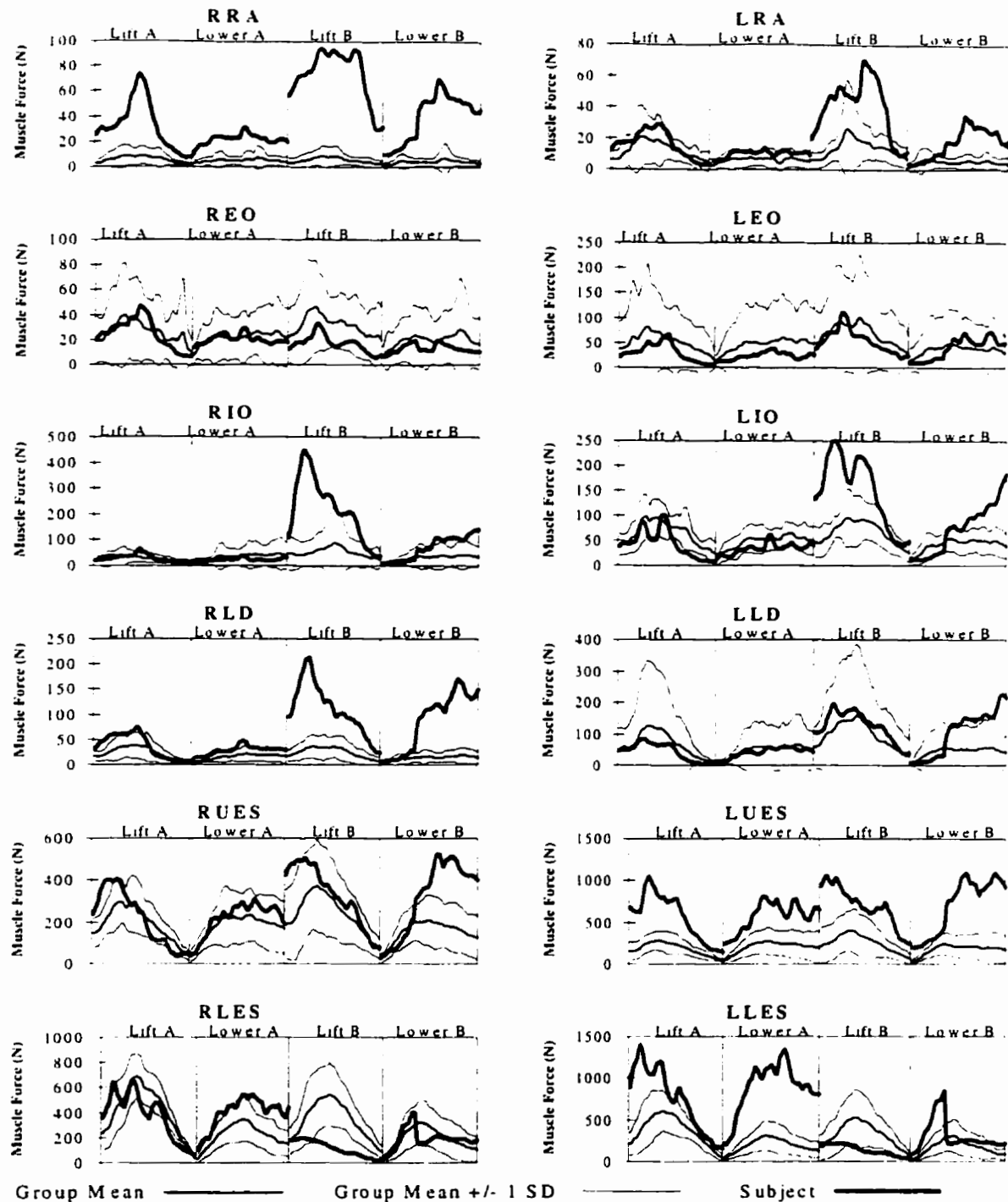


**Figure D20** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) muscle force (N) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.

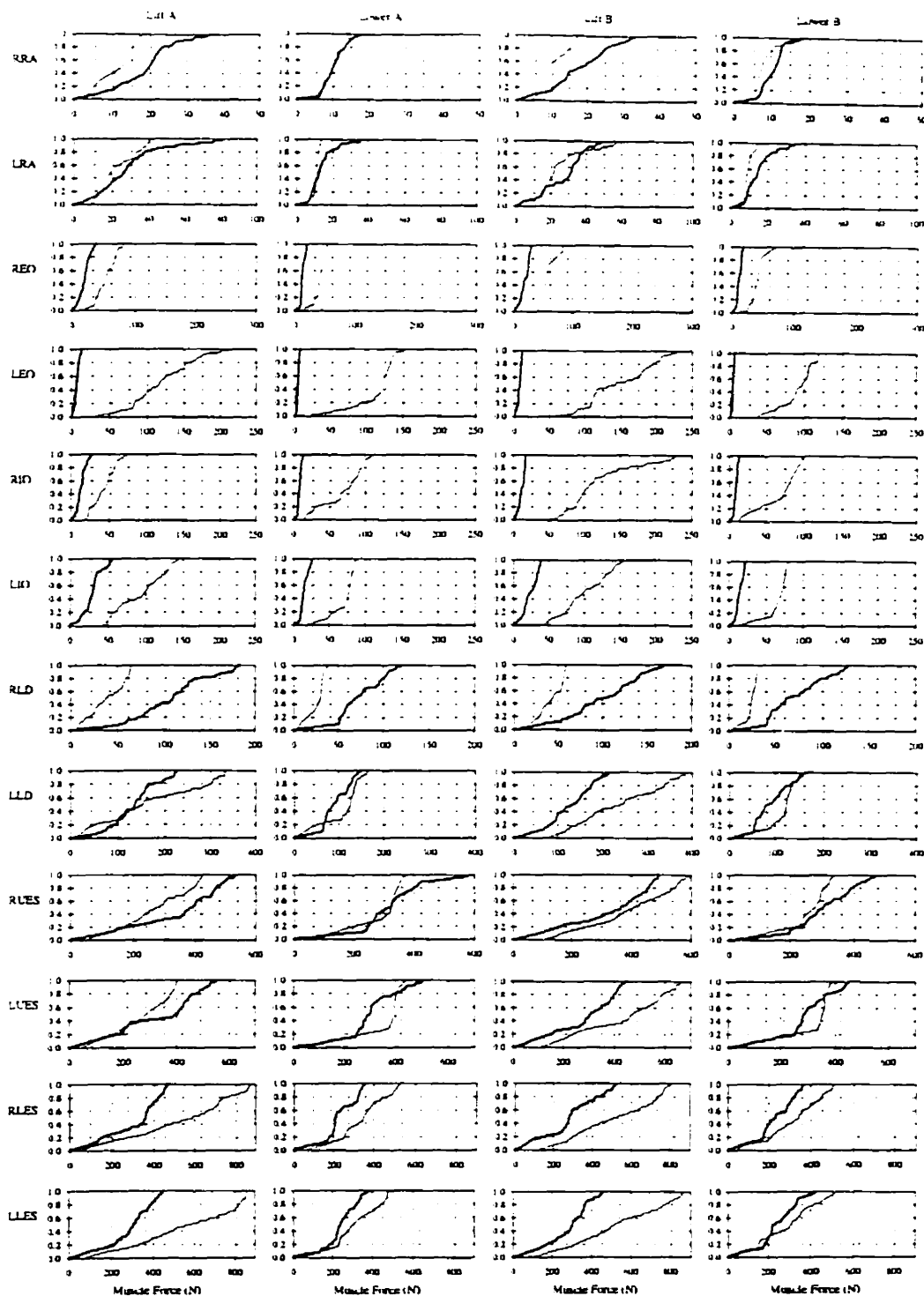




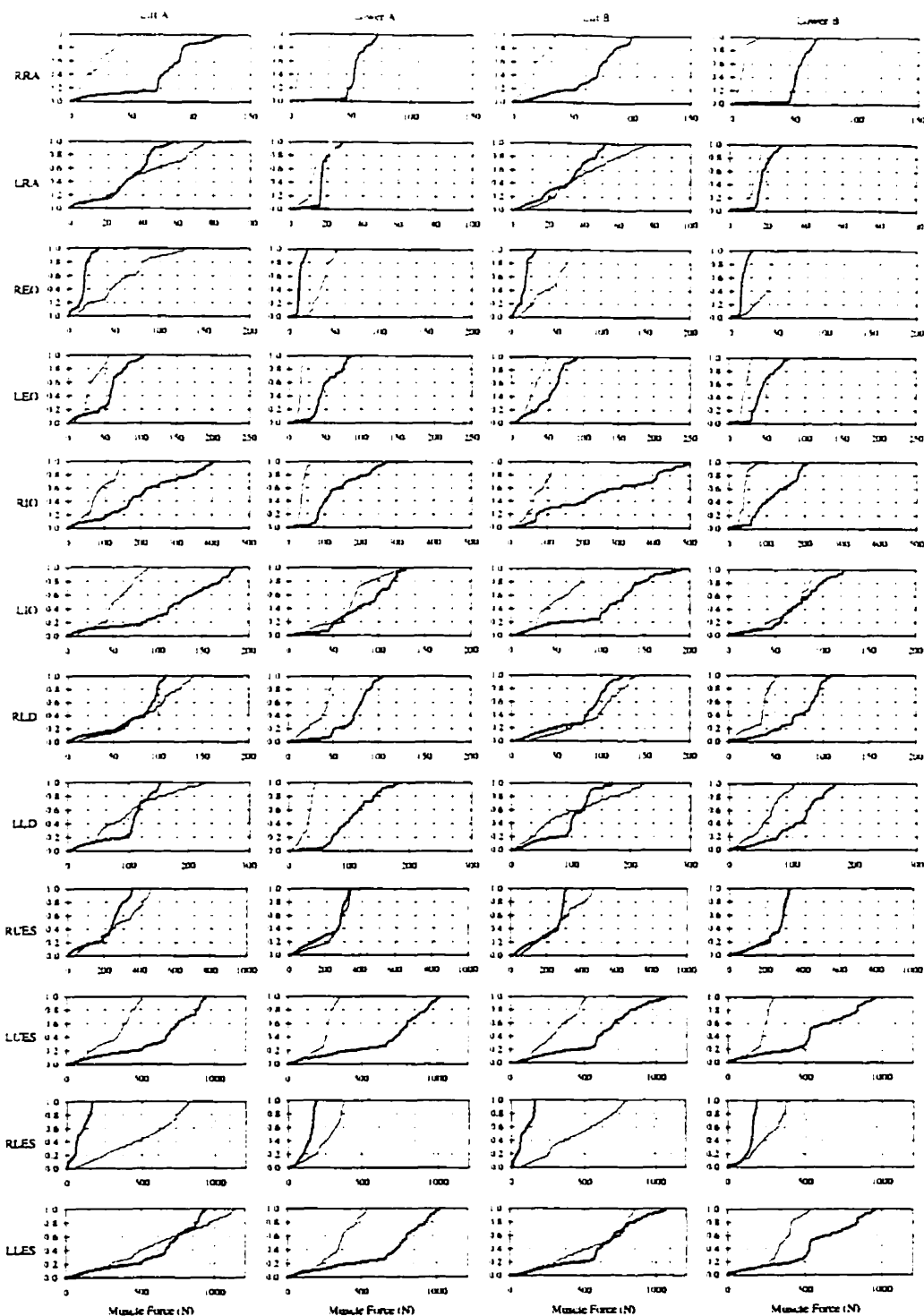
**Figure D21** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (8 subjects) muscle force (N) for Day 1, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Good Day.



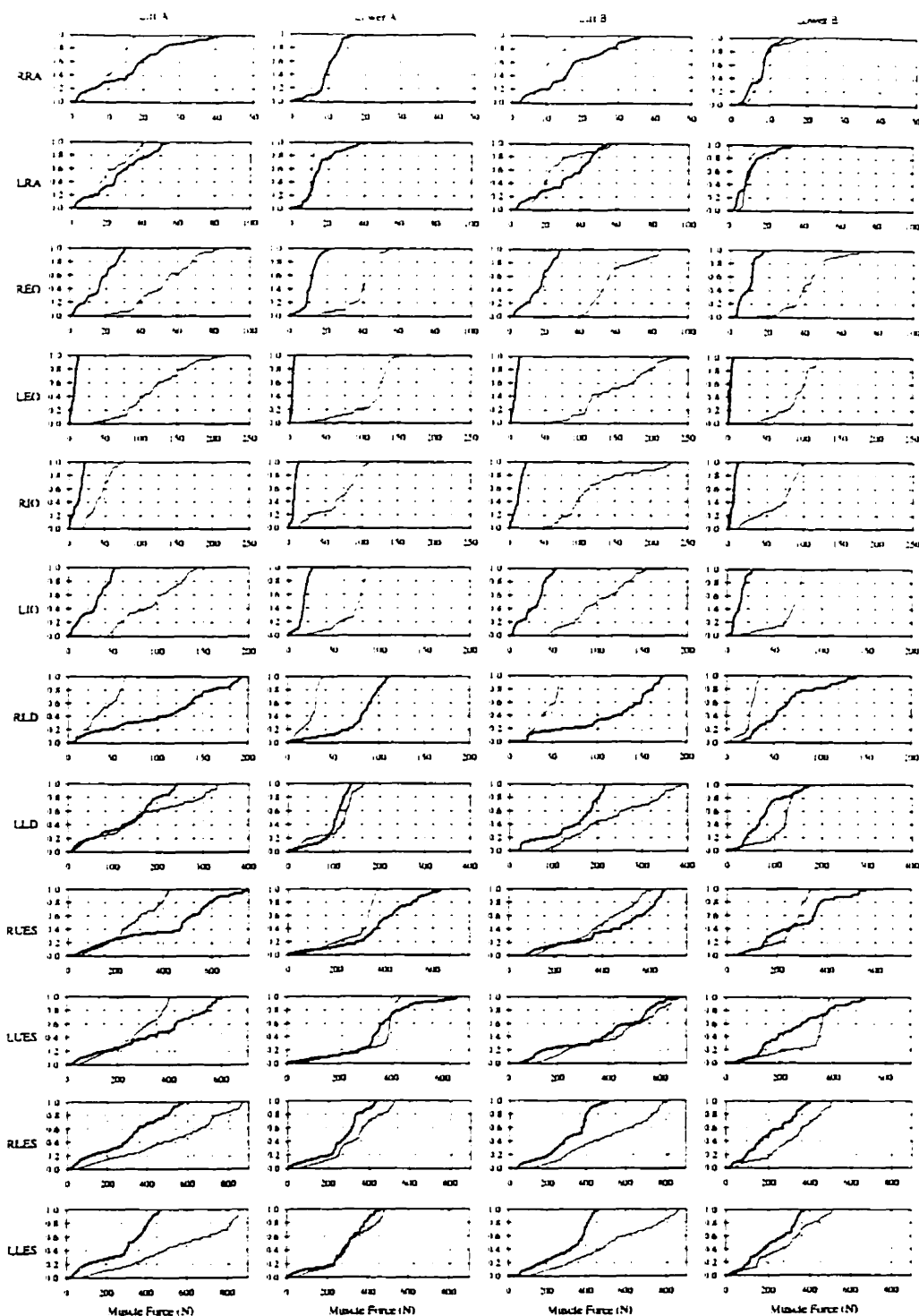
**Figure D22** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (8 subjects) muscle force (N) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA= rectus abdominis, EO= external oblique, IO= internal oblique, LD= latissimus dorsi, UES= upper erector spinae, LES= lower erector spinae. Note: Day 2 = Bad Day.



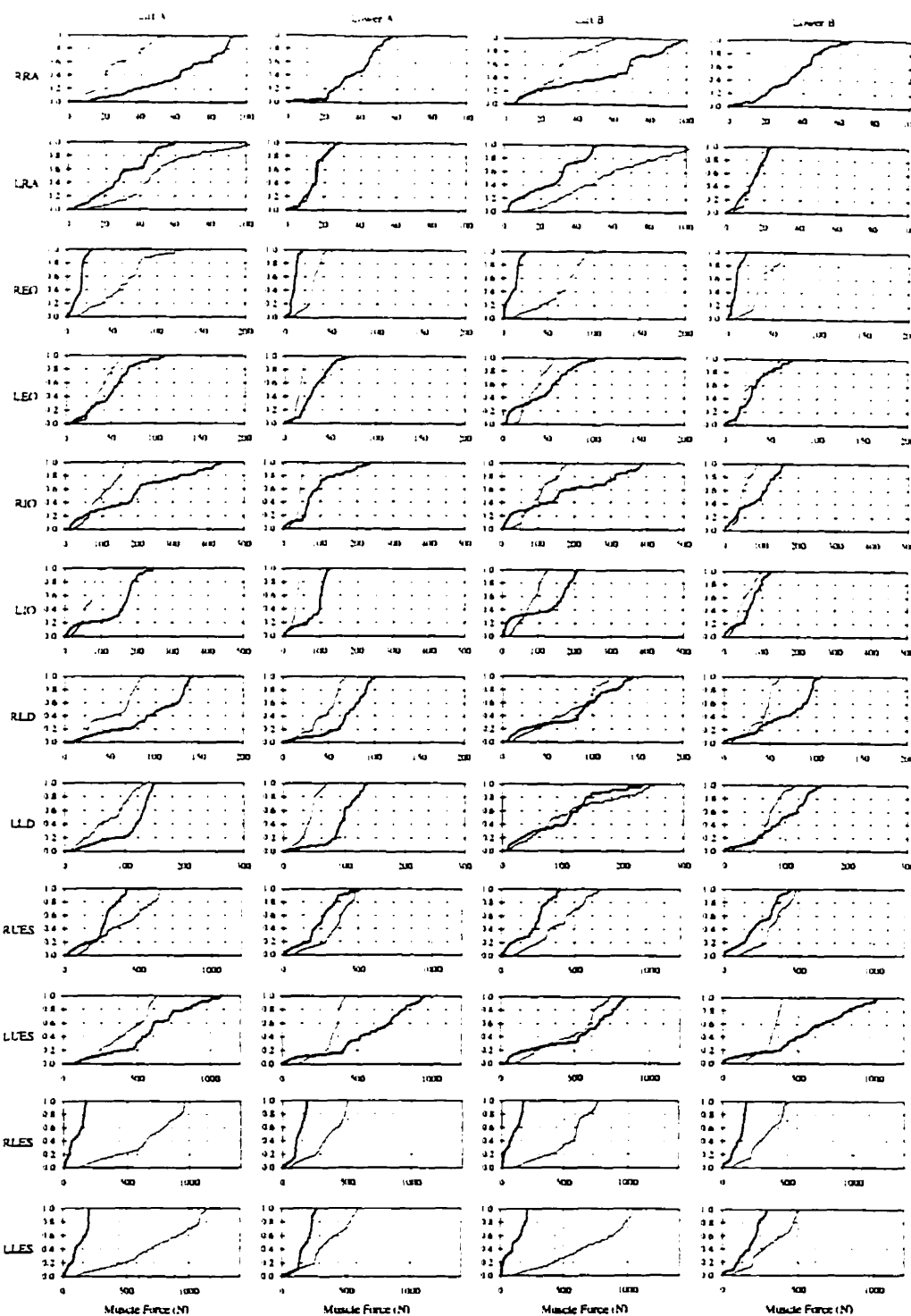
**Figure D23** Summary APDF profile of the muscle forces (N) by each electrode site for Day 1, 0 kg load and Case Study #2. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 9$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 1 = Good Day.



**Figure D24** Summary APDF profile of the muscle forces (N) by each electrode site for Day 2, 0 kg load and Case Study #2. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 10$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 2 = Bad Day.

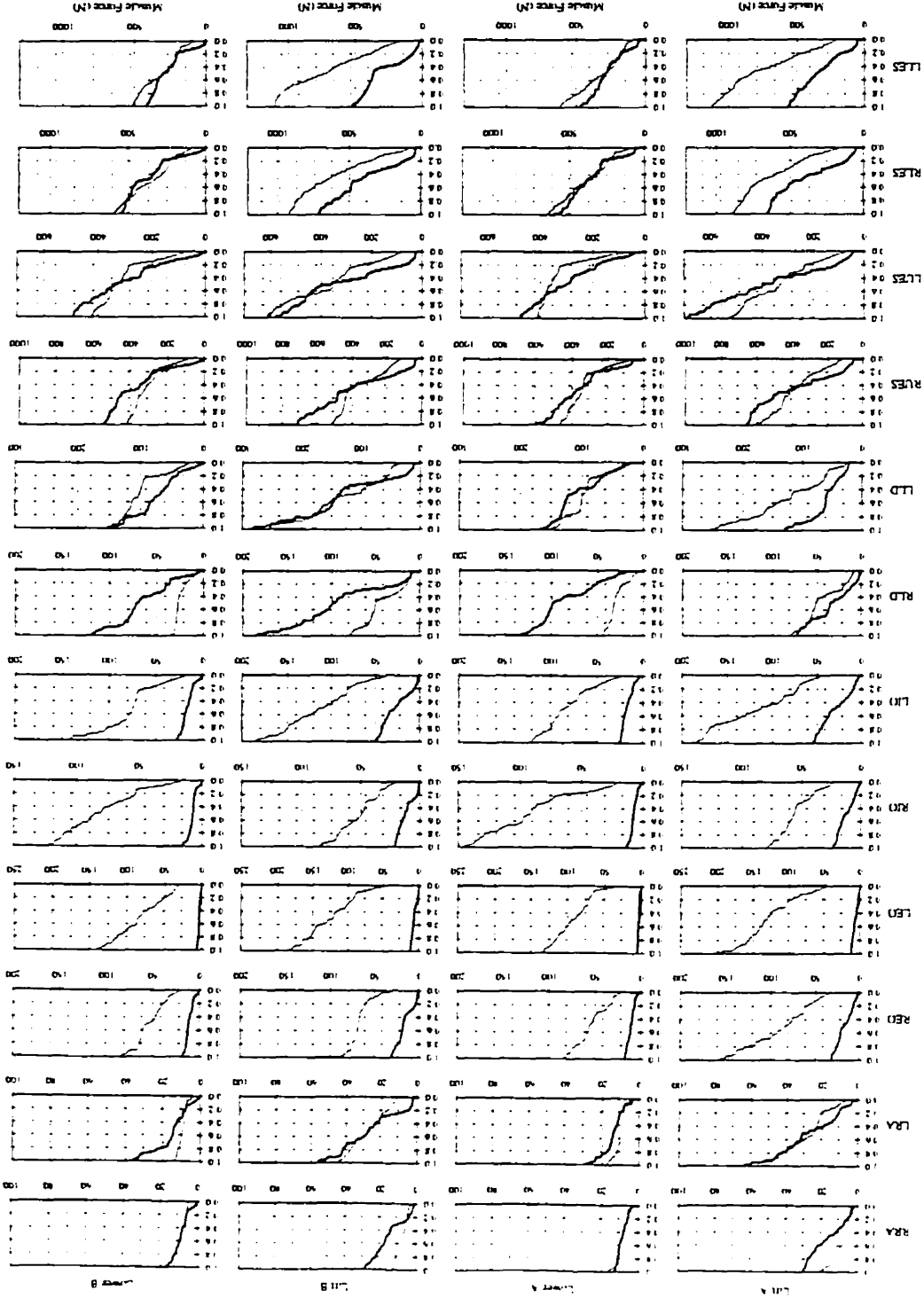


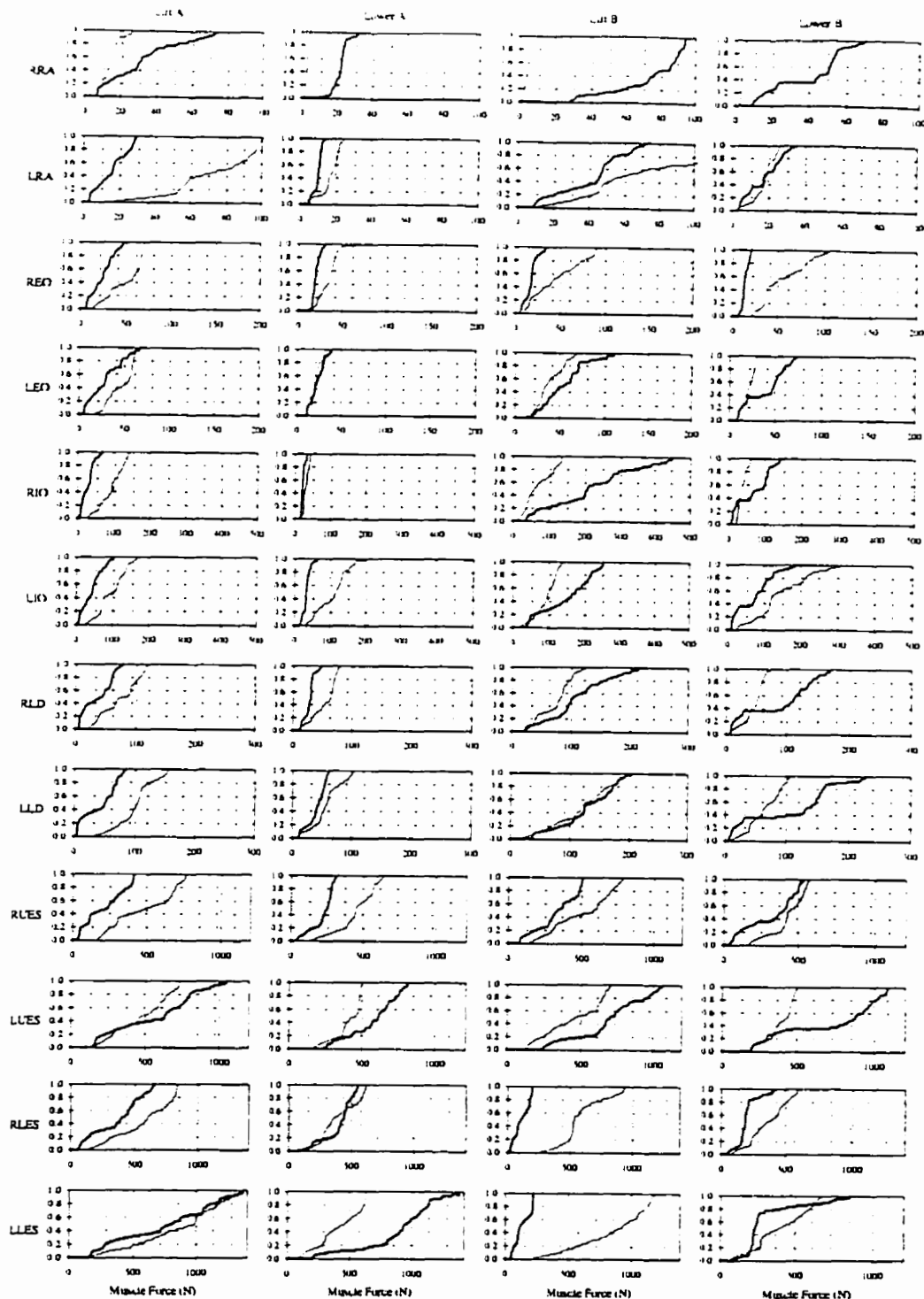
**Figure D25** Summary APDF profile of the muscle forces (N) by each electrode site for Day 1, 5 kg load and Case Study #2. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 9$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 1 = Good Day.



**Figure D26** Summary APDF profile of the muscle forces (N) by each electrode site for Day 2, 5 kg load and Case Study #2. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 9$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 2 = Bad Day.

**Figure D27 Summary APDF profile of the muscle forces (N) by each electrode site for Day 1.** 10 kg load and Case Study #2. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP (n = 8 subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 1 = Good Day.





**Figure D28** Summary APDF profile of the muscle forces (N) by each electrode site for Day 2, 10 kg load and Case Study #2. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP (n = 8 subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 2 = Bad Day.



OOC Evaluation Results for

#94219  
09-DEC-94

Demographic Data

Resistance Settings

Age : 33	Sex : M	Rotation 25% : 28 N-m
Height : 177.3 cm	Weight : 47.2 kg	Rotation 50% : 57 N-m
Diagnosis :		Flex/Ext 25% : 50 N-m
Surgical Category :		Flex/Ext 50% : 102 N-m
Activity Level Category :		Lat Flex 25% : 49 N-m
		Lat Flex 50% : 85 N-m X

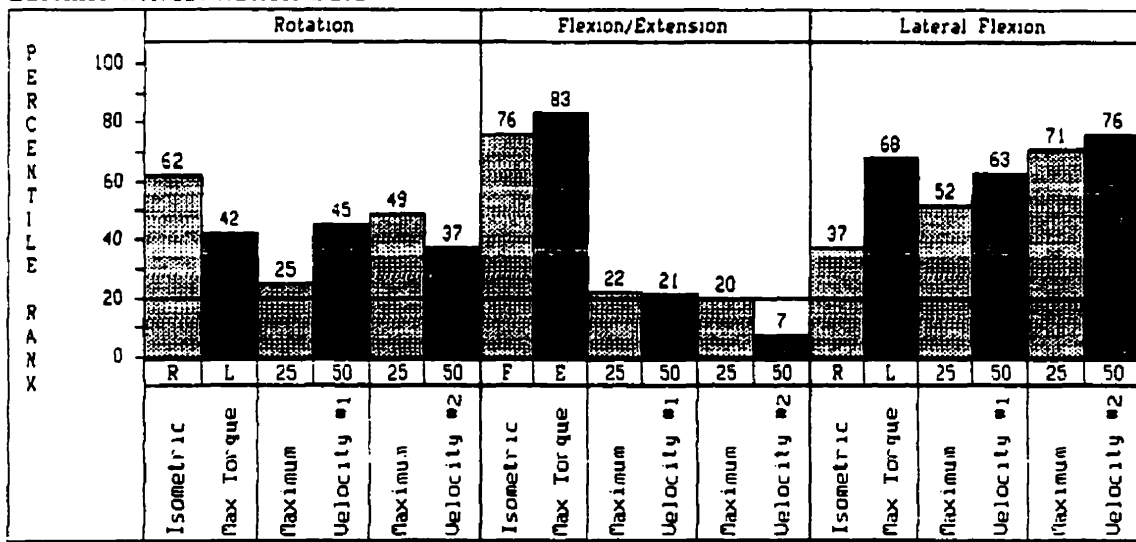
Abnormal Indicators 0

	Rotation		Flex/Ext		Lat Flex	
	25%	50%	25%	50%	25%	50%
Isometric Max Torque						
Max Velocity						
Rotation Sec Max Torque						
Flex/Ext Sec Max Torque						
Lat Flex Sec Max Torque						

Non-physiological Indicators 0

- 1) not observed
- 2) not observed
- 3) not observed
- 4) not observed
- 5) not observed
- 6) not observed

Baseline Rehabilitation Data



Test Administered By : Kardy Frazer      Signed : \_\_\_\_\_      Date : \_\_\_\_\_

Figure D29 Isostaion B-200 report (page 1) for Case Study #2 on Day 1 (Good Day).

## OOC Evaluation Results for

#94219  
09-DEC-94

OOC Test	Rotation	Flex/Ext	Lat Flex
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## Range of Motion

ROM (deg) #1	67.0	79.6	53.4
#2	91.7	92.1	74.4
ROM #2 (Δ%)			
ROM #1	36.87	15.70	39.33†

## Isometric

Max Torque (N-m)	113.1	203.0	276.9	193.7
------------------	-------	-------	-------	-------

## Dynamic

	25%	50%	25%	50%	25%	50%
Resisted ROM (Δ%) #1	40.88	25.97	7.23	13.25	33.61†	17.21
ROM #2	2.82	-0.81	-1.56	-1.04	-4.71	-15.88
Avg Vel 50% (Δ%) #1	-19.67		12.79		-6.91	
Avg Vel 25% #2	-24.30		-44.24‡		-27.47	
Max Velocity (deg/sec) #1	130.5	128.5	140.8	139.4	161.6	159.0
#2	152.2	120.6	148.9	108.3	197.5	171.9

## Secondary Axes

Rot Secondary #1	--	--	10.7	14.9	21.3	49.0
Max Torq (N-m) #2	--	--	12.8	17.1	46.9	44.8
F/E Secondary #1	107.7	123.0	--	--	132.3	141.5
Max Torq (N-m) #2	64.6	64.6	--	--	147.6	144.6
L F Secondary #1	47.4	71.1	23.7	25.8	--	--
Max Torq (N-m) #2	66.8	75.4	17.2	21.5	--	--

## Key

- ‡ Subnormal, less than critical level
- † Supernormal, greater than critical level  
(see Statistical Review in OOC Back Evaluation System)
- ‡ Lateral Flexion Isometric max torque greater than 170 Nm  
(50% Dynamic tests not scored)

Version 3.0

Figure D30 Isostaion B-200 report (page 2) for Case Study #2 on Day 1 (Good Day).

OOC Evaluation Results for

#94219

26-JAN-95

Demographic Data

Resistance Settings

Age : 33	Sex : M	Rotation 25% : 24 N-m
Height : 177.8 cm	Weight : 47.2 kg	Rotation 50% : 50 N-m
Diagnosis :		Flex/Ext 25% : 31 N-m
Surgical Category :		Flex/Ext 50% : 61 N-m
Activity Level Category :		Lat Flex 25% : 27 N-m
		Lat Flex 50% : 56 N-m

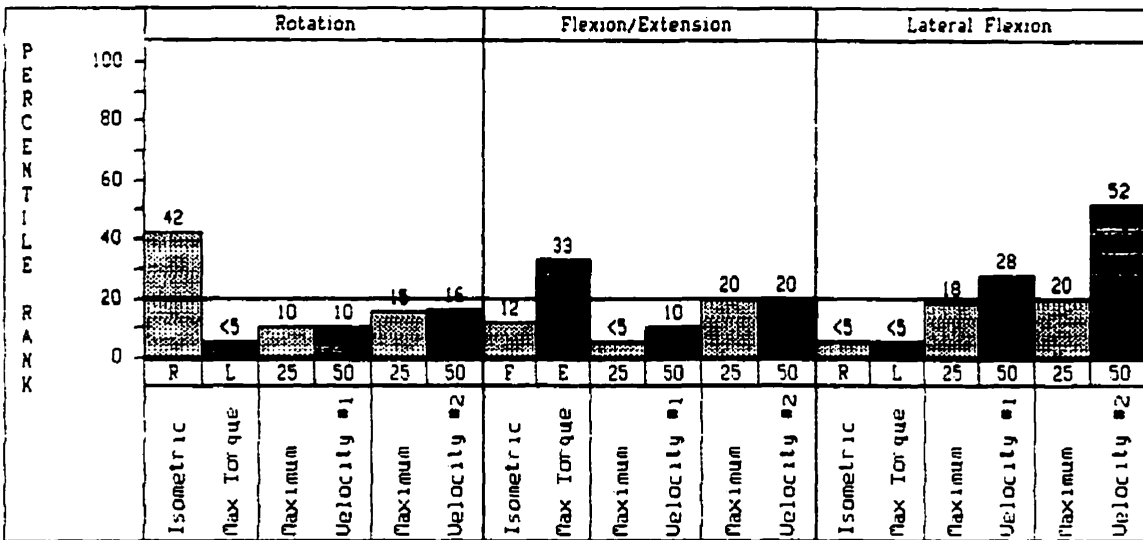
Abnormal Indicators 0

	Rotation		Flex/Ext		Lat Flex	
	25%	50%	25%	50%	25%	50%
Isometric Max Torque						
Max Velocity						
Rotation Sec Max Torque						
Flex/Ext Sec Max Torque						
Lat Flex Sec Max Torque						

Non-physiological Indicators 1

- 1) not observed
- 2) Max velocity 50% greater than or equal 25%. seq 2 : L F
- 3) not observed
- 4) not observed
- 5) not observed
- 6) not observed

Baseline Rehabilitation Data



Test Administered By : Kardy Frazer      Signed : \_\_\_\_\_      Date : \_\_\_\_\_

Figure D31 Isostaion B-200 report (page 1) for Case Study #2 on Day 2 (Bad Day).

## OOC Evaluation Results for

#94219  
26-JAN-95

OOC Test	Rotation	Flex/Ext	Lat Flex
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## Range of Motion

ROM (deg) #1	55.8	79.2	58.6
#2	88.4	74.3	56.9
ROM #2 (Δ%)			
ROM #1	34.35	-4.59	-2.30

## Isometric

Max Torque (N-m)	100.2	123.0	215.3	109.9
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## Dynamic

		25%	50%	25%	50%	25%	50%
Resisted ROM (Δ%) #1		36.52	-2.81	-19.02	-18.40	2.24	-0.75
ROM #2		-4.18	-13.81	13.55	-0.65	6.92	7.69†
Avg Vel 50% (Δ%) #1		-27.35		12.34		2.63	
Avg Vel 25% #2		-23.61		-29.19		-8.62	
Max Velocity (deg/sec) #1		110.7	90.9	111.0‡	119.1	118.0	123.1
#2		122.5	102.8	151.6	127.3	133.4	143.7

## Secondary Axes

Rot Secondary #1	--	--	10.7	17.1	72.5†	46.9
Max Torq (N-m) #2	--	--	21.3	21.3	34.1	49.0
F/E Secondary #1	135.3	49.2	--	--	80.0	116.9
Max Torq (N-m) #2	76.9	107.7	--	--	110.7	132.3
L F Secondary #1	47.4	73.2	19.4	30.1	--	--
Max Torq (N-m) #2	56.0	73.2	23.7	30.1	--	--

## Key

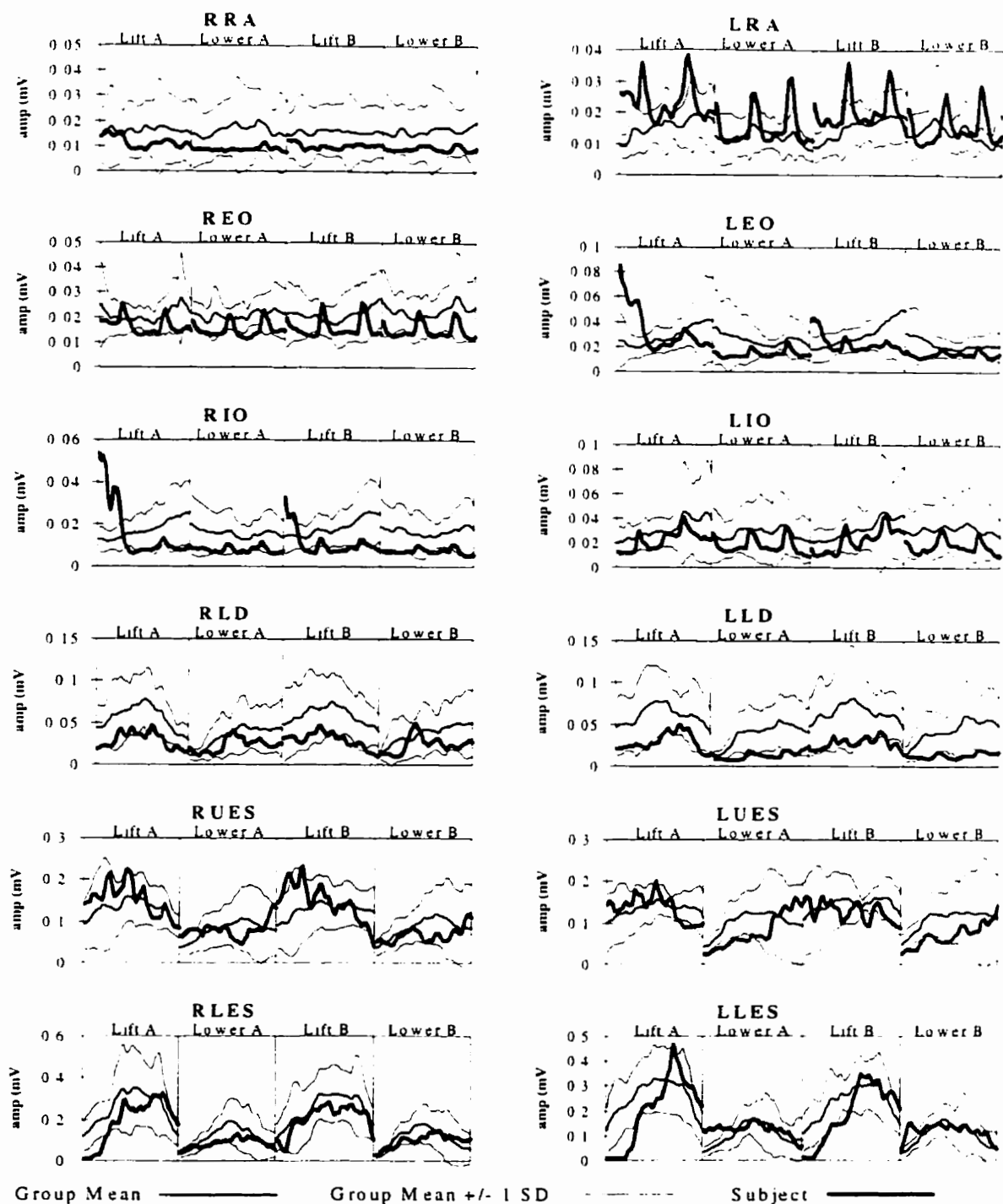
‡ Subnormal, less than critical level  
 † Supernormal, greater than critical level  
 (see Statistical Review in OOC Back Evaluation System)

Version 3.0

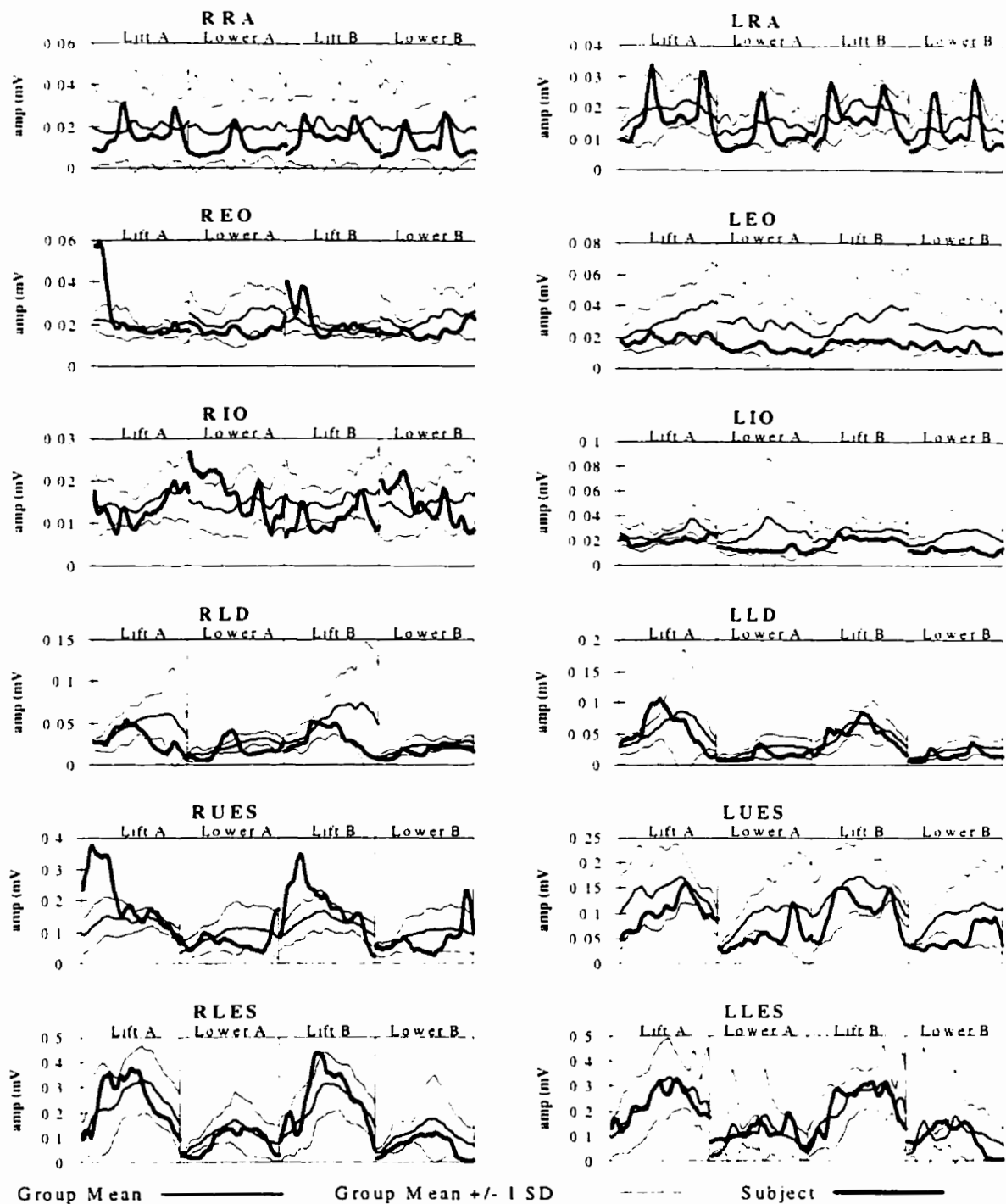
Figure D32 Isostaion B-200 report (page 2) for Case Study #2 on Day 2 (Bad Day).

## **Appendix E**

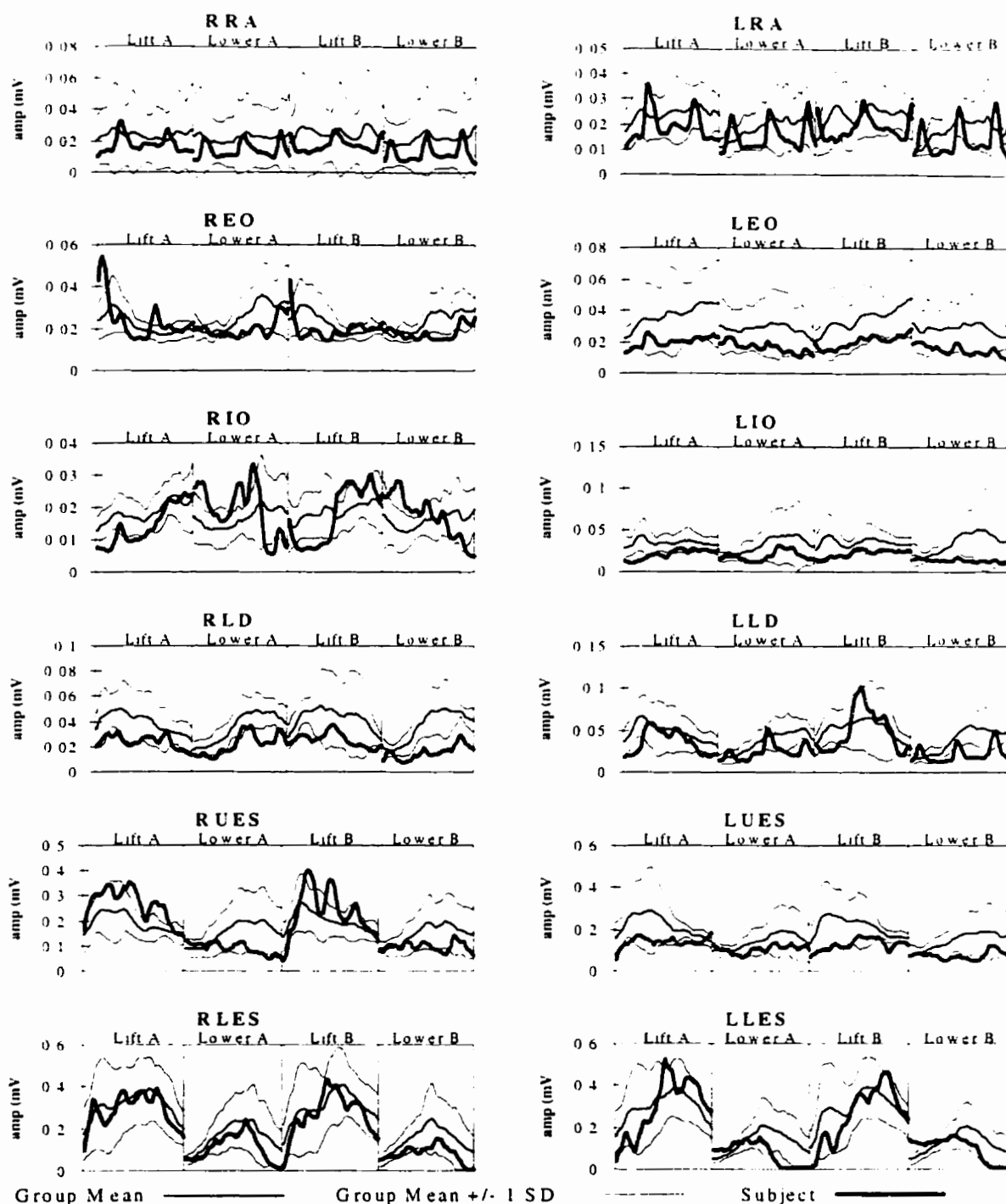
Case Study #3



**Figure E1** Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (mV) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Good Day.

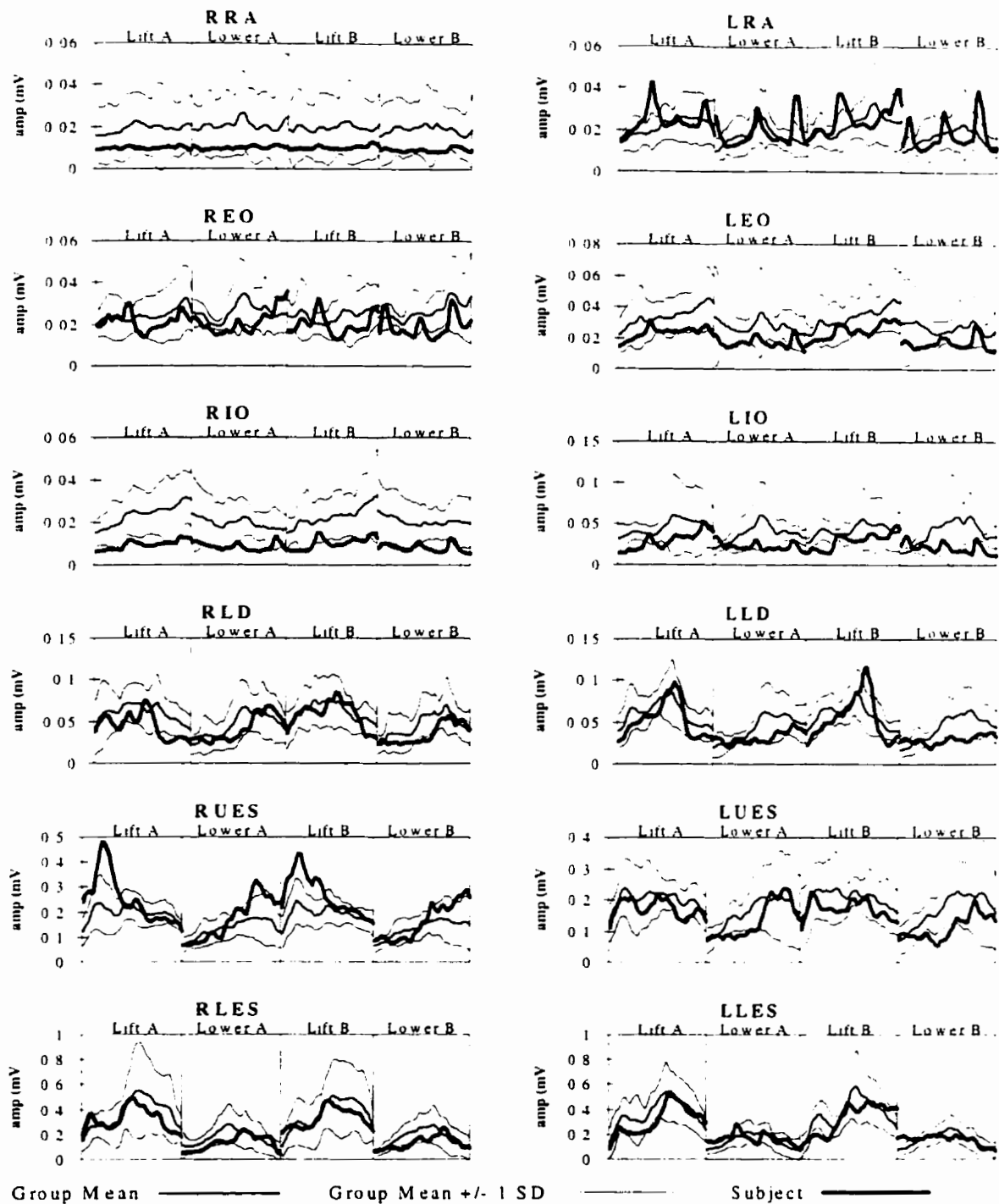


**Figure E2** Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (mV) for Day 2, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.

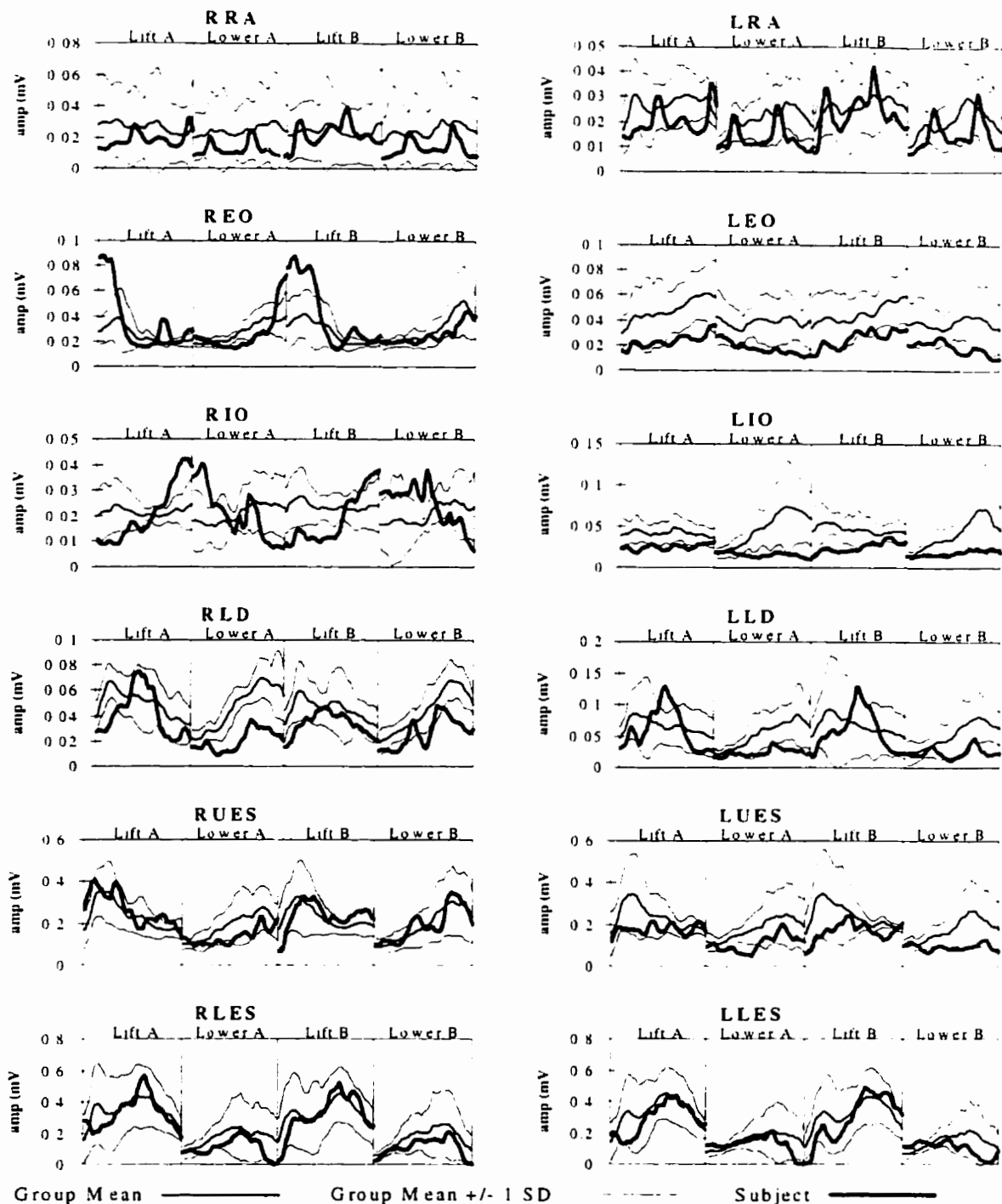


**Figure E3** Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (mV) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.

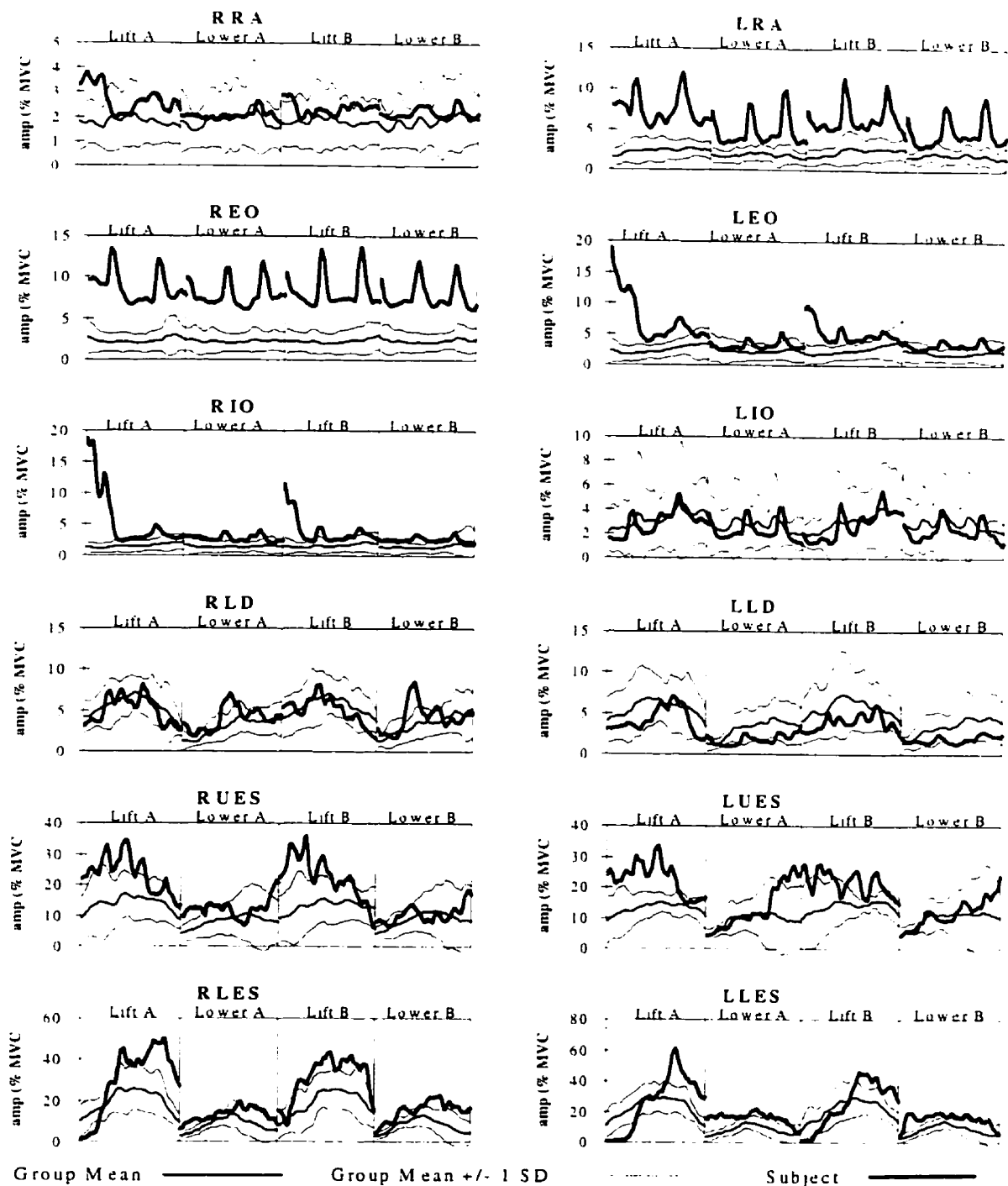




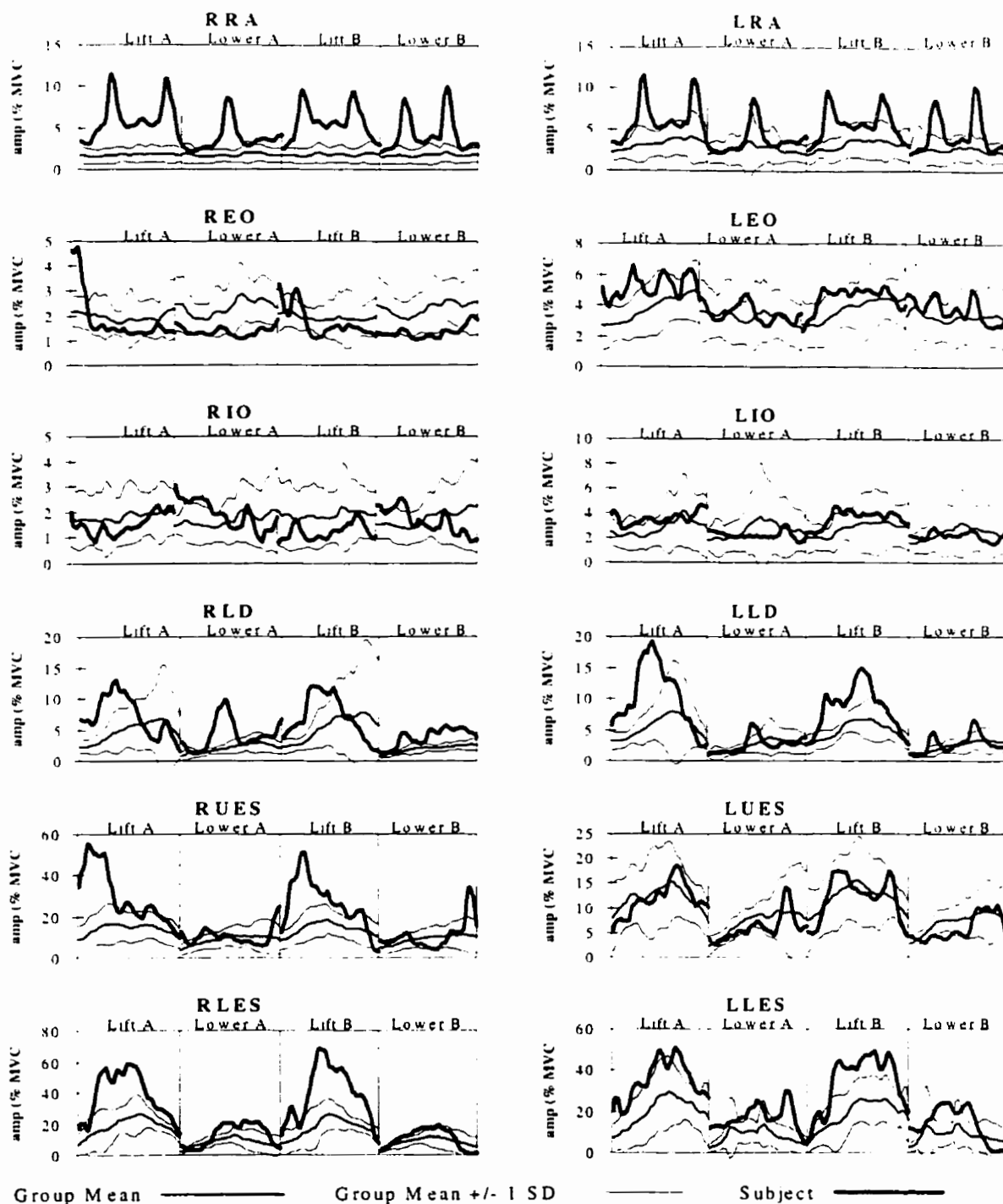
**Figure E4** Case Study #3 graphed against the summary profile of the mean ( $\pm$  1 SD) ASYMP (9 subjects) EMG activity levels (mV) for Day 1, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Good Day.



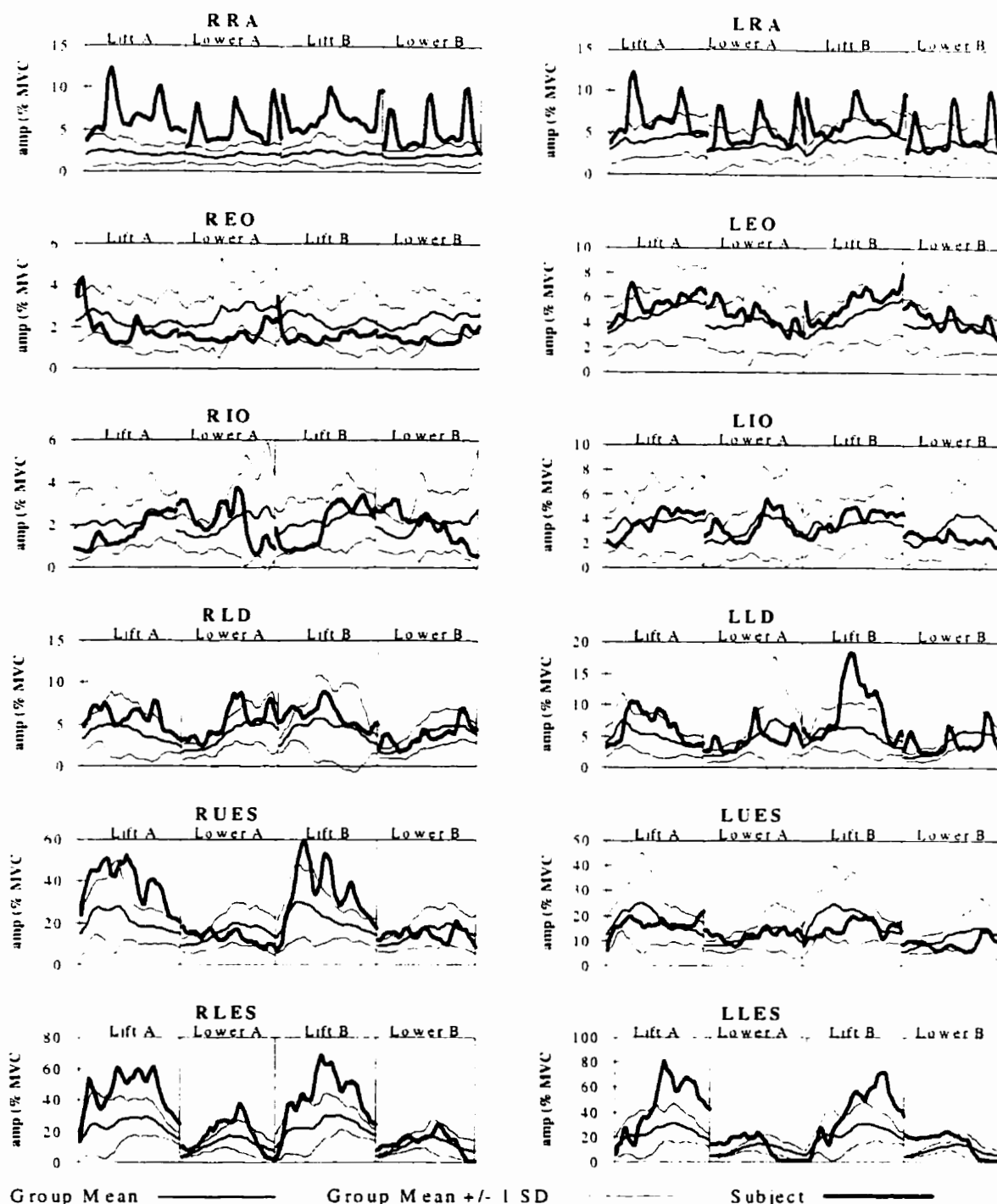
**Figure E5** Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (mV) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.



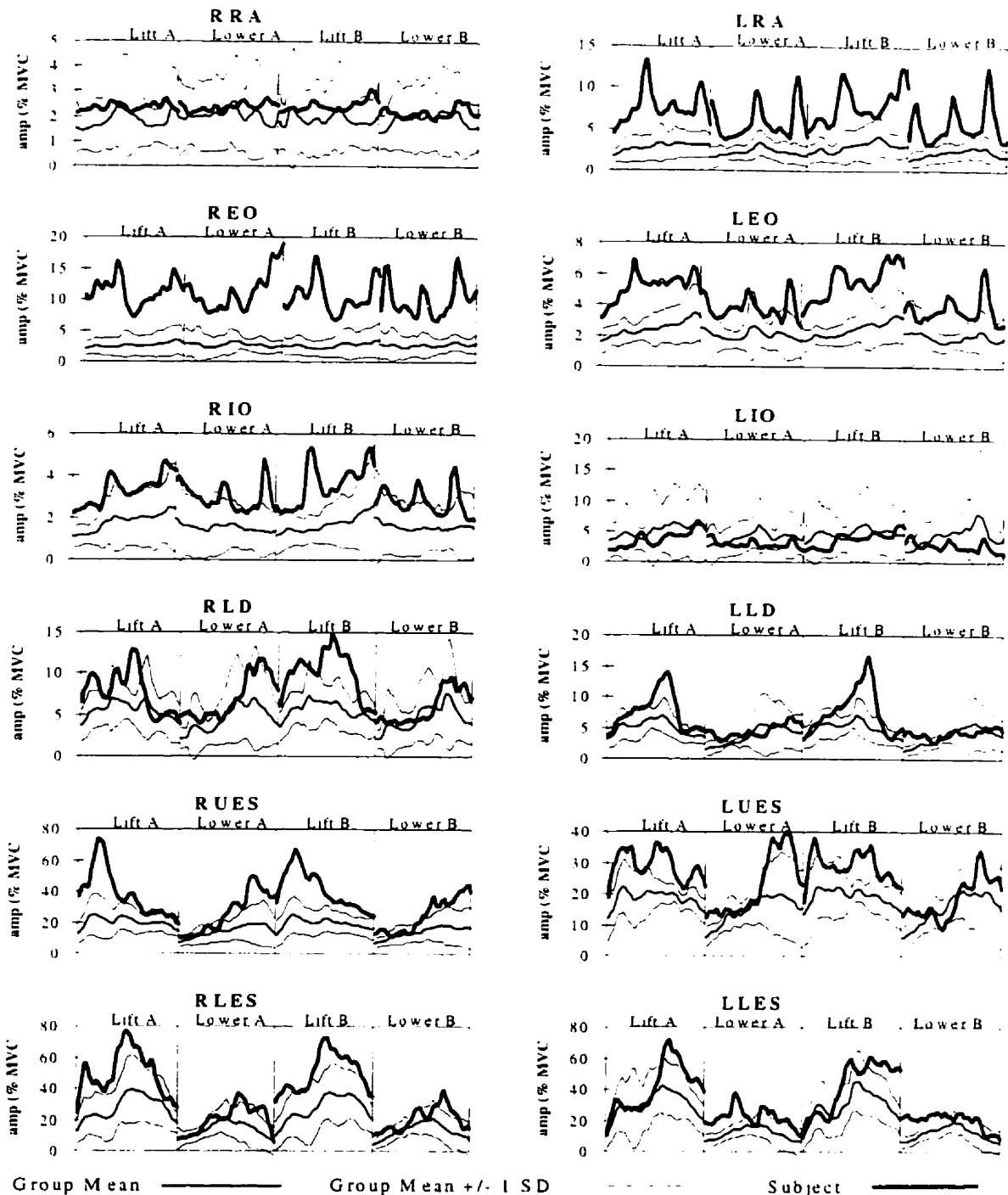
**Figure E6** Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (% MVC) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Good Day.



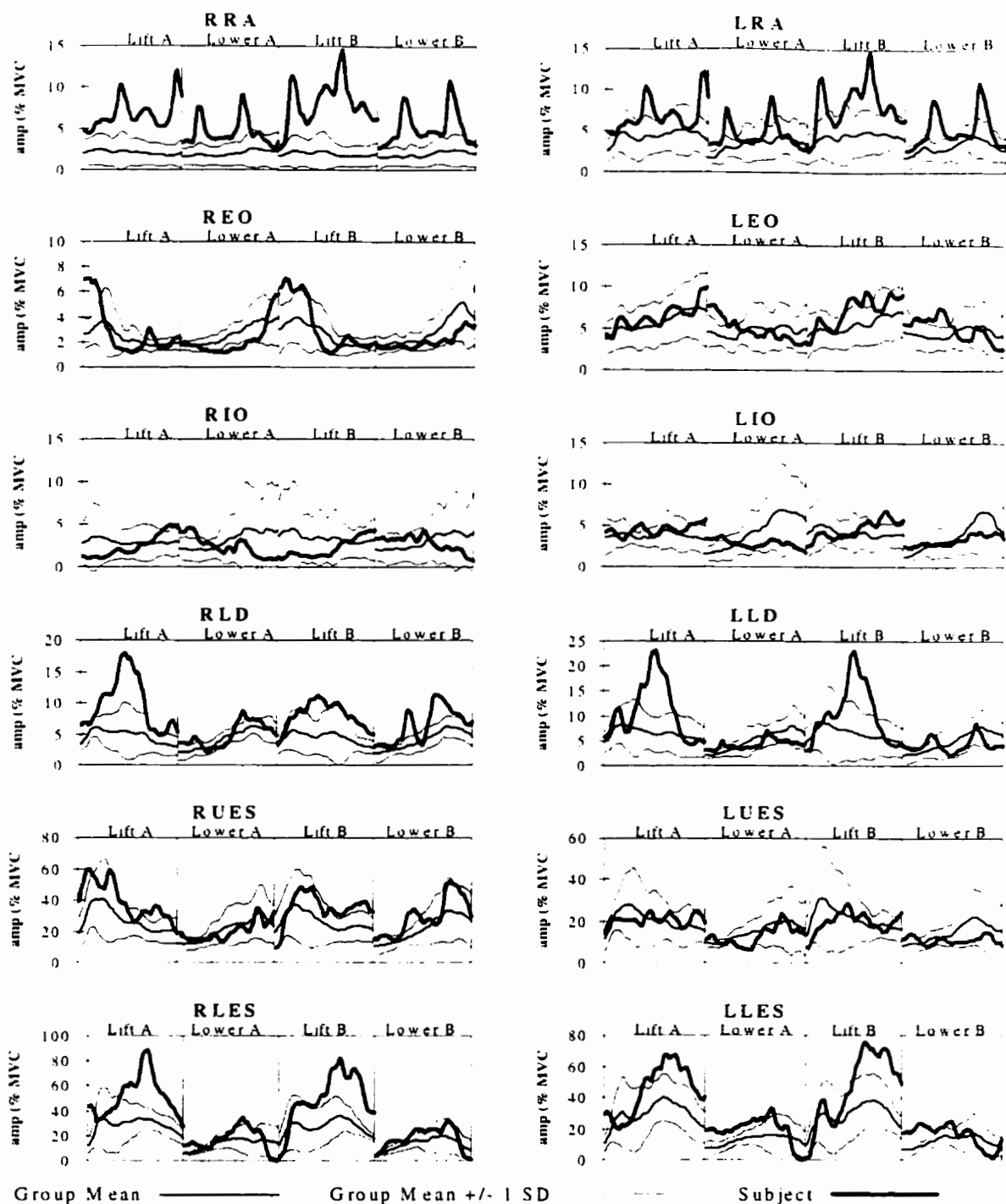
**Figure E7** Case Study #2 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 2, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.



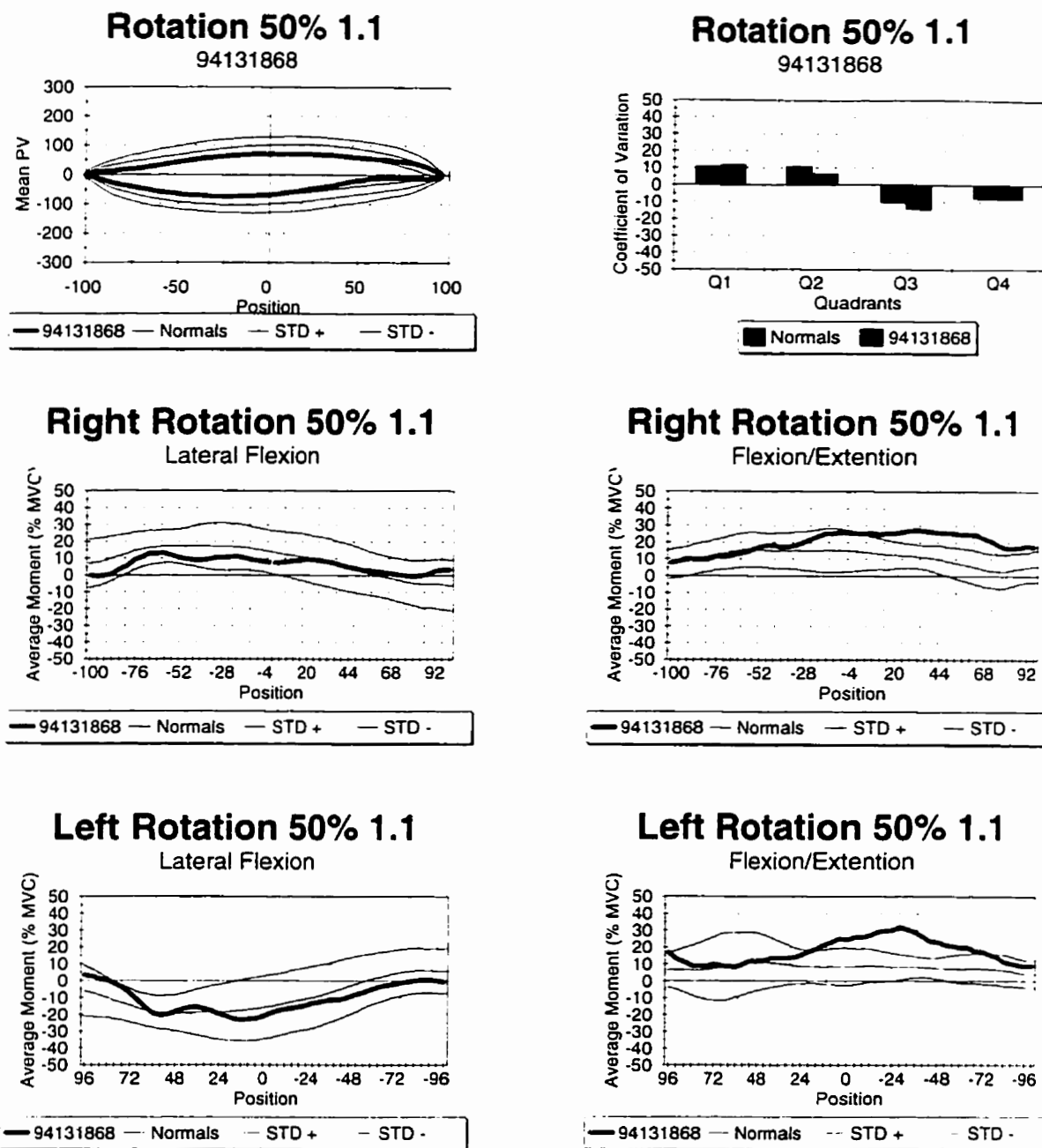
**Figure E8** Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.



**Figure E9** Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (% MVC) for Day 1, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Good Day.

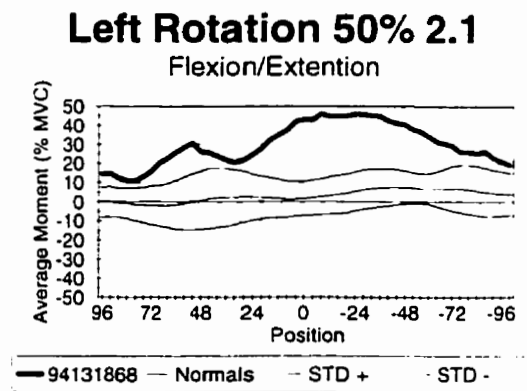
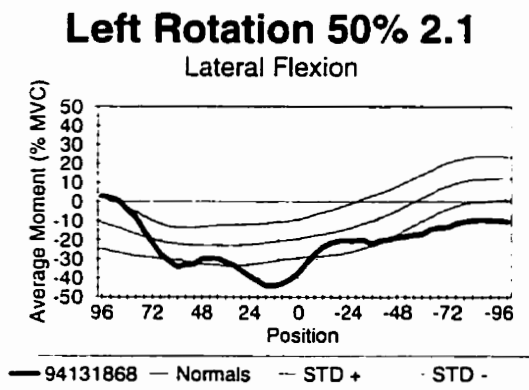
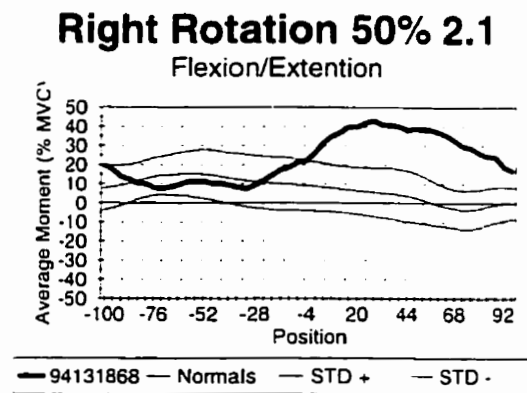
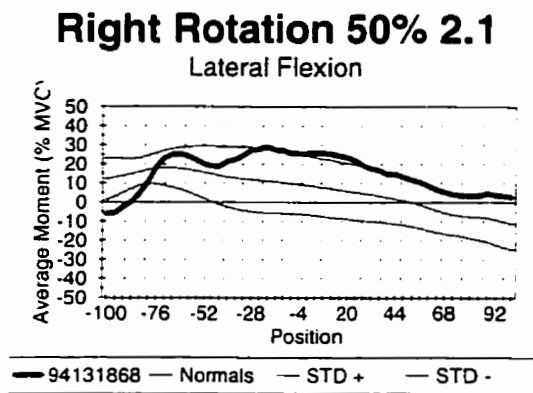
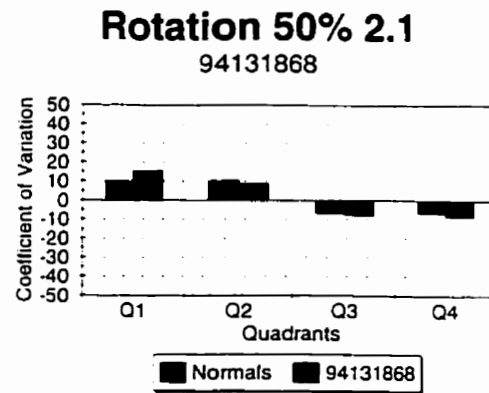
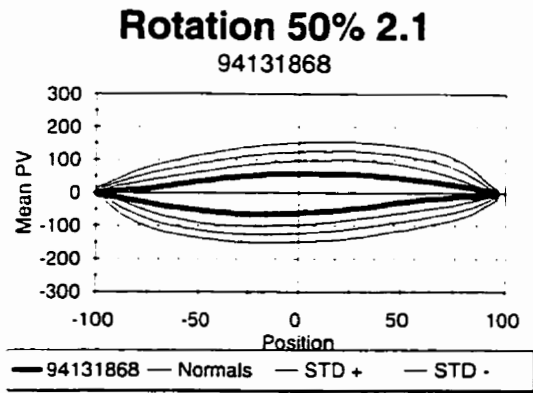


**Figure E10** Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.

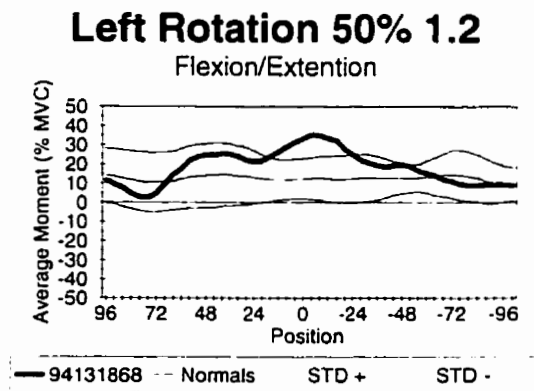
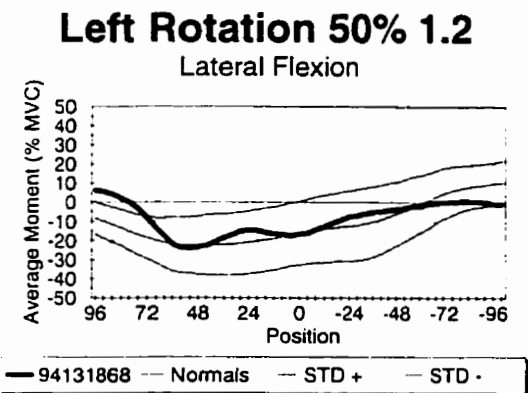
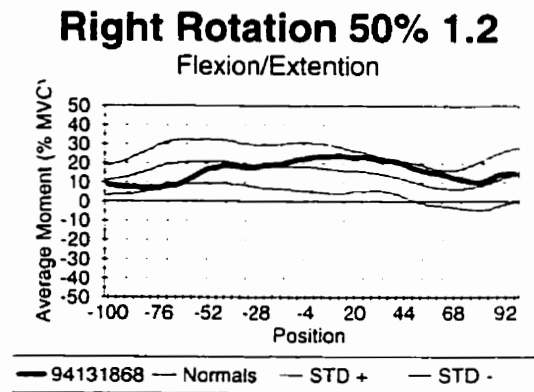
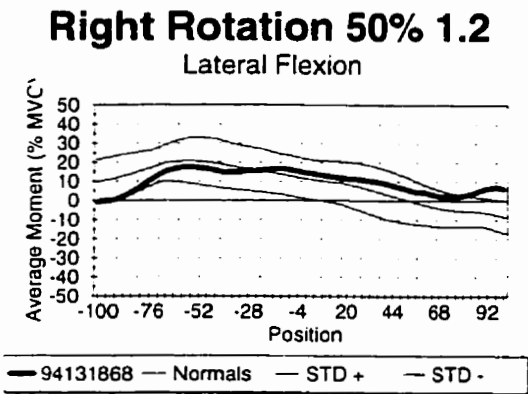
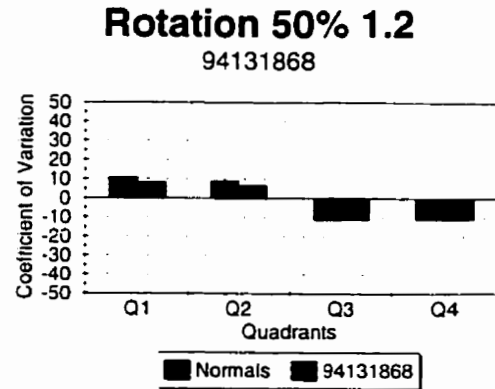
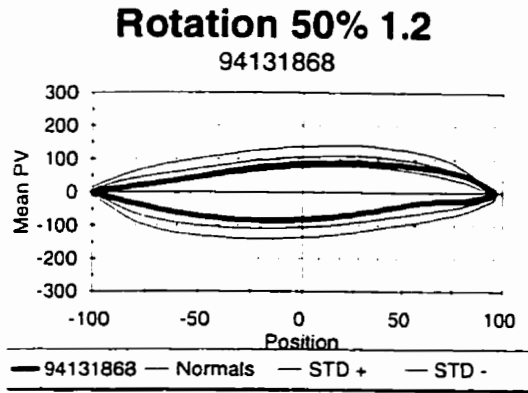


**Figure E11 B-200** summary performance for Case Study #3 (Good day), for dynamic rotation at 50% resistance, day 1, test sequence #1.

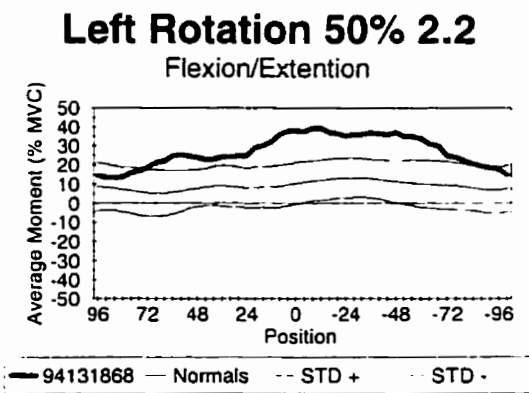
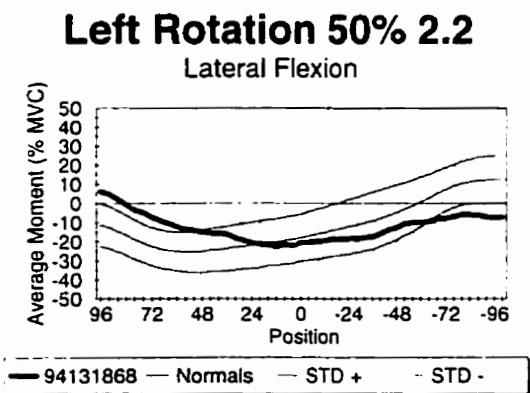
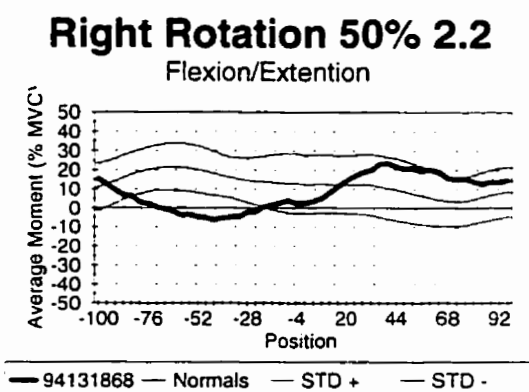
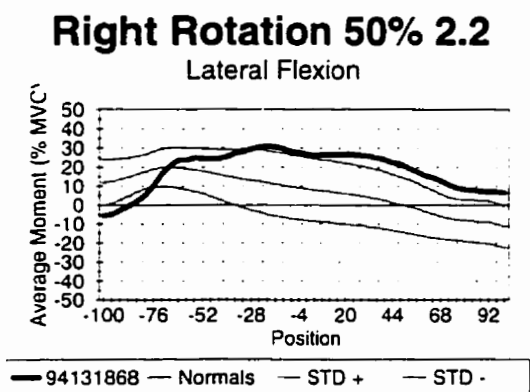
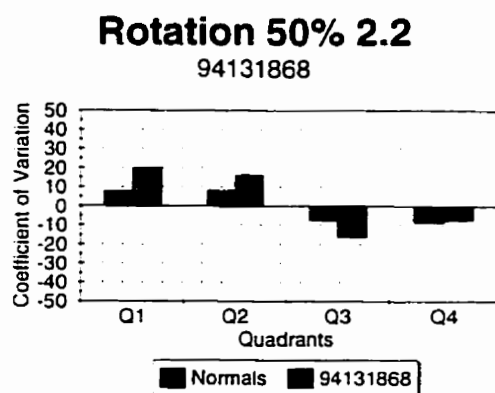
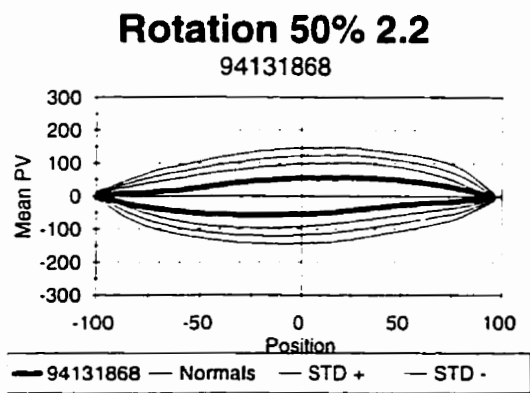




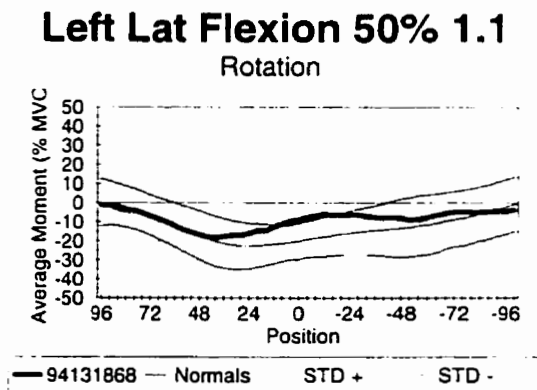
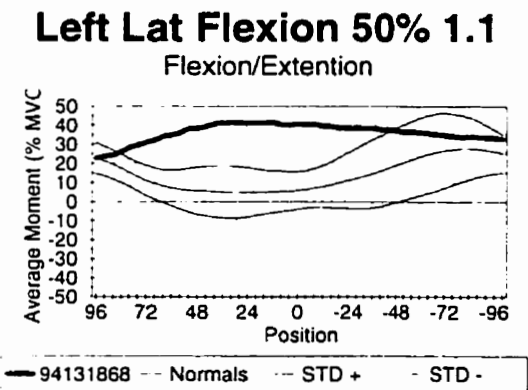
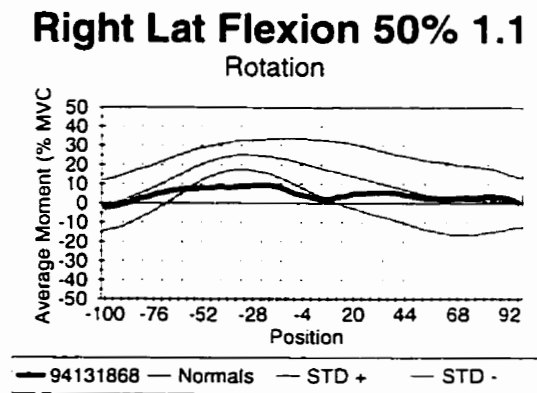
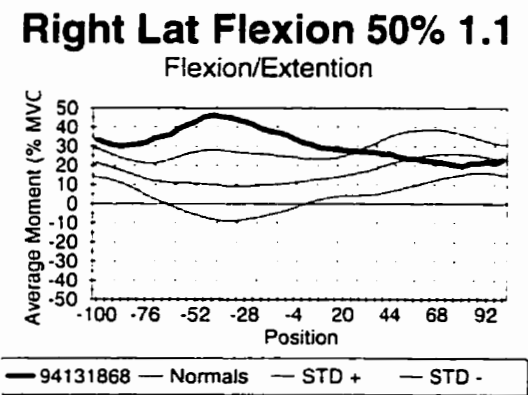
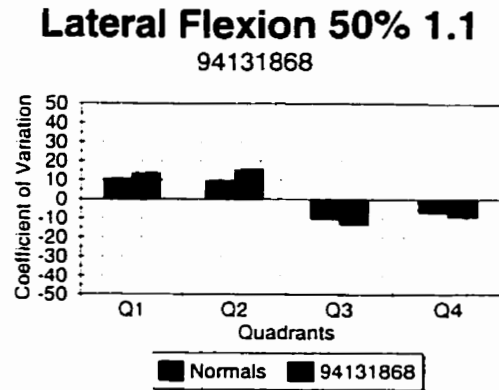
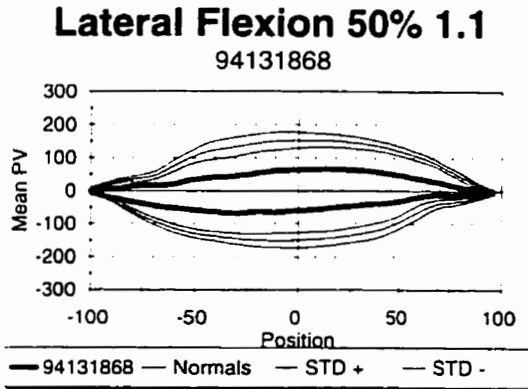
**Figure E12 B-200** summary performance for Case Study #3 (Bad day), for dynamic rotation at 50% resistance, day 2, test sequence #1.



**Figure E13 B-200** summary performance for Case Study #3 (Good day), for dynamic rotation at 50% resistance, day 1, test sequence #2.



**Figure E14 B-200** summary performance for Case Study #3 (Bad day), for dynamic rotation at 50% resistance, day 2, test sequence #2.



**Figure E15 B-200** summary performance for Case Study #3 (Good day), for dynamic lateral flexion at 50% resistance, day 1, test sequence #1.

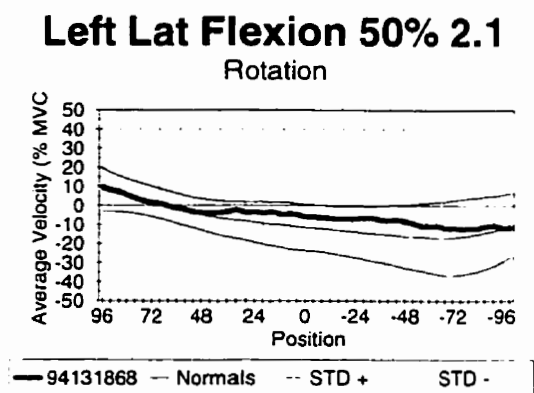
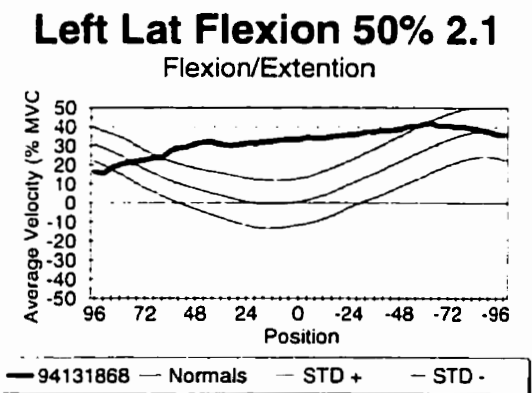
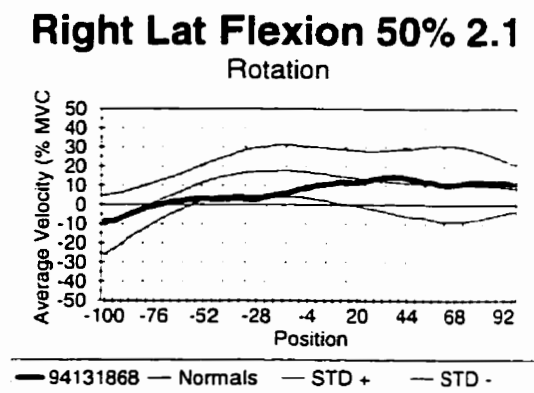
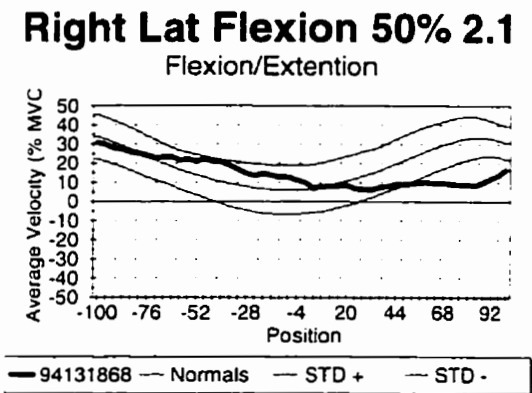
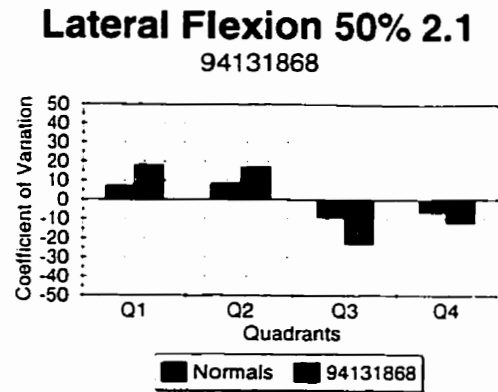
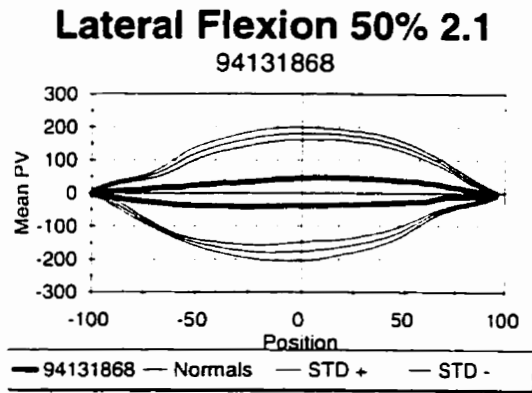


Figure E16 B-200 summary performance for Case Study #2 (Bad day), for dynamic lateral flexion at 50% resistance, day 2, test sequence #1.

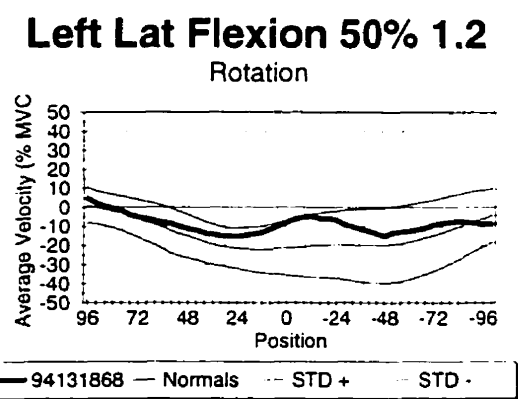
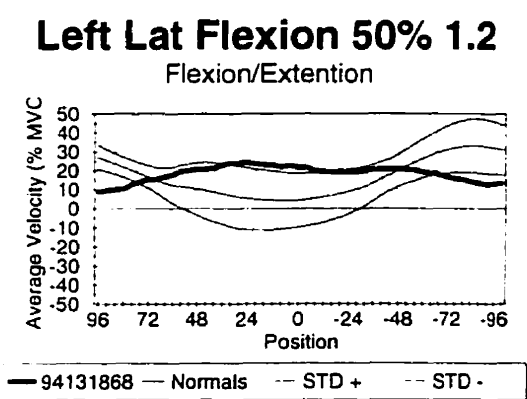
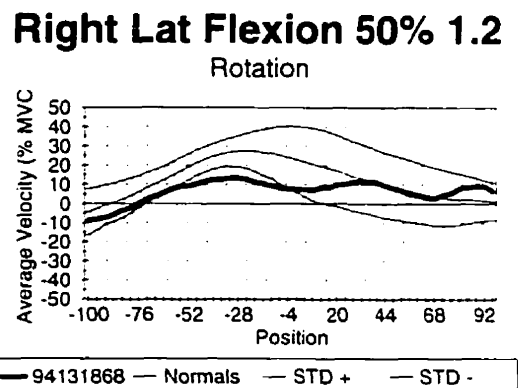
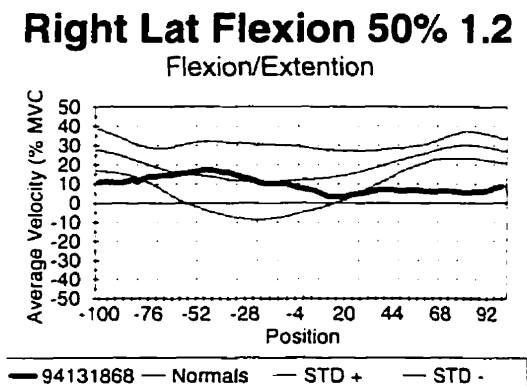
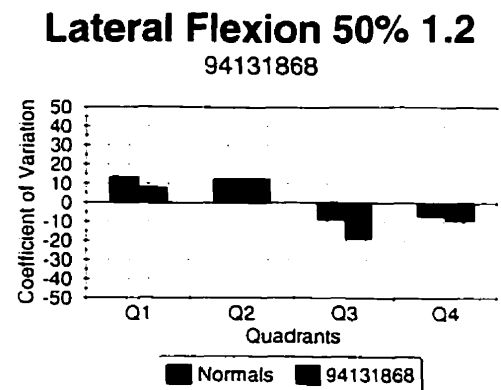
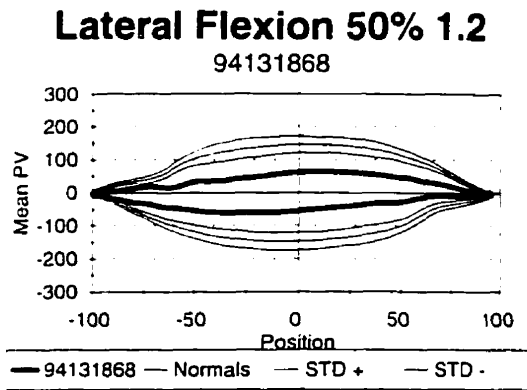


Figure E17 B-200 summary performance for Case Study #3 (Good day), for dynamic lateral flexion at 50% resistance, day 1, test sequence #2.

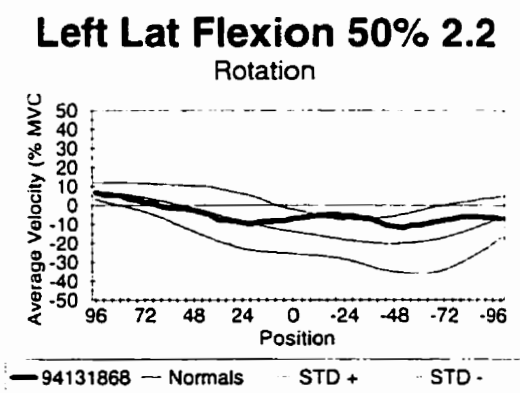
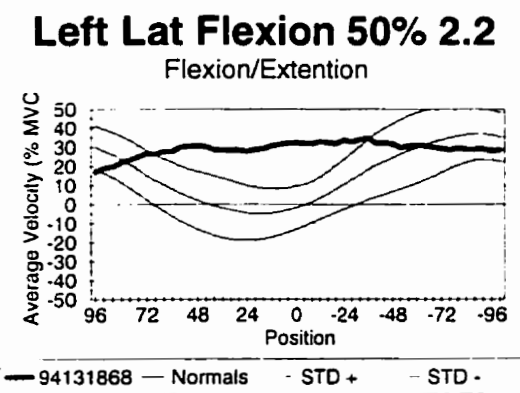
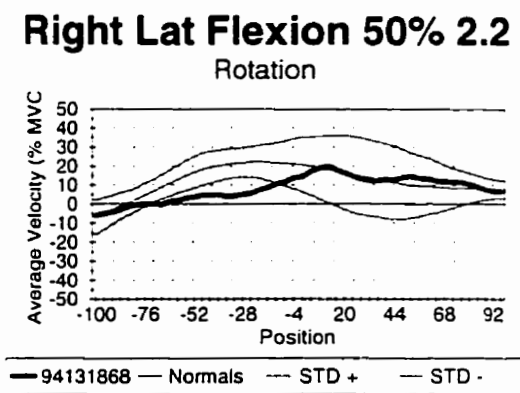
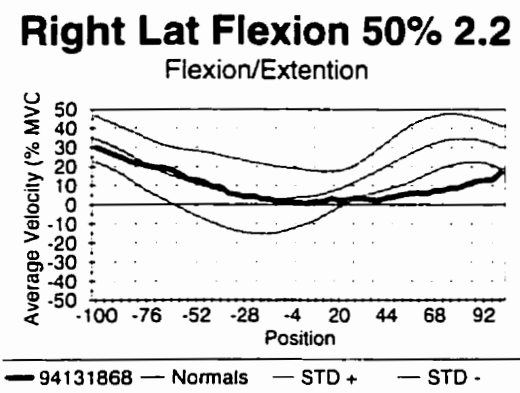
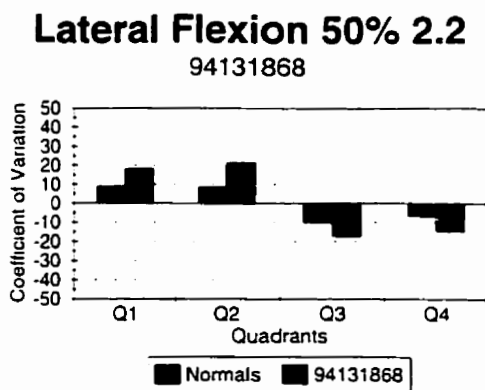
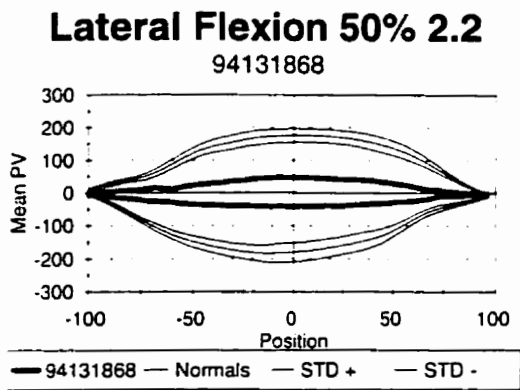
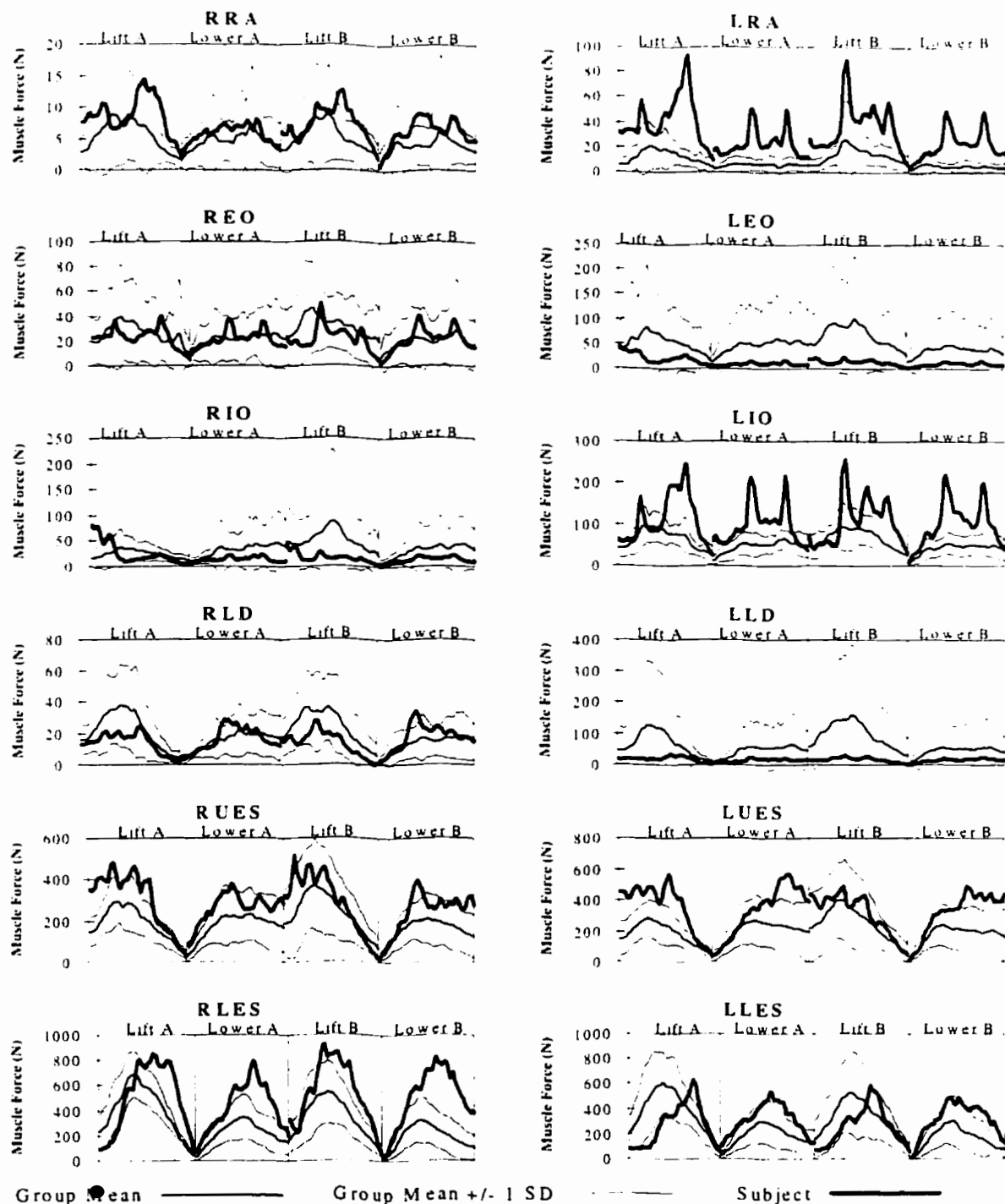
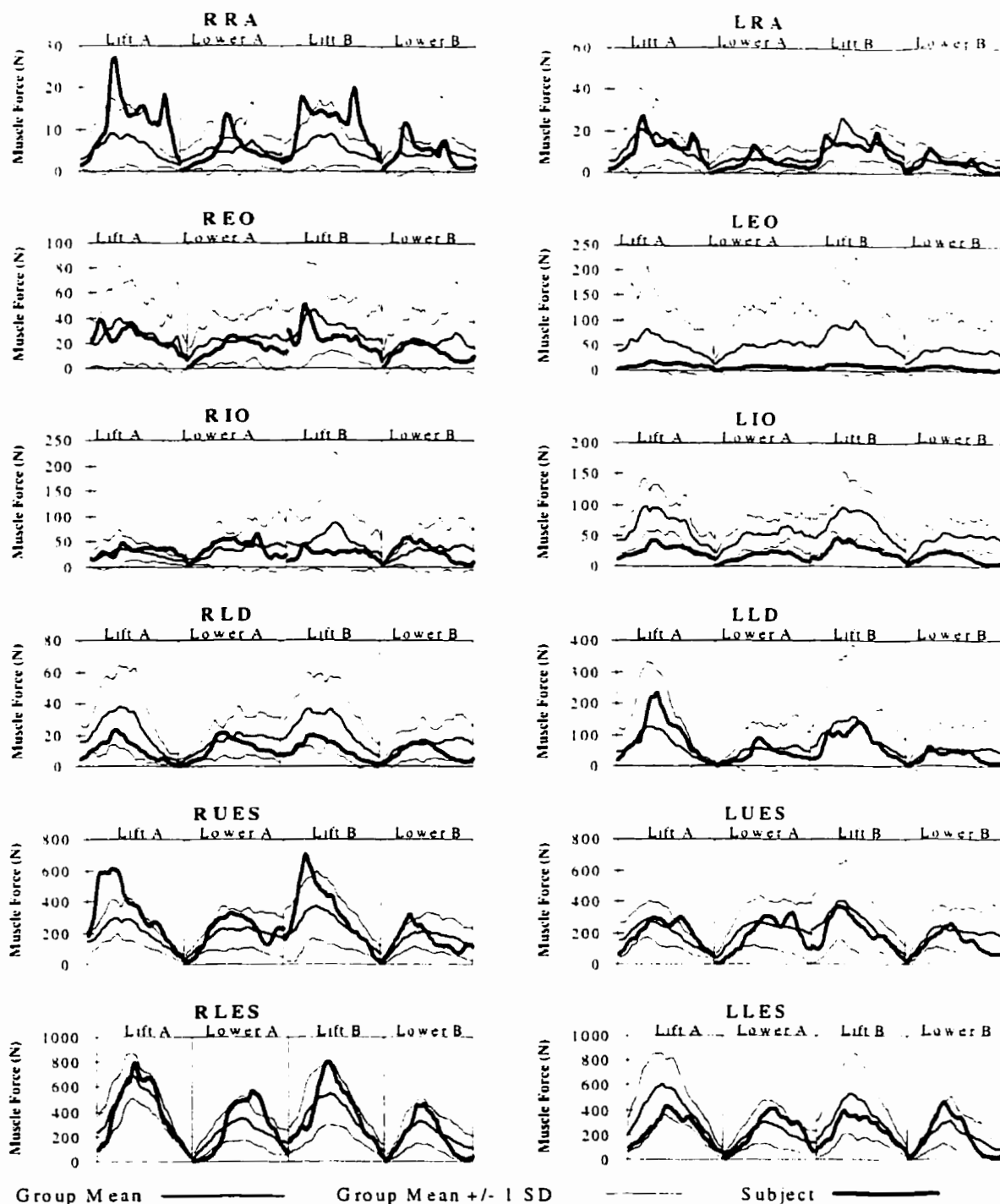


Figure E18 B-200 summary performance for Case Study #3 (Bad day), for dynamic lateral flexion at 50% resistance, day 2, test sequence #2.

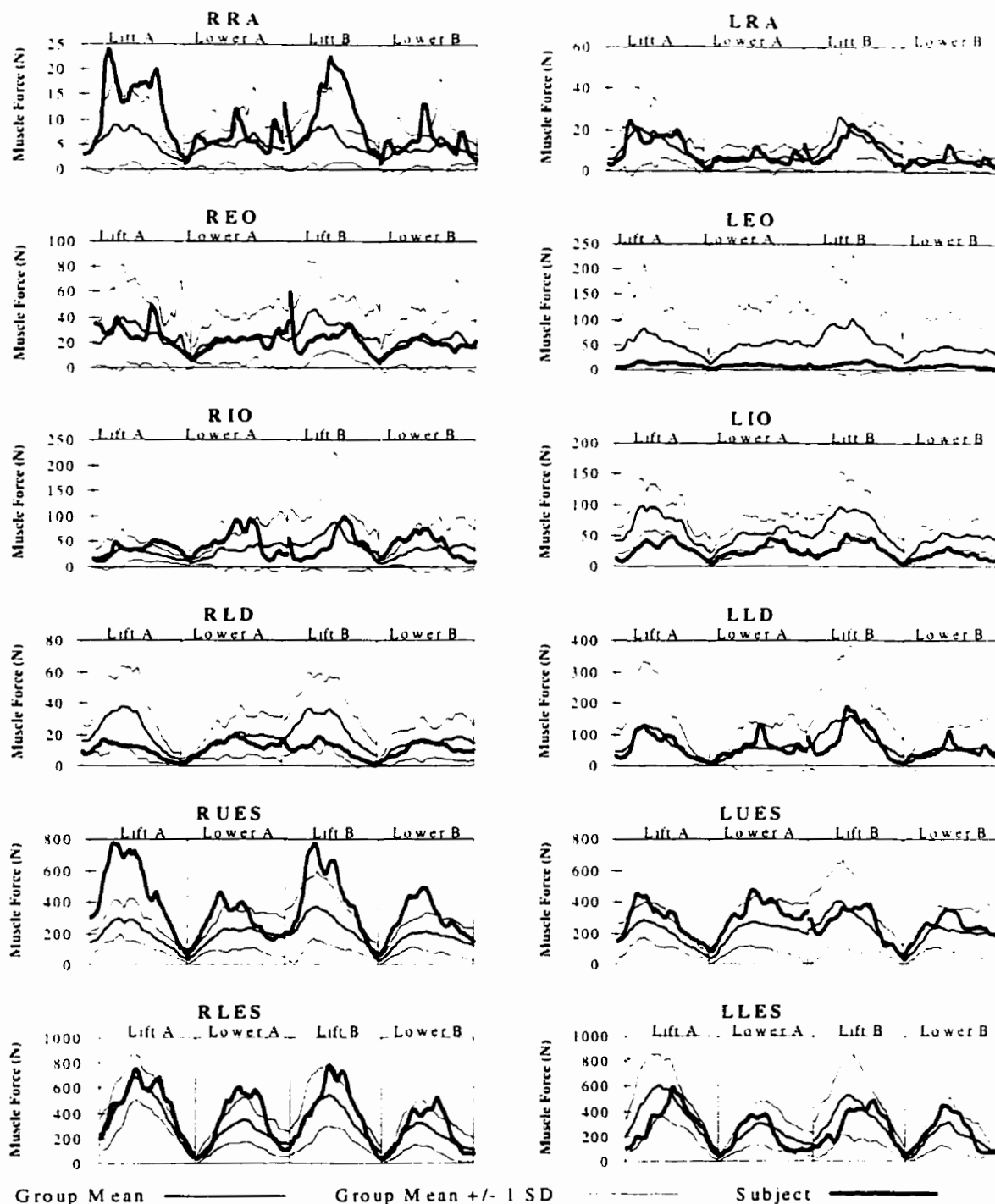


**Figure E19** Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) muscle force (N) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Good Day.

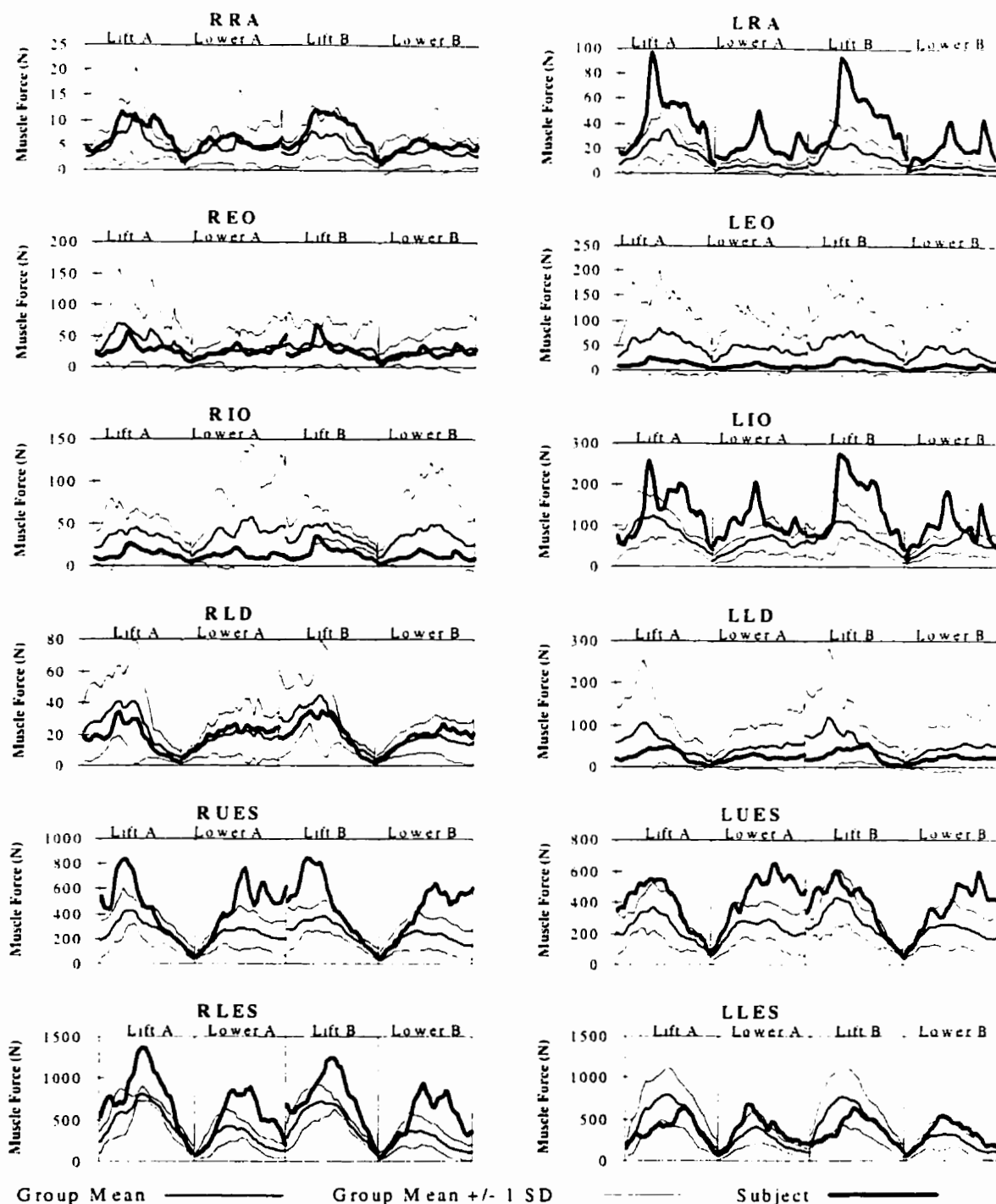




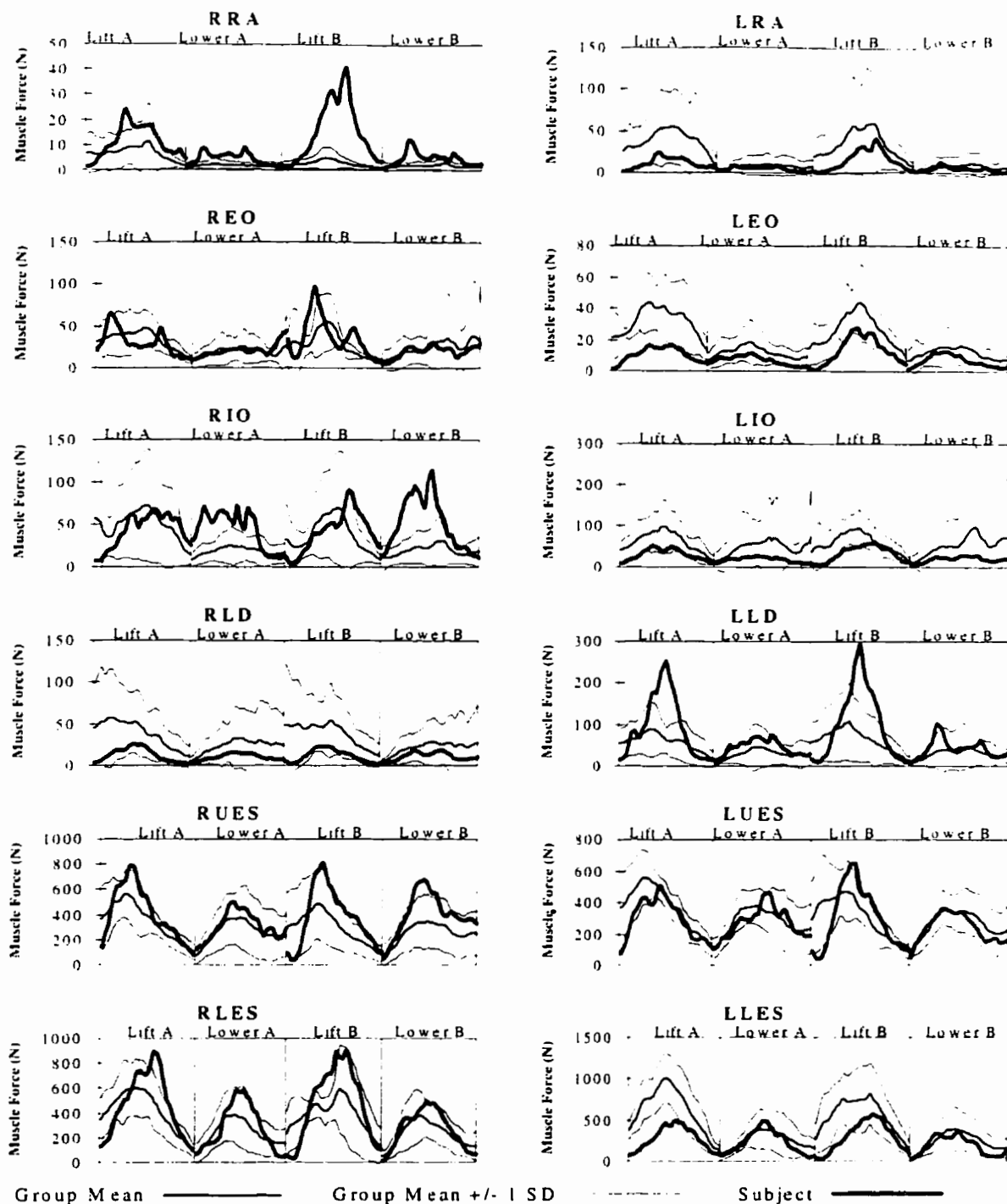
**Figure E20** Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) muscle force (N) for Day 2, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left. RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.



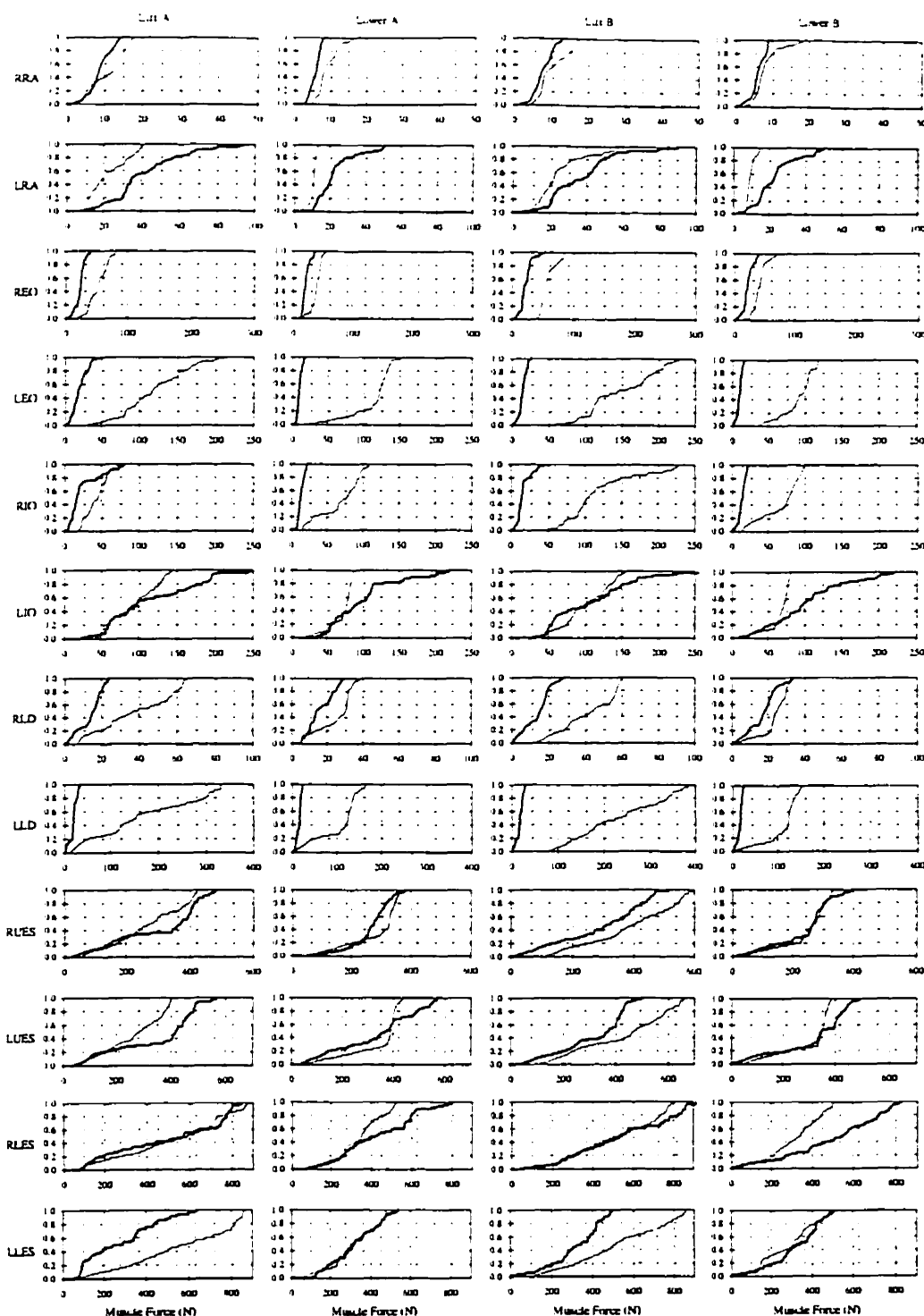
**Figure E21** Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) muscle force (N) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.



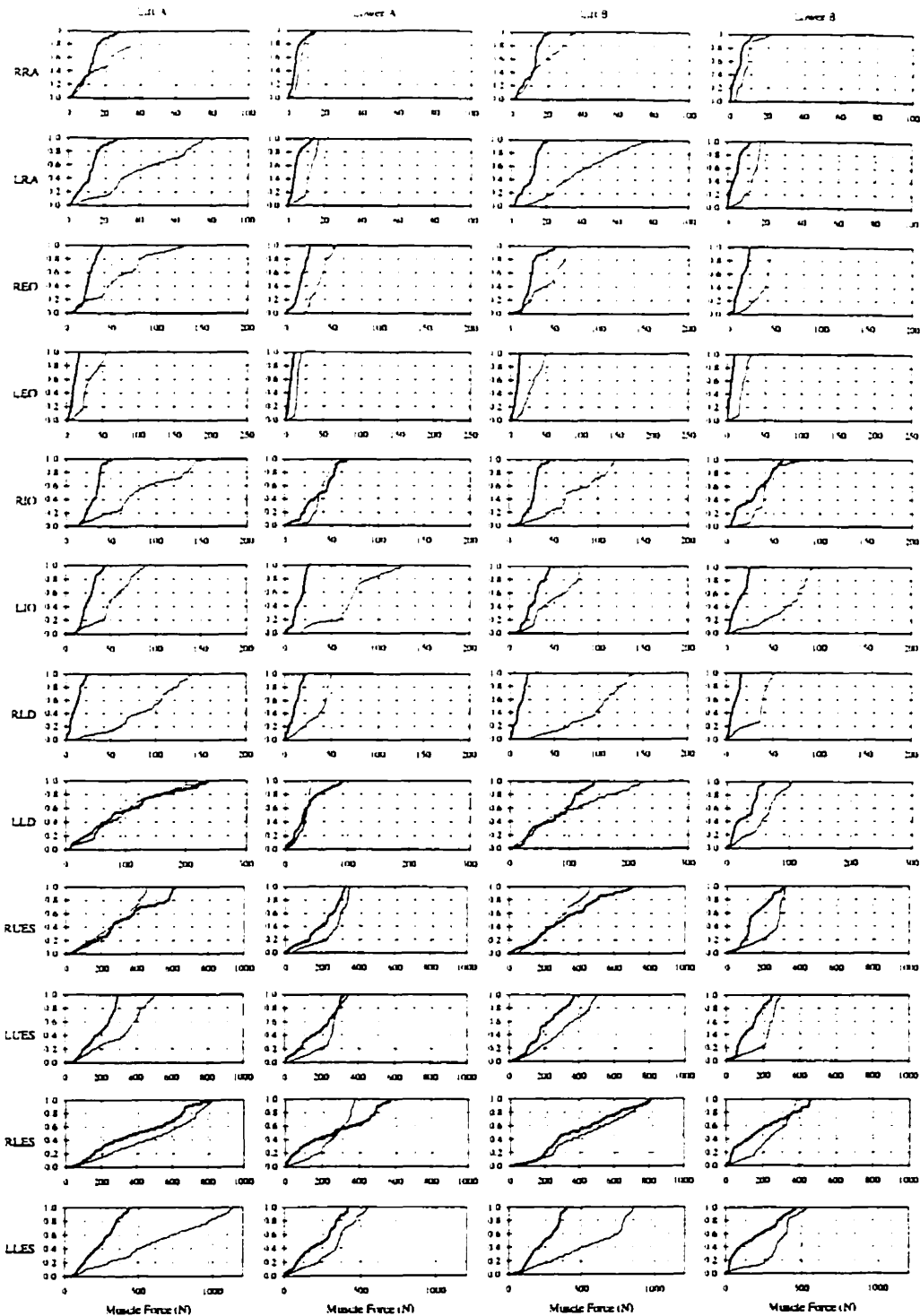
**Figure E22** Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (8 subjects) muscle force (N) for Day 1, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R = right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Good Day.



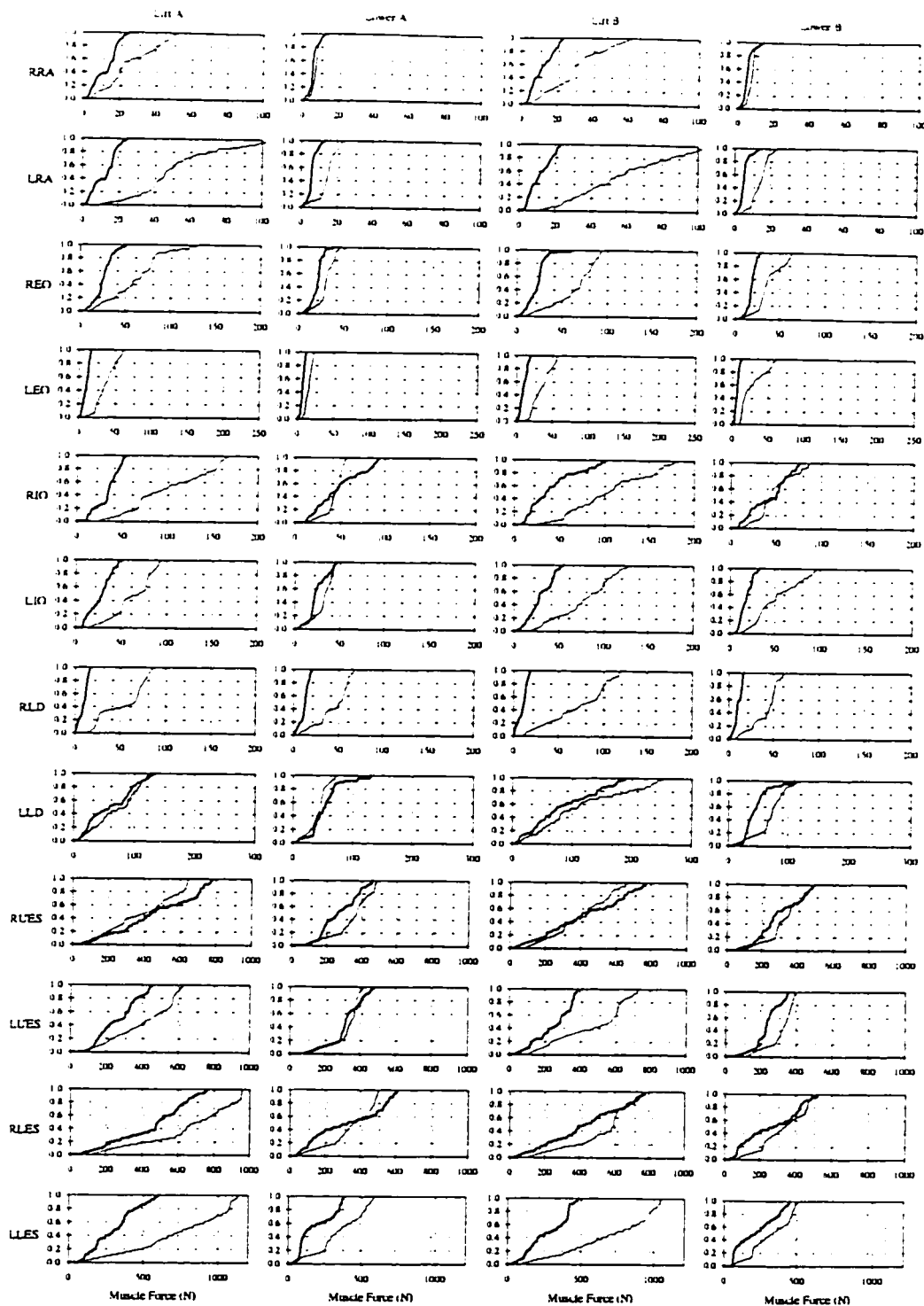
**Figure E23** Case Study #3 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (8 subjects) muscle force (N) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Bad Day.



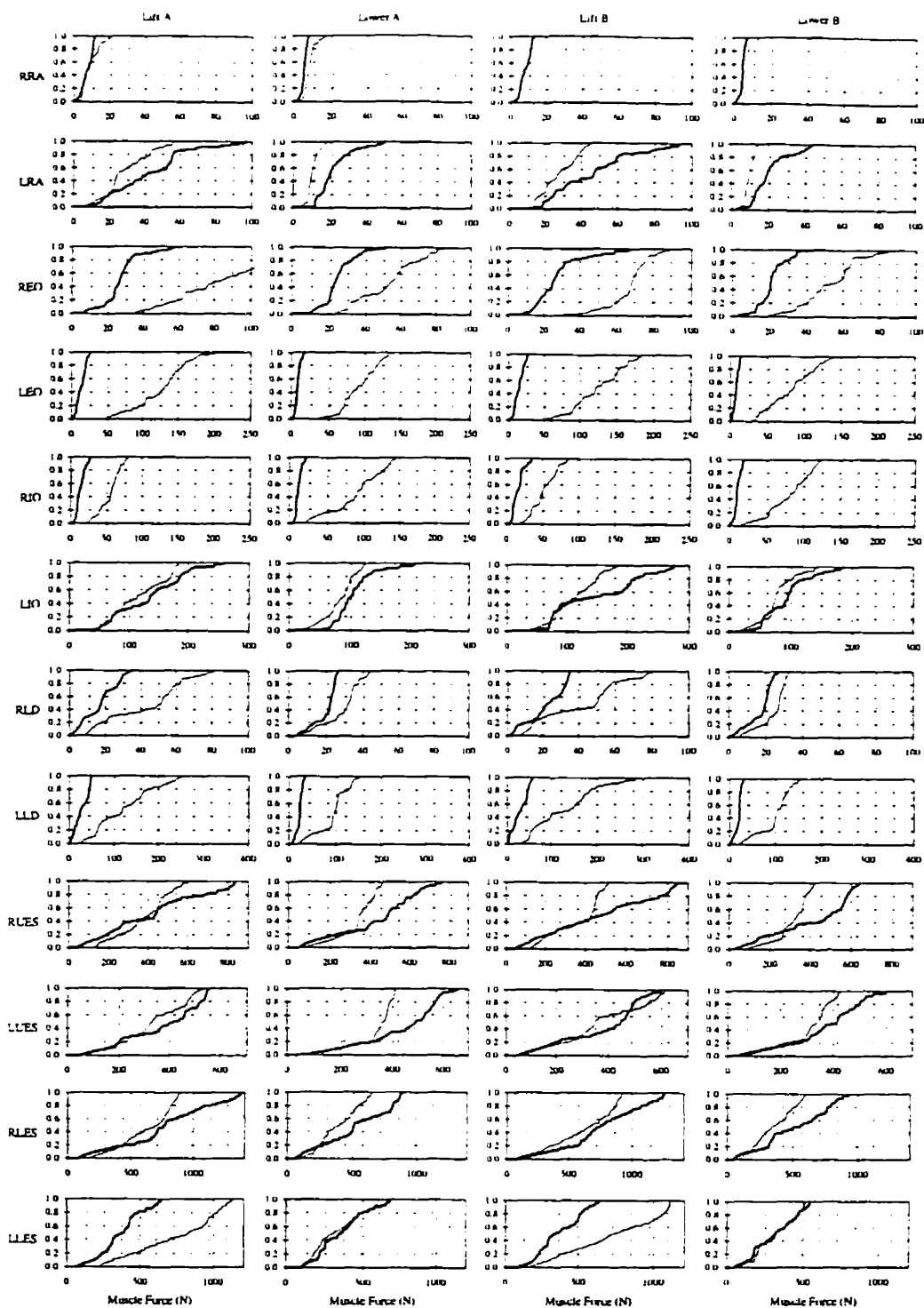
**Figure E24** Summary APDF profile of the muscle forces (N) by each electrode site for Day 1, 0 kg load and Case Study #3. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP (n = 9 subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 1 = Good Day.



**Figure E25** Summary APDF profile of the muscle forces (N) by each electrode site for Day 2, 0 kg load and Case Study #3. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 9$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 2 = Bad Day.

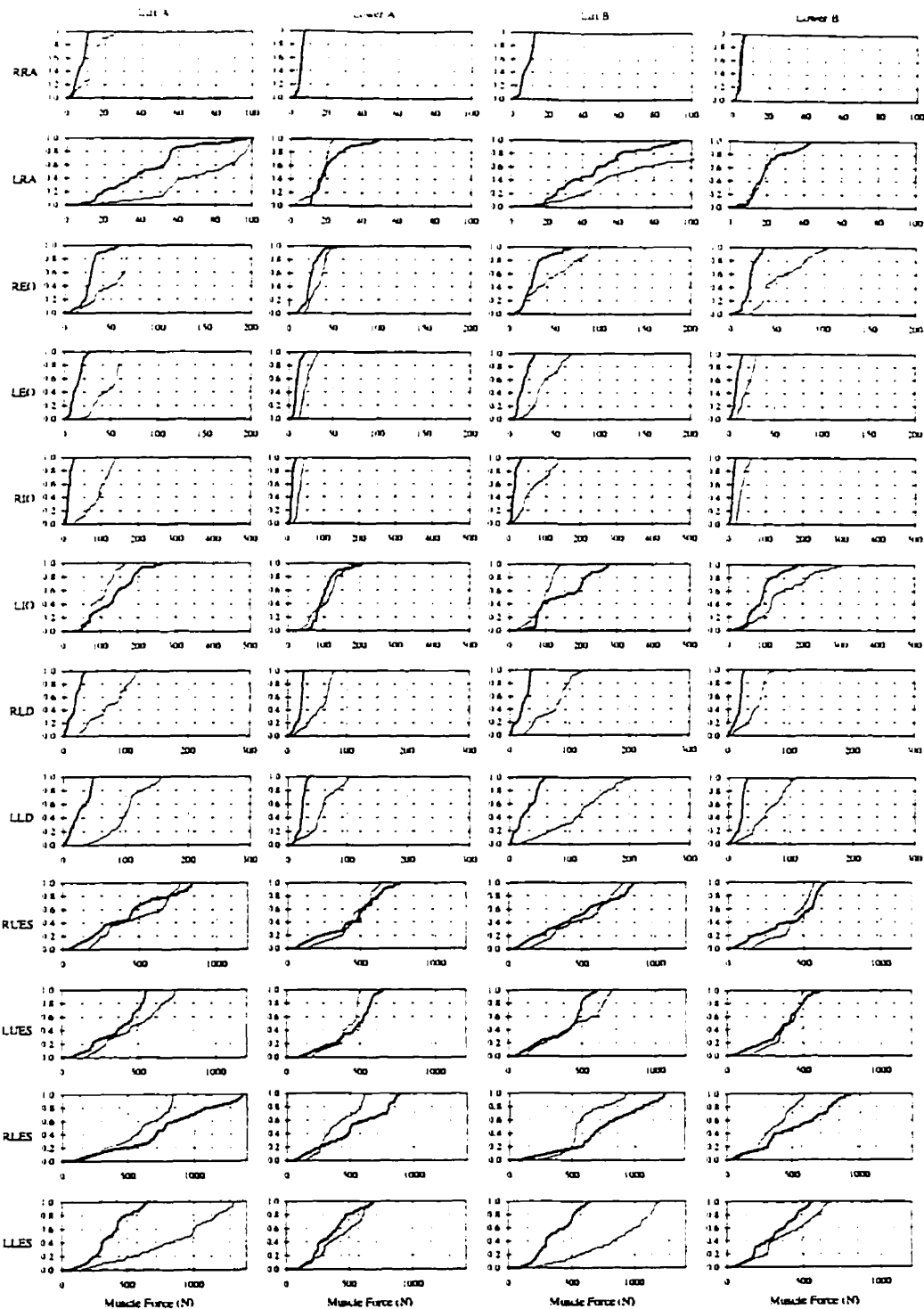


**Figure E26** Summary APDF profile of the muscle forces (N) by each electrode site for Day 2, 5 kg load and Case Study #3. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 9$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 2 = Bad Day.



**Figure E27** Summary APDF profile of the muscle forces (N) by each electrode site for Day 1, 10 kg load and Case Study #3. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 9$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 1 = Good Day.





**Figure E28** Summary APDF profile of the muscle forces (N) by each electrode site for Day 2, 10 kg load and Case Study #3. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 9$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 2 = Bad Day.

OOE Evaluation Results for

#94131868  
28-NOV-94

Demographic Data

Resistance Settings

Age : 33	Sex : M	Rotation 25% : 20 N-m
Height : 188.0 cm	Weight : 9.0 kg	Rotation 50% : 39 N-m
Diagnosis :		Flex/Ext 25% : 20 N-m
Surgical Category :		Flex/Ext 50% : 59 N-m
Activity Level Category :		Lat Flex 25% : 39 N-m
		Lat Flex 50% : 77 N-m

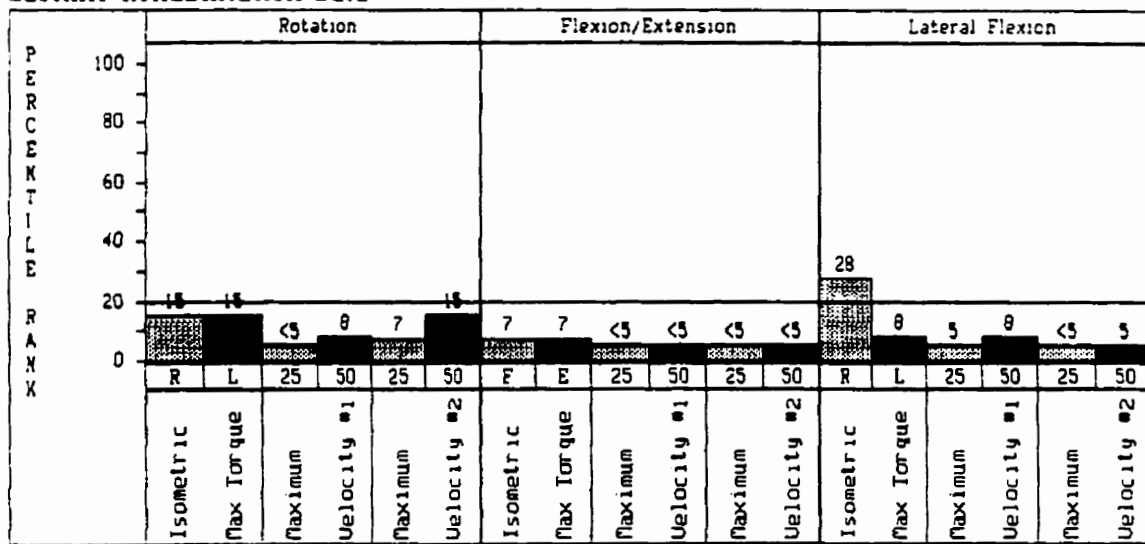
Abnormal Indicators 4

	Rotation		Flex/Ext		Lat Flex	
	25%	50%	25%	50%	25%	50%
Isometric Max Torque						
Max Velocity			↓	↓	↓	
Rotation Sec Max Torque						
Flex/Ext Sec Max Torque						
Lat Flex Sec Max Torque	↓					

Non-physiological Indicators 0

- 1) not observed
- 2) not observed
- 3) not observed
- 4) not observed
- 5) not observed
- 6) not observed

Baseline Rehabilitation Data



Test Administered By : Kardy Frazer      Signed : \_\_\_\_\_      Date : \_\_\_\_\_

Figure E29 Isostaion B-200 report (page 1) for Case Study #3 on Day 1 (Good Day).

## OOC Evaluation Results for

#94131868  
28-NOV-94

OOC Test	Rotation	Flex/Ext	Lat Flex
----------	----------	----------	----------

## Range of Motion

ROM (deg) #1	60.7	76.7	56.0
#2	71.8	77.2	55.1
ROM #2 (Δ%)	18.29	0.65	-1.61
ROM #1			

## Isometric

Max Torque (N-m)	78.9	116.9	163.0	155.1
------------------	------	-------	-------	-------

## Dynamic

		25%	50%	25%	50%	25%	50%
Resisted ROM (Δ%) #1		42.07	39.63†	3.75	5.63	-3.13	-13.29
	ROM #2	29.38†	22.16†	-8.07	-18.01	-1.59	-17.46
Avg Vel 50% (Δ%) #1		7.07		9.18		-4.09	
	Avg Vel 25% #2	-19.03		-27.35		-25.50	
Max Velocity (deg/sec) #1		73.1↓	83.0	89.4↓	94.8↓	74.4↓	77.0
	#2	104.8	94.9	97.5↓	83.9↓	79.5↓	69.3

## Secondary Axes

Rot Secondary #1	--	--	8.5	12.8	14.9↓	32.0
Max Torq (N-m) #2	--	--	17.1	8.5↓	32.0	25.6
F/E Secondary #1	40.0	58.4	--	--	21.5↓	83.0
Max Torq (N-m) #2	52.3	58.4	--	--	46.1	46.1↓
L F Secondary #1	30.1↓	56.0	12.9	15.1	--	--
Max Torq (N-m) #2	30.1↓	49.5	12.9	8.6	--	--

## Key

↓ Subnormal, less than critical level  
 † Supernormal, greater than critical level  
 (see Statistical Review in QOC Back Evaluation System)

Version 3.0

Figure E30 Isostaion B-200 report (page 2) for Case Study #3 on Day 1 (Good Day).

OOE Evaluation Results for

#94131868  
06-JAN-95

Demographic Data

Resistance Settings

Age : 33	Sex : M	Rotation 25% : 20 N-m
Height : 189.0 cm	Weight : 00 kg	Rotation 50% : 42 N-m
Diagnosis :		Flex/Ext 25% : 19 N-m
Surgical Category :		Flex/Ext 50% : 37 N-m
Activity Level Category :		Lat Flex 25% : 30 N-m
		Lat Flex 50% : 60 N-m

Abnormal Indicators 9

	Rotation		Flex/Ext		Lat Flex	
	25%	50%	25%	50%	25%	50%
Isometric Max Torque			↓			
Max Velocity	↓	↓	↓	↓	↓	↓
Rotation Sec Max Torque						
Flex/Ext Sec Max Torque						
Lat Flex Sec Max Torque	↓					

Non-physiological Indicators 0

- 1) not observed
- 2) not observed
- 3) not observed
- 4) not observed
- 5) not observed
- 6) not observed

Baseline Rehabilitation Data

PERCENTILE RANK	Rotation						Flexion/Extension						Lateral Flexion							
	R		L		S		F		E		S		R		L		S			
	25	50	25	50	25	50	25	50	25	50	25	50	25	50	25	50	25	50		
	Maximum	Velocity #1	Maximum	Velocity #2	Maximum	Velocity #1	Maximum	Velocity #2	Maximum	Velocity #1	Maximum	Velocity #2	Maximum	Velocity #1	Maximum	Velocity #2	Maximum	Velocity #1	Maximum	Velocity #2
100																				
80																				
60																				
40																				
20																				
15																				
10																				
5																				
0																				

Test Administered By : Nardy Frazer      Signed : \_\_\_\_\_      Date : \_\_\_\_\_

Figure E31 Isostaion B-200 report (page 1) for Case Study #3 on Day 2 (Bad Day).

## OOC Evaluation Results for

#94131868

06-JAN-95

OOC Test	Rotation	Flex/Ext	Lat Flex
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## Range of Motion

ROM (deg) #1	90.6	91.5	53.8
#2	91.4	71.4	54.3
ROM #2 (Δ%)			
ROM #1	0.99	-12.39	0.93

## Isometric

Max Torque (N-m)	83.1	73.8↓ 147.6	118.5
------------------	------	-------------	-------

## Dynamic

		25%	50%	25%	50%	25%	50%
Resisted ROM (Δ%)	#1	5.50	-10.09	-25.29↓	-29.41↓	-15.45	-19.51
	#2	6.36	-15.00	-0.67	-15.44	-1.61	-11.29
Avg Vel 50% (Δ%)	#1	-19.13		-10.55		7.98	
	#2	-22.88		-12.44		-32.76	
Max Velocity (deg/sec)	#1	79.1↓	69.2↓	75.8↓	67.7↓	43.6↓	53.9↓
	#2	79.1↓	69.2↓	75.8↓	70.4↓	69.3↓	53.9↓

## Secondary Axes

Rot Secondary #1	..	..	8.5	8.5↓	19.2	23.5↓
Max Torq (N-m) #2	..	..	12.8	12.8	25.6	27.7
F/E Secondary #1	30.8	58.4	..	..	15.4↓	52.3
Max Torq (N-m) #2	30.8↓	46.1	..	..	30.8↓	49.2↓
L F Secondary #1	32.3↓	62.4	10.8	15.1	..	..
Max Torq (N-m) #2	36.6↓	68.9	12.9	12.9	..	..

## Key

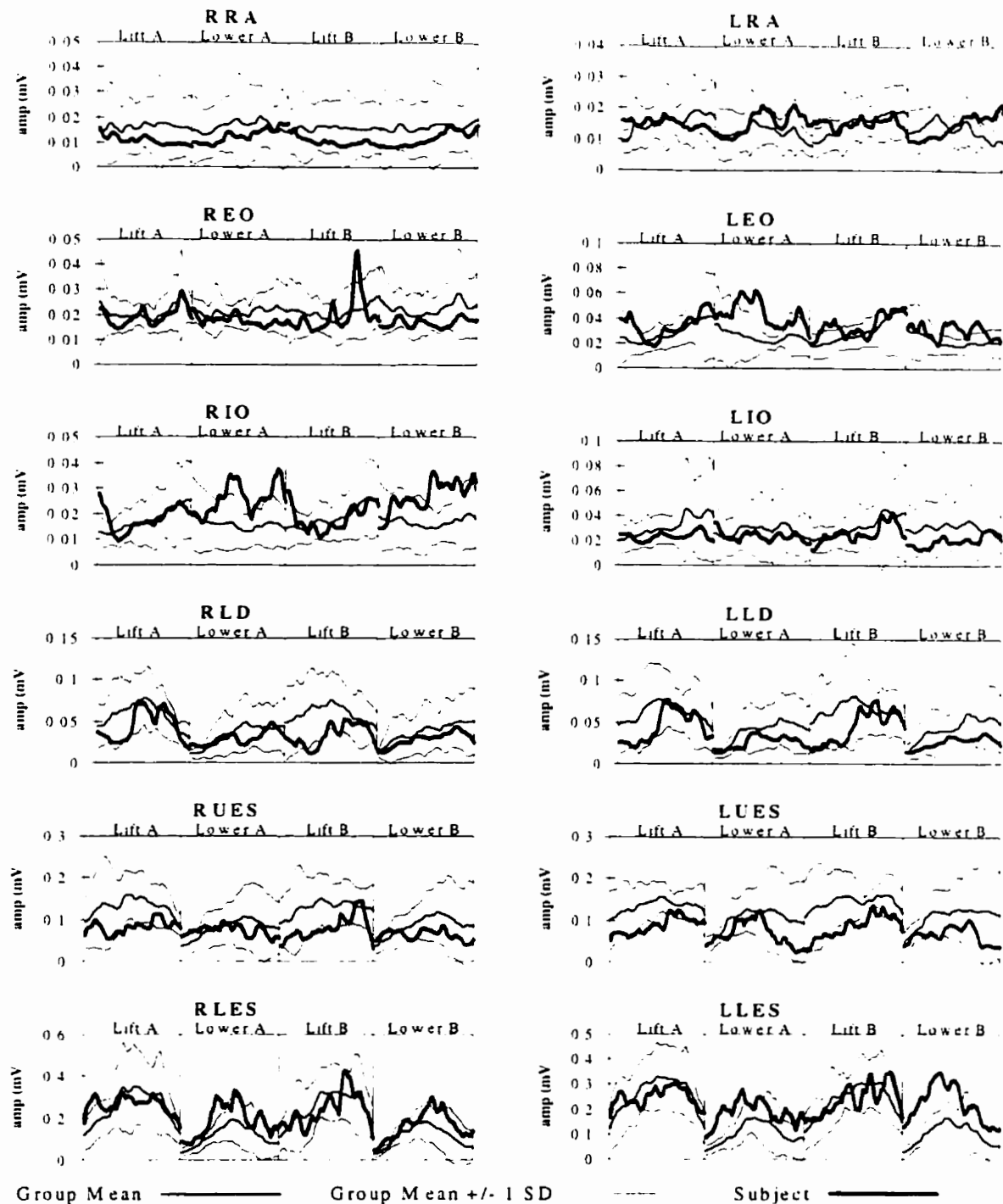
↓ Subnormal, less than critical level  
 ↑ Supernormal, greater than critical level  
 (see Statistical Review in OOC Back Evaluation System)

Version 3.0

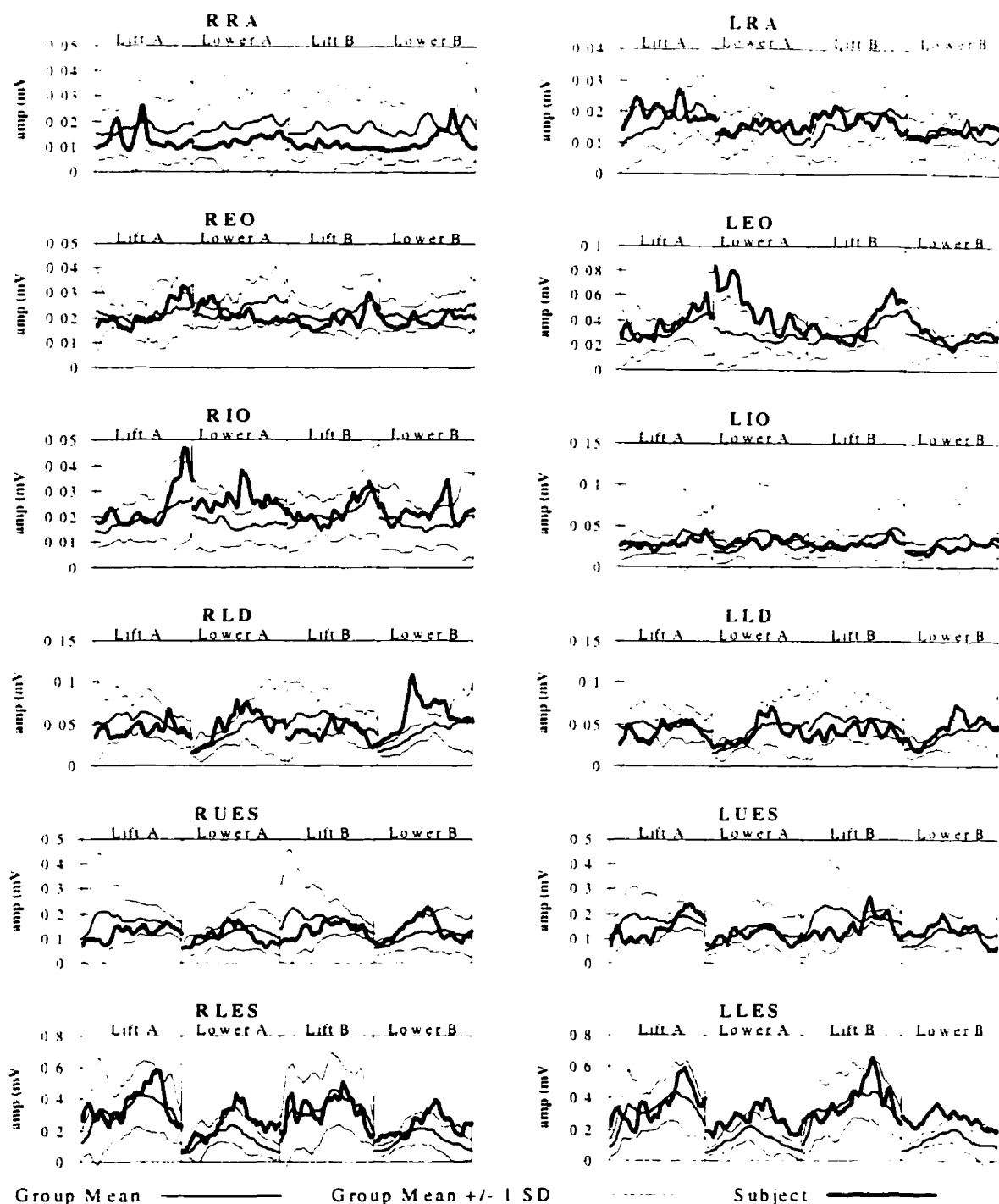
Figure E32 Isostaion B-200 report (page 2) for Case Study #3 on Day 2 (Bad Day).

## **Appendix F**

Case Study #4

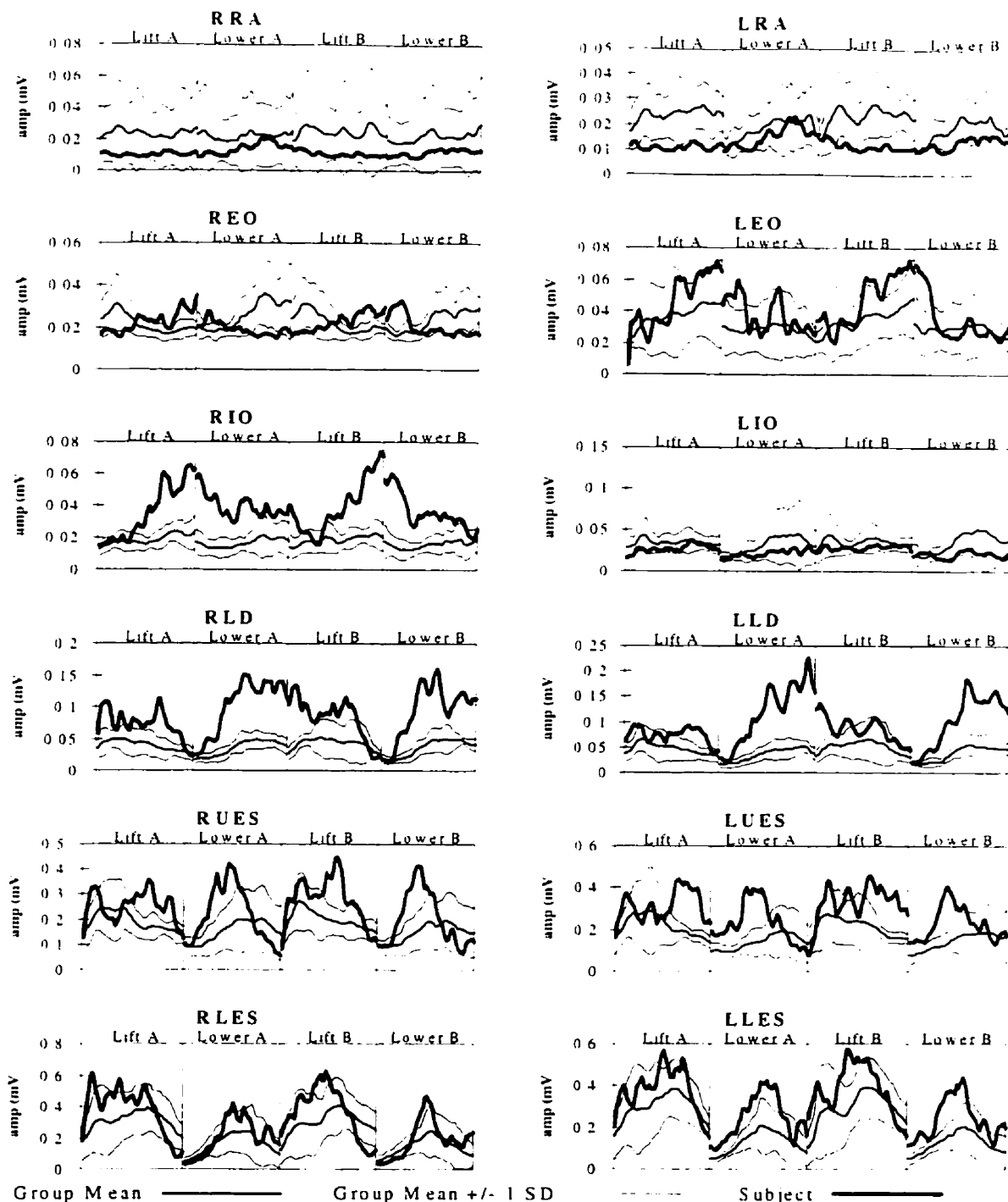


**Figure F1** Case Study #4 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (mV) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Bad Day.

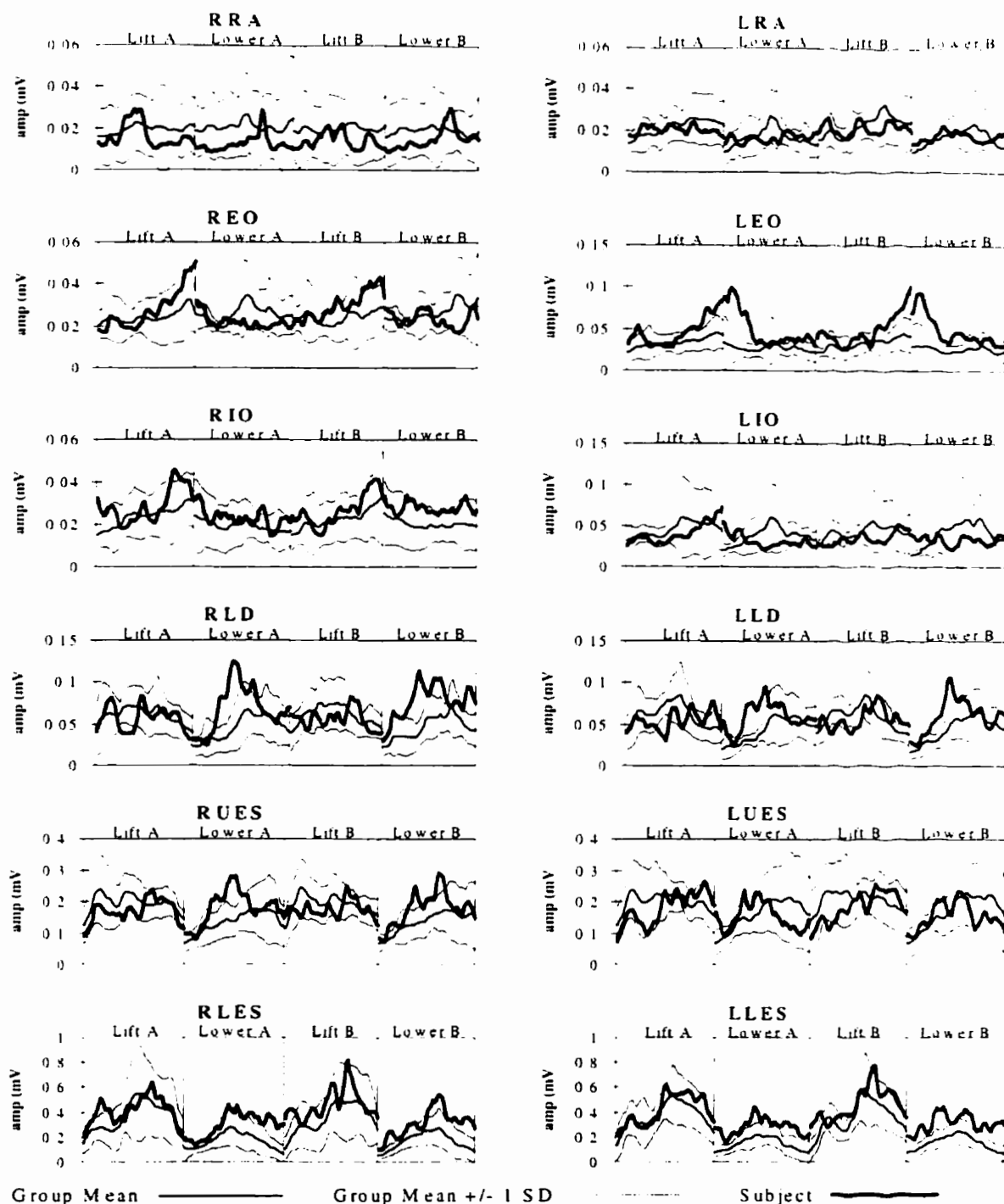


**Figure F2** Case Study #4 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (mV) for Day 1, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Bad Day.

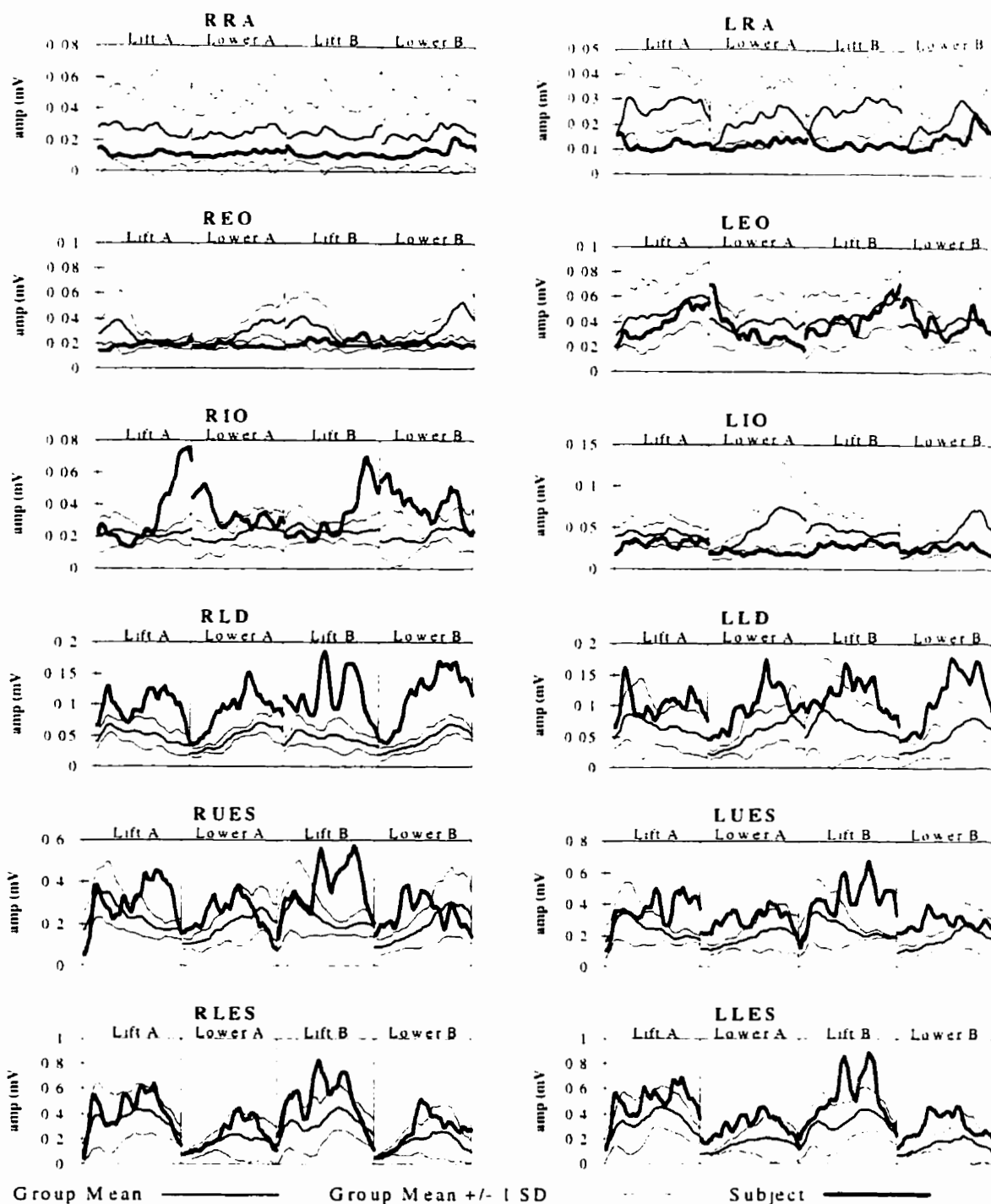




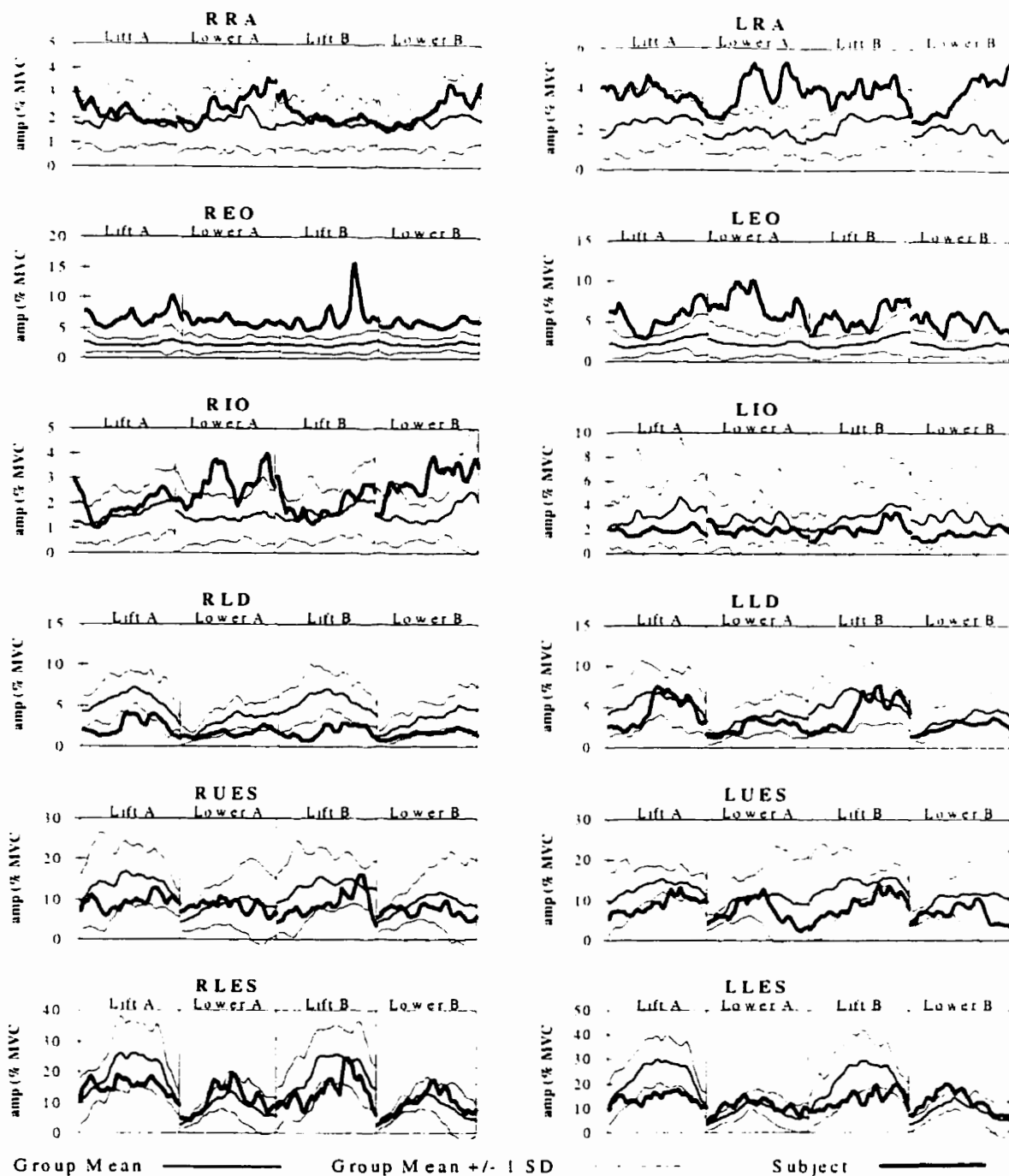
**Figure F3** Case Study #4 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (mV) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Good Day.



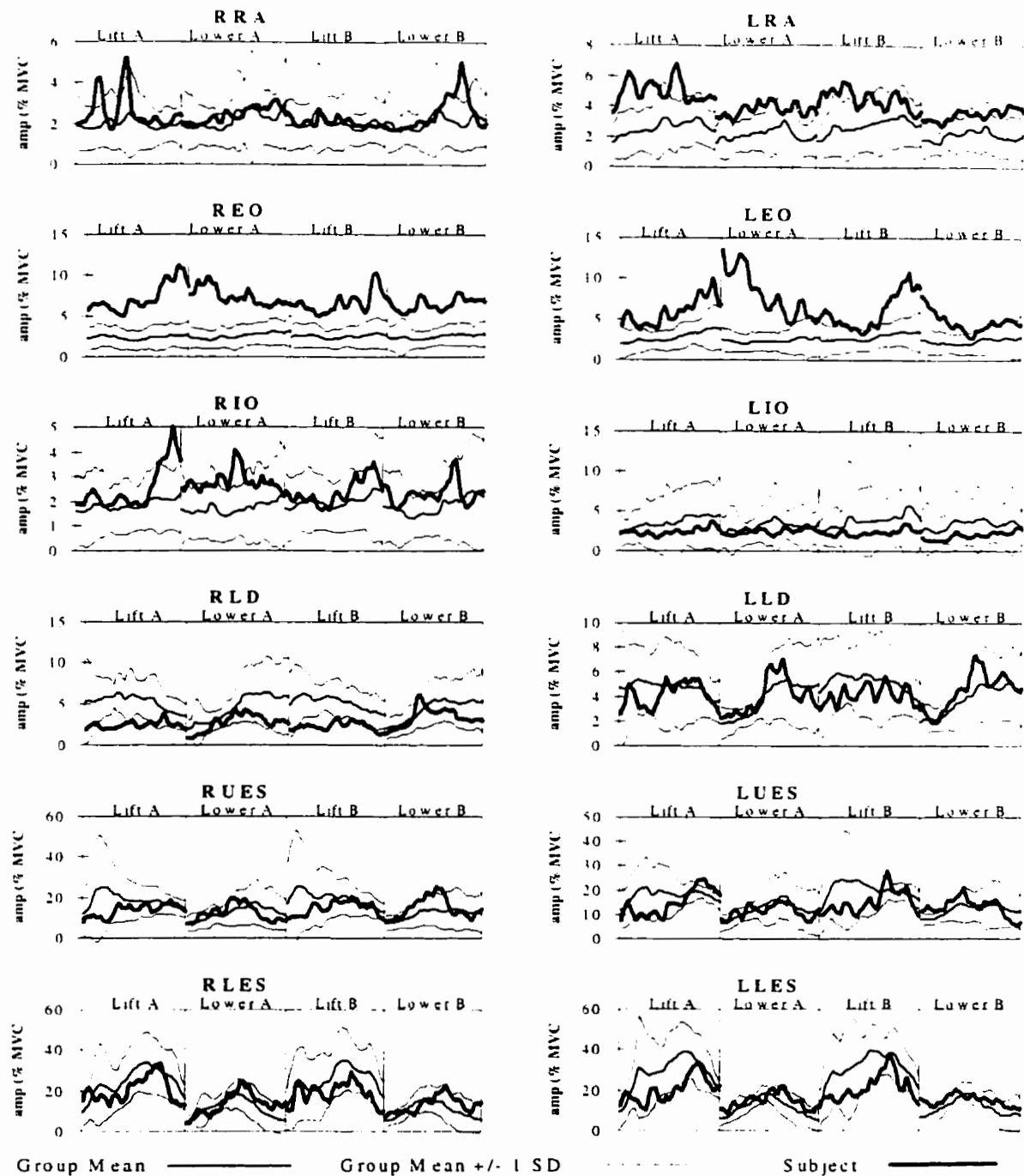
**Figure F4** Case Study #4 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (mV) for Day 1, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Bad Day.



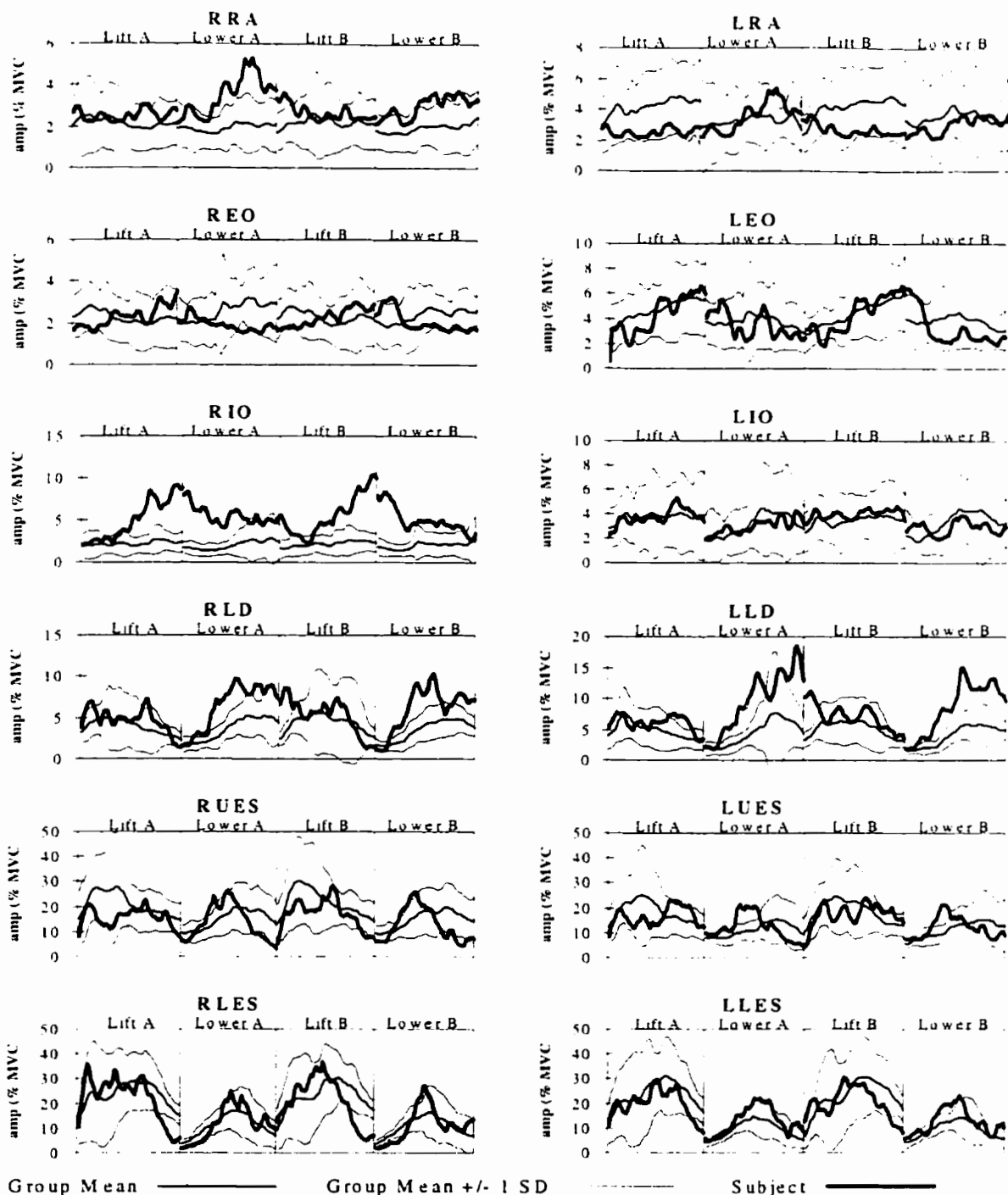
**Figure F5** Case Study #4 graphed against the summary profile of the mean ( $\pm$ 1 SD) ASYMP (10 subjects) EMG activity levels (mV) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Good Day.



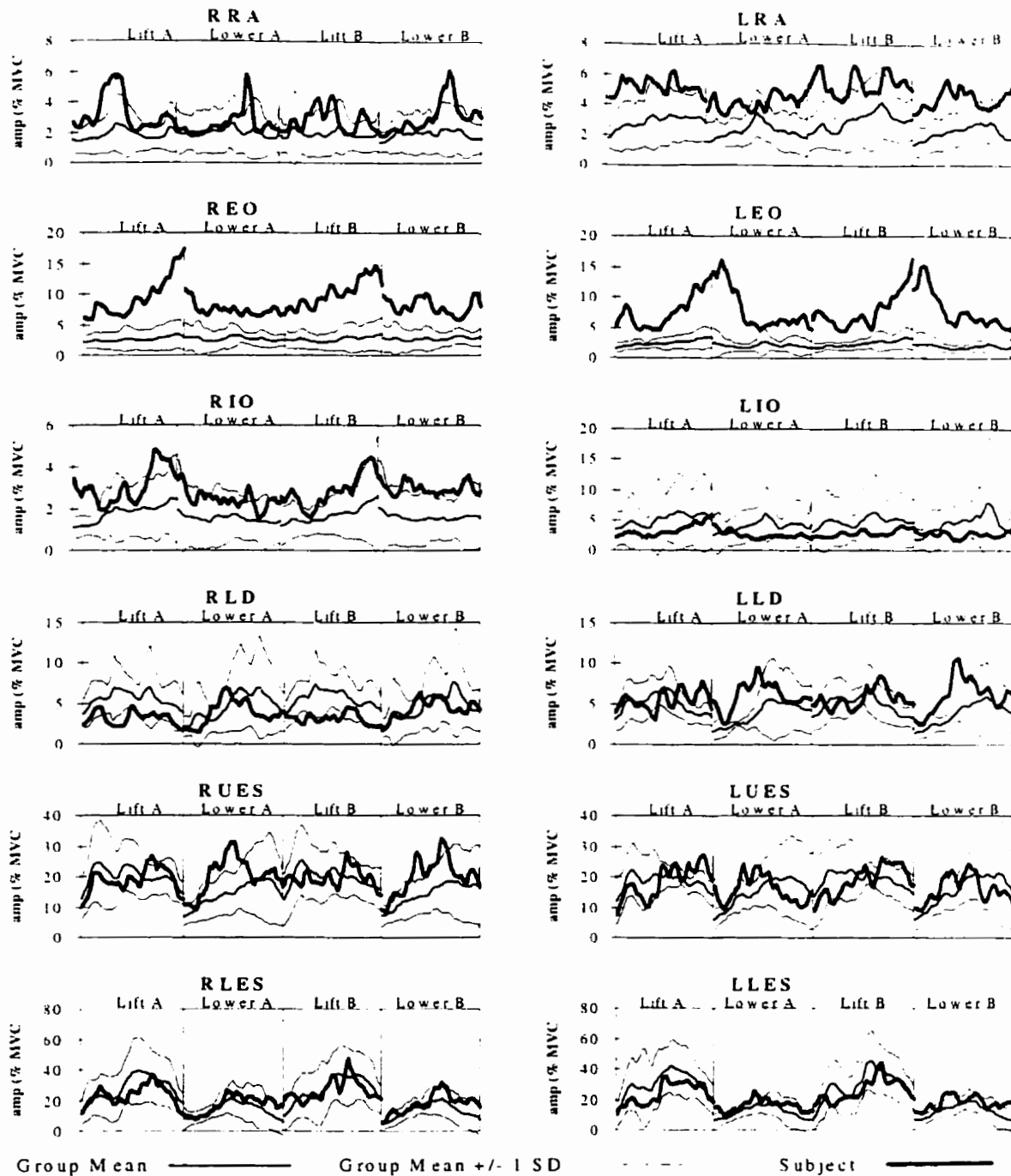
**Figure F6** Case Study #4 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (% MVC) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Bad Day.



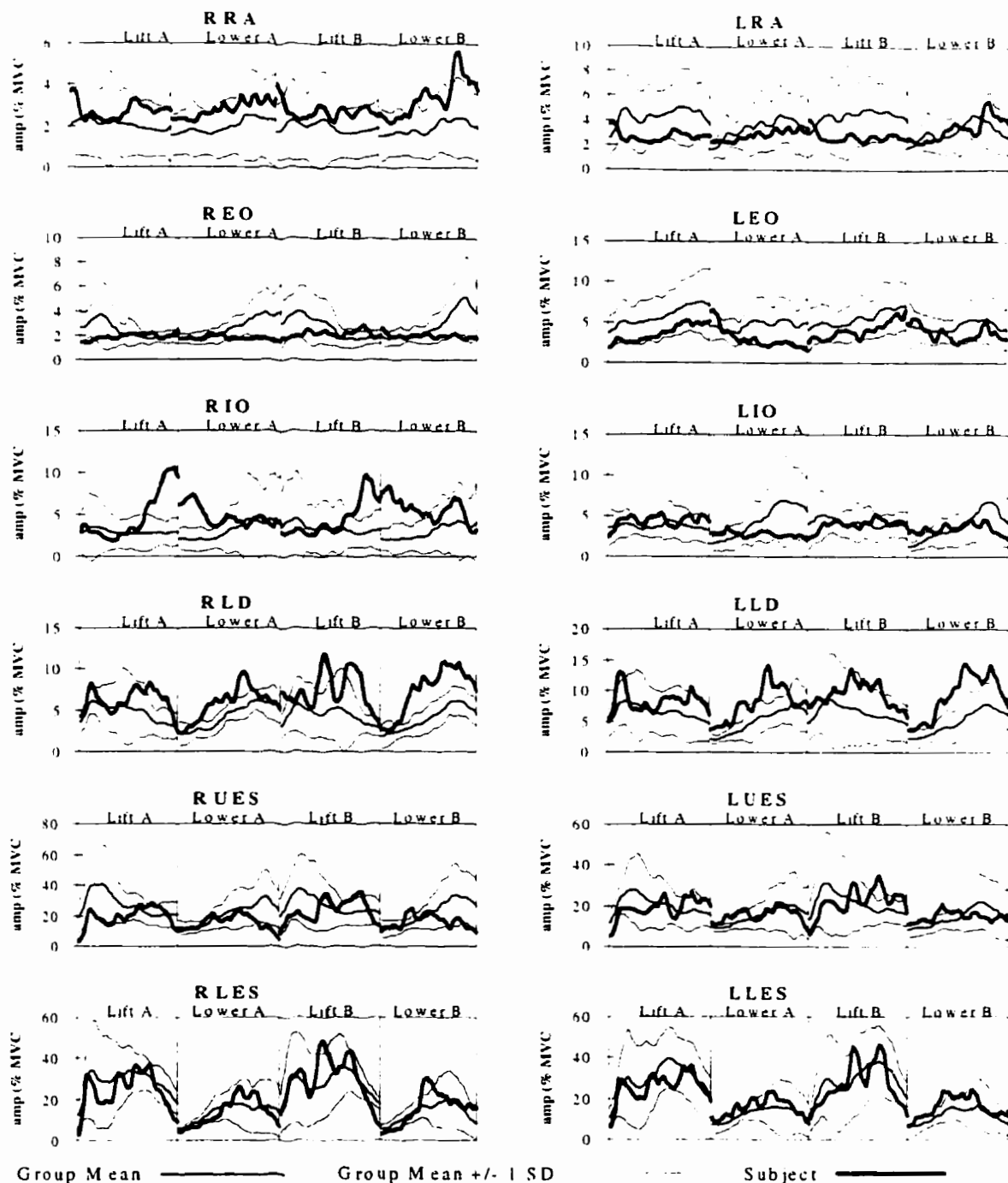
**Figure F7** Case Study #4 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (% MVC) for Day 1, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Bad Day.



**Figure F8** Case Study #4 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Good Day.

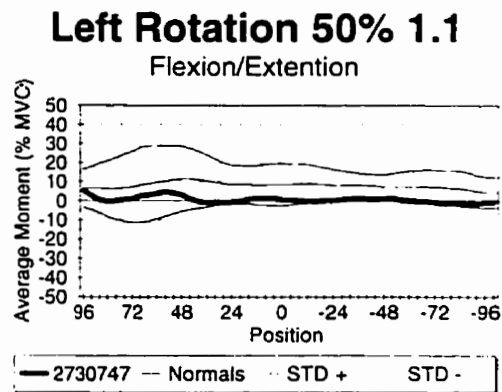
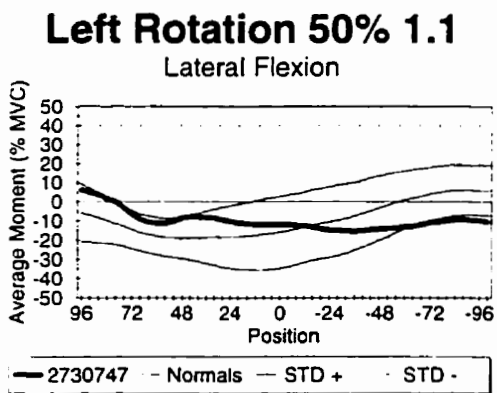
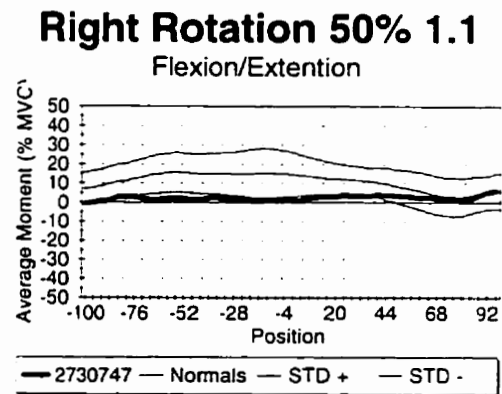
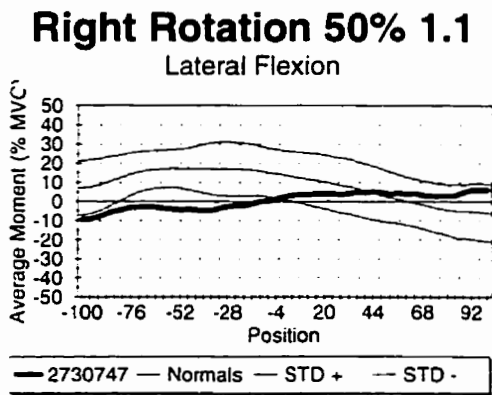
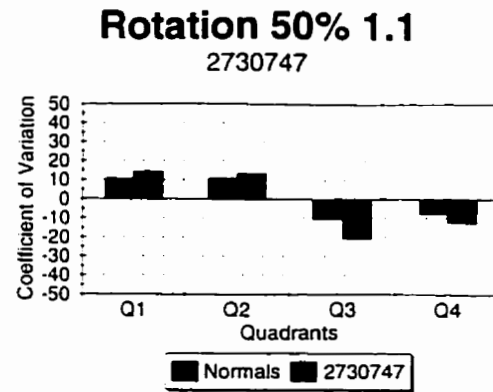
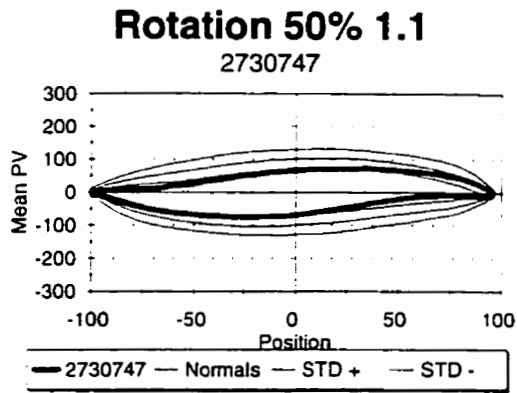


**Figure F9** Case Study #4 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) EMG activity levels (% MVC) for Day 1, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Bad Day.

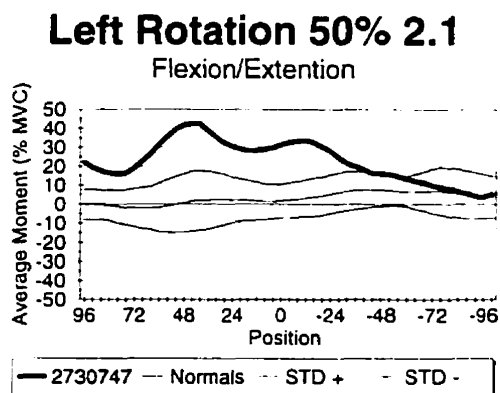
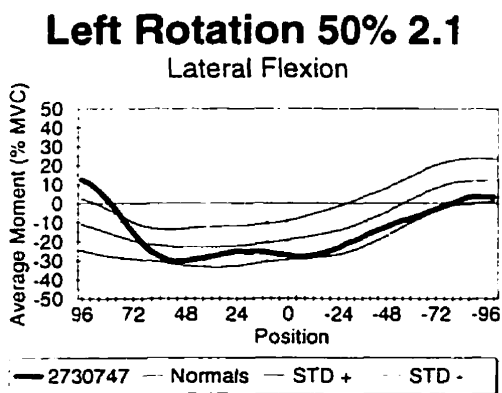
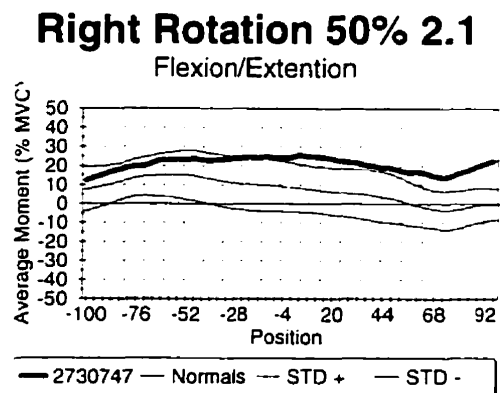
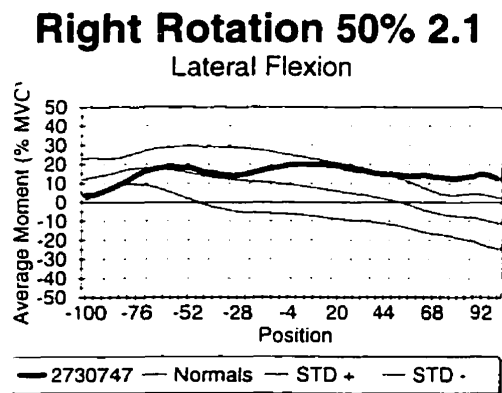
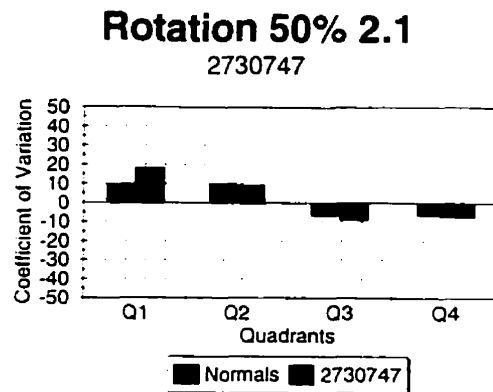
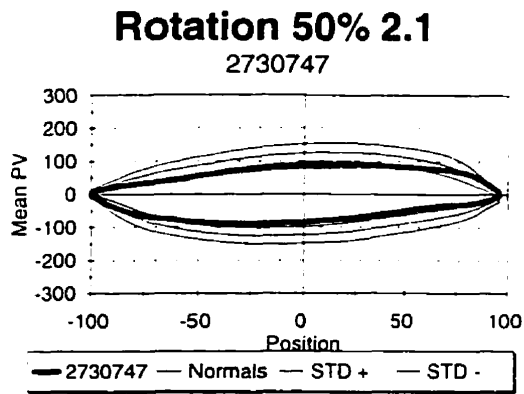


**Figure F10** Case Study #4 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (10 subjects) EMG activity levels (% MVC) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA = rectus abdominis, EO = external oblique. IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Good Day.

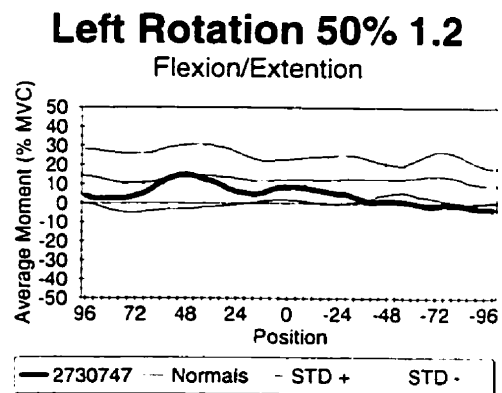
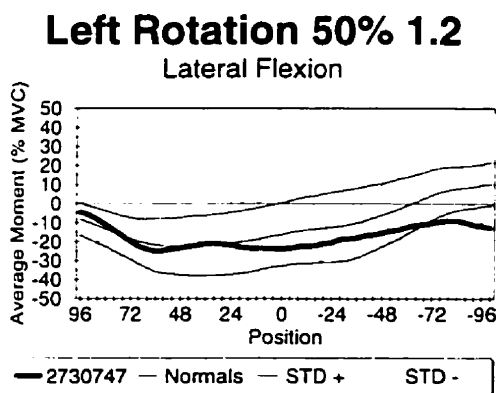
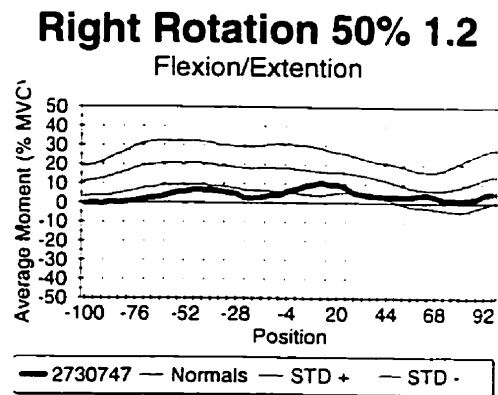
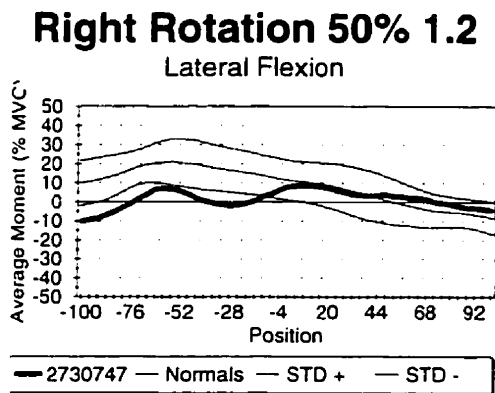
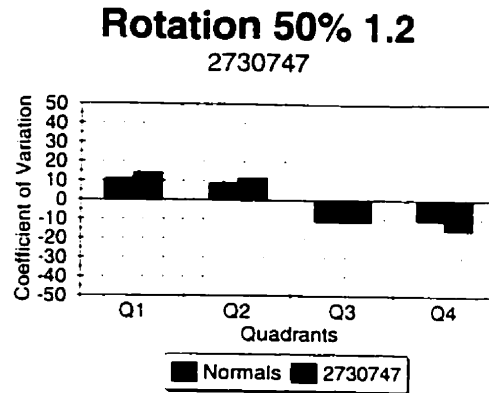
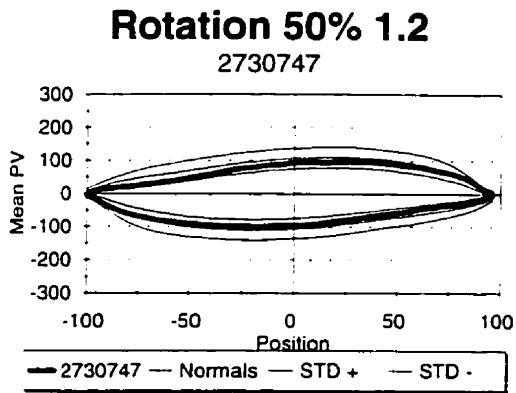




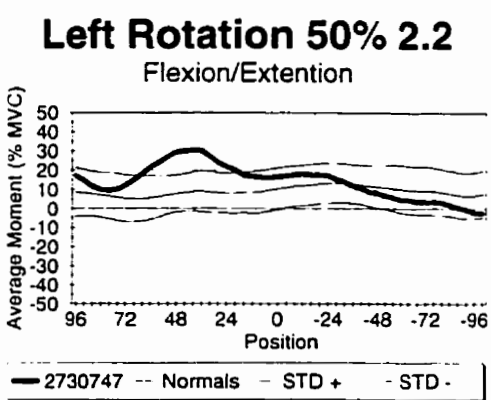
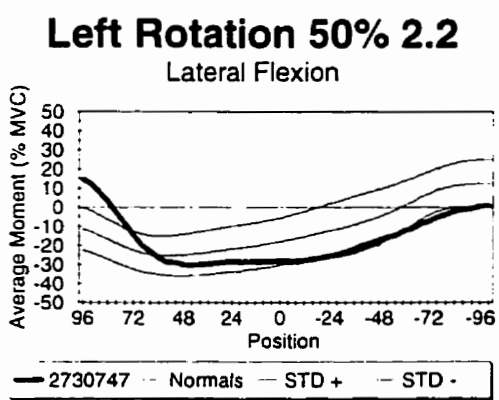
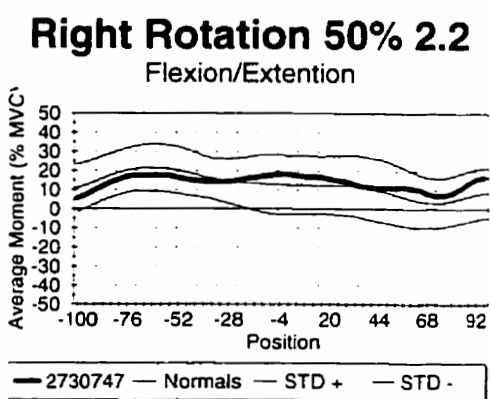
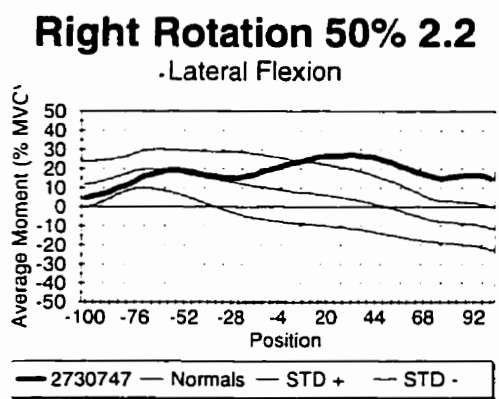
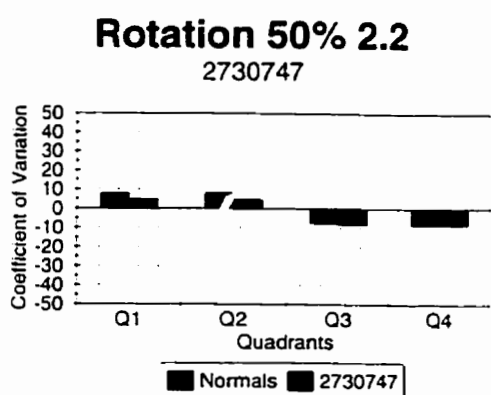
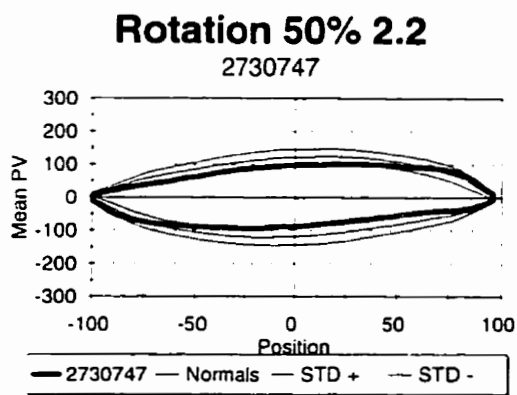
**Figure F11 B-200** summary performance for Case Study #4 (Bad day), for dynamic rotation at 50% resistance, day 1, test sequence #1.



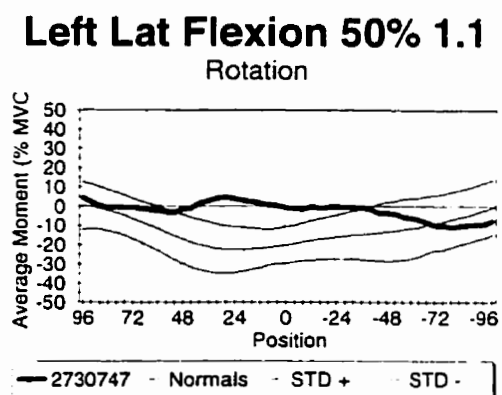
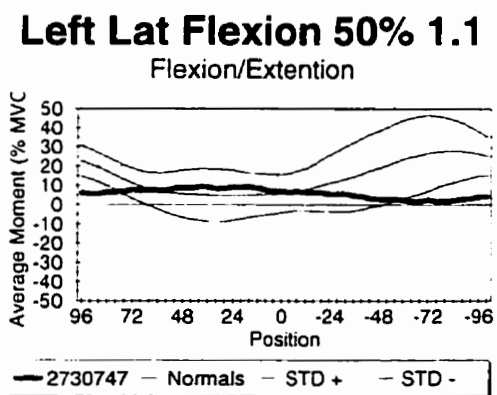
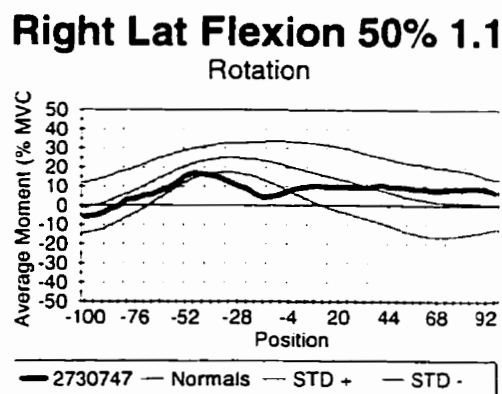
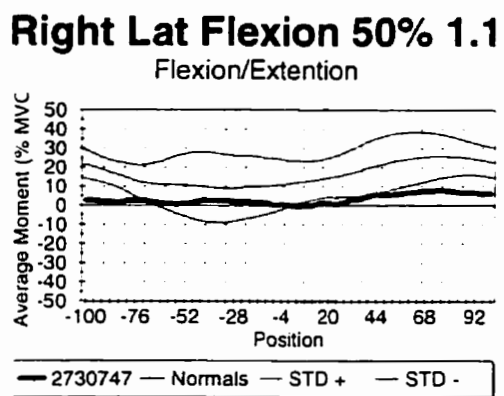
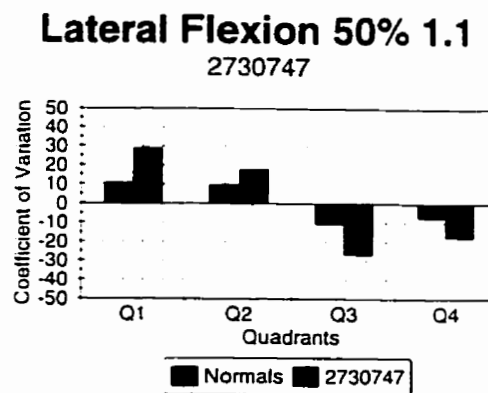
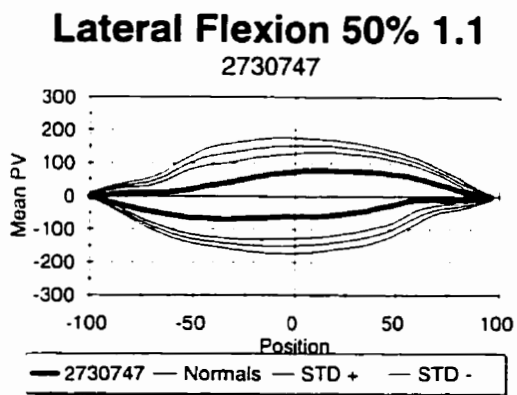
**Figure F12** B-200 summary performance for Case Study #4 (Good day), for dynamic rotation at 50% resistance, day 2, test sequence #1.



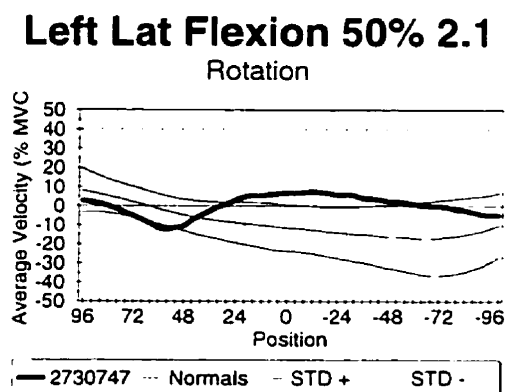
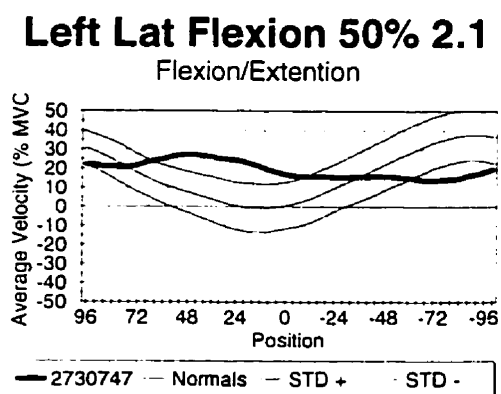
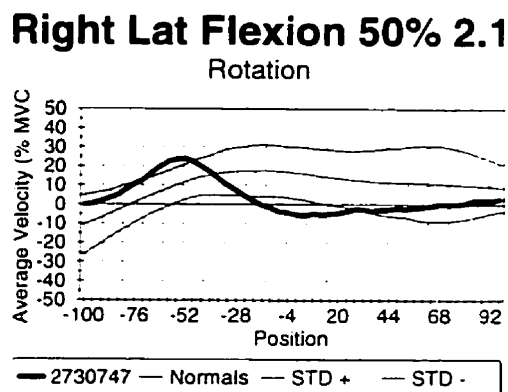
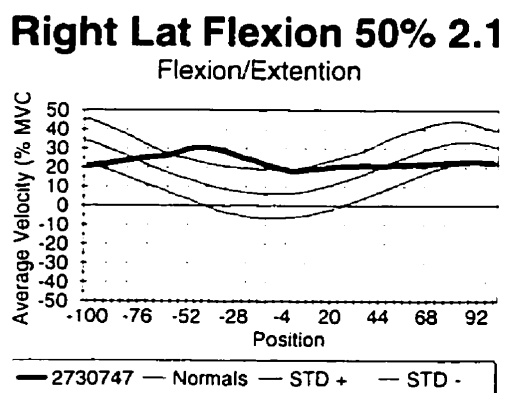
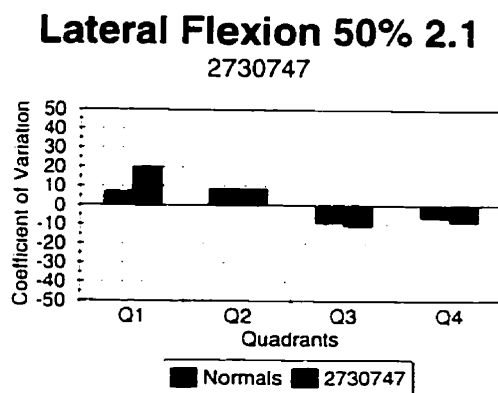
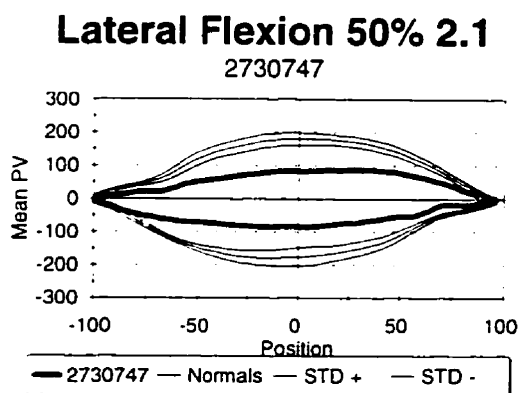
**Figure F13** B-200 summary performance for Case Study #4 (Bad day), for dynamic rotation at 50% resistance, day 1, test sequence #2.



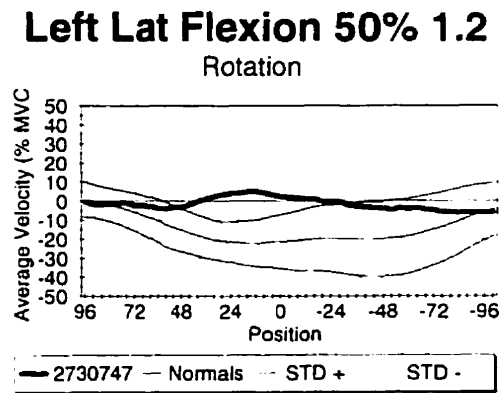
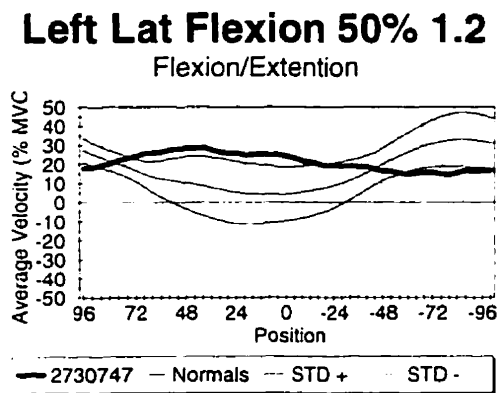
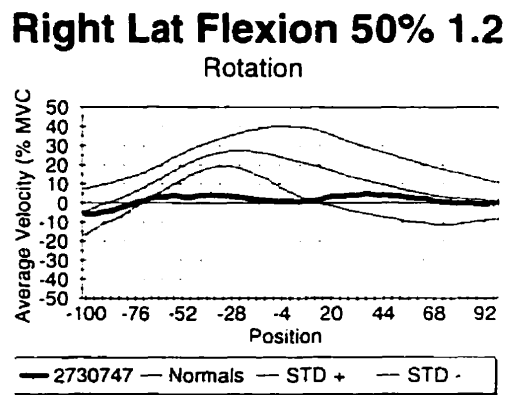
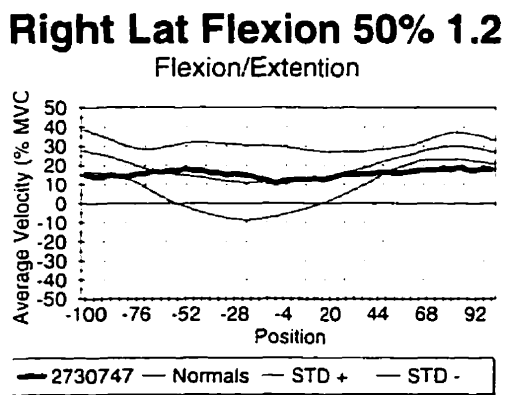
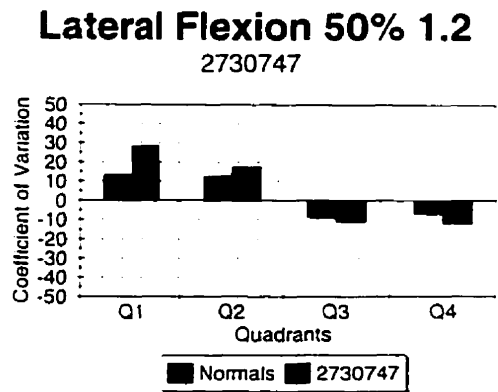
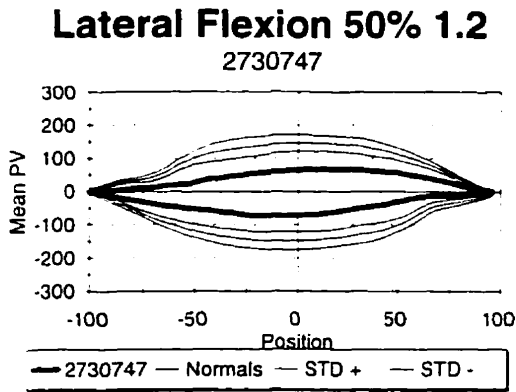
**Figure F14 B-200** summary performance for Case Study #4 (Good day), for dynamic rotation at 50% resistance, day 2, test sequence #2.



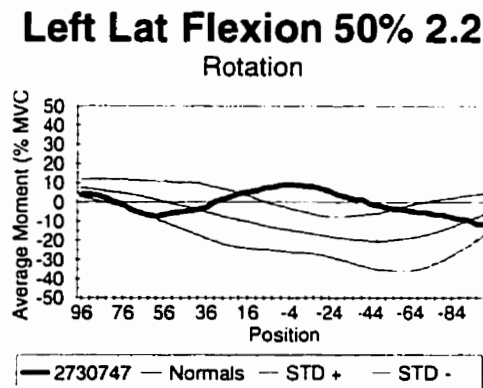
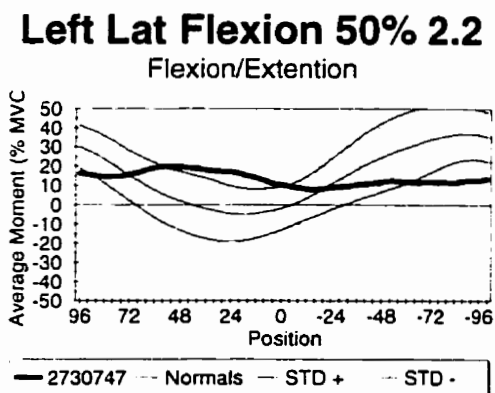
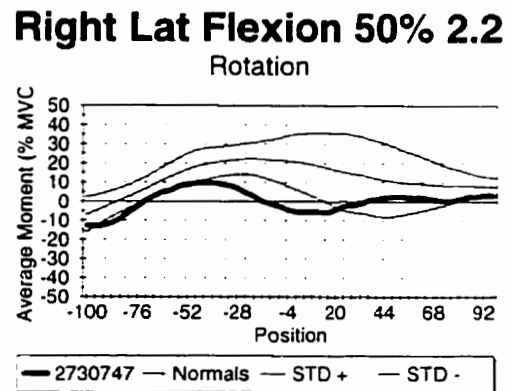
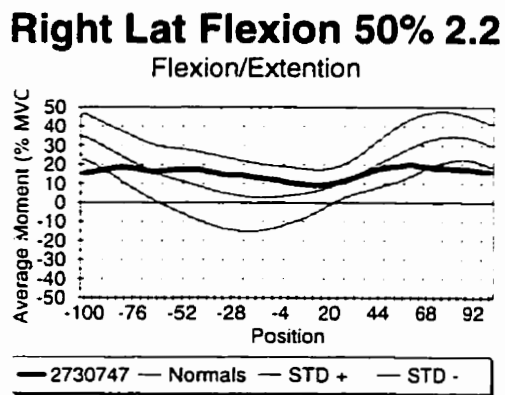
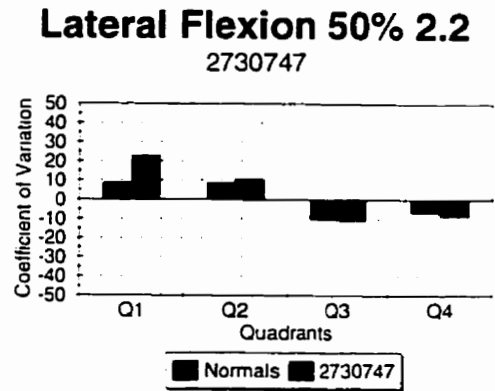
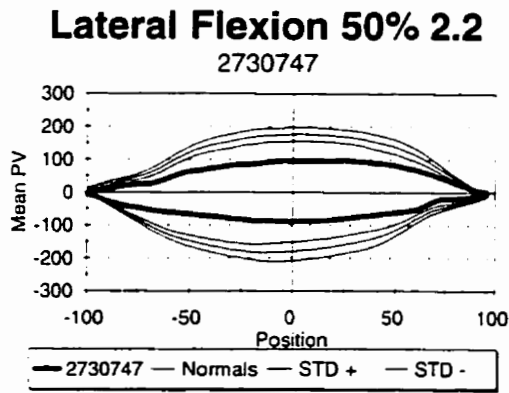
**Figure F15** B-200 summary performance for Case Study #4 (Bad day), for dynamic lateral flexion at 50% resistance, day 1, test sequence #1.



**Figure F16** B-200 summary performance for Case Study #4 (Good day), for dynamic lateral flexion at 50% resistance, day 2, test sequence #1.

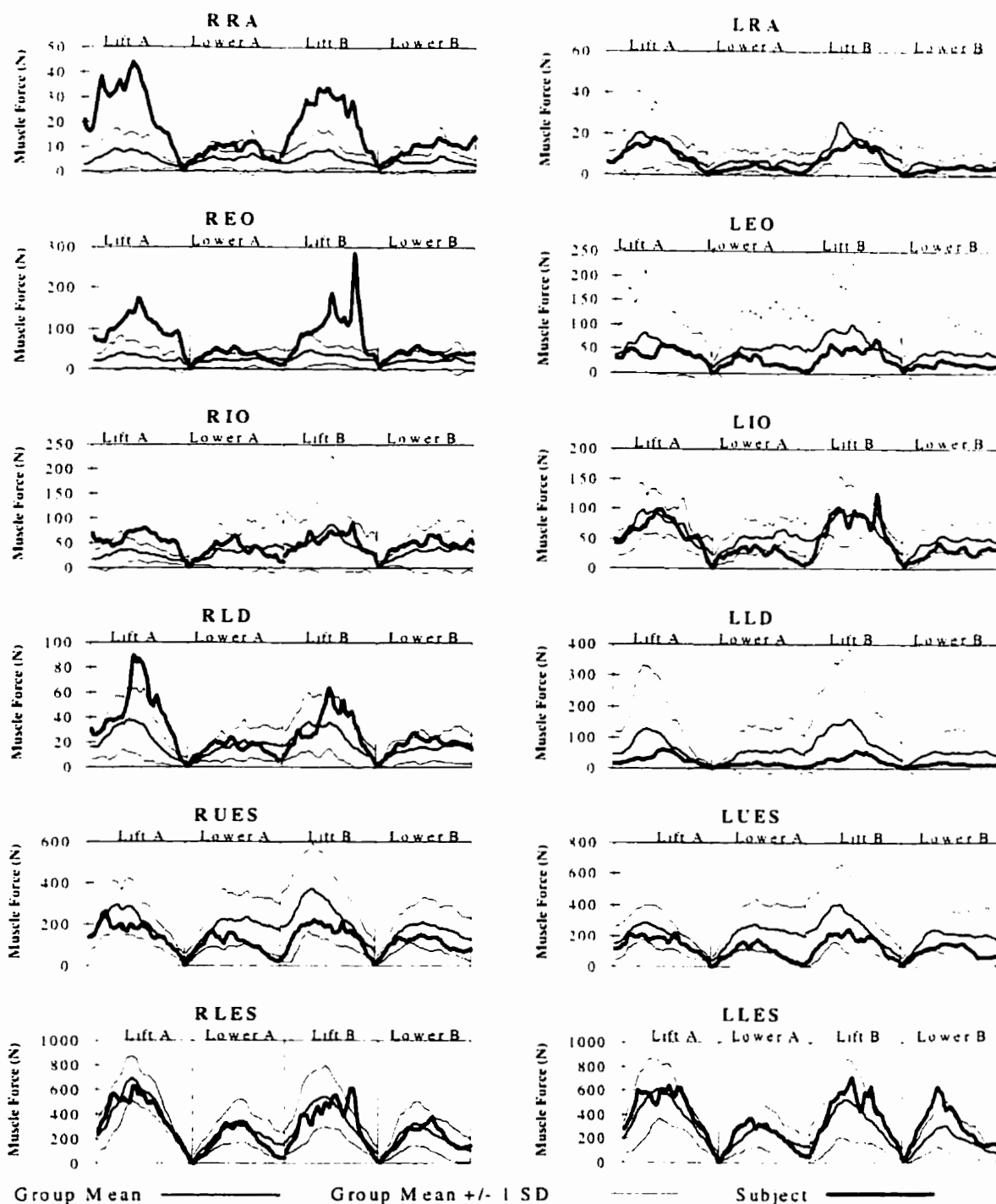


**Figure F17 B-200** summary performance for Case Study #4 (Bad day), for dynamic lateral flexion at 50% resistance, day 1, test sequence #2.

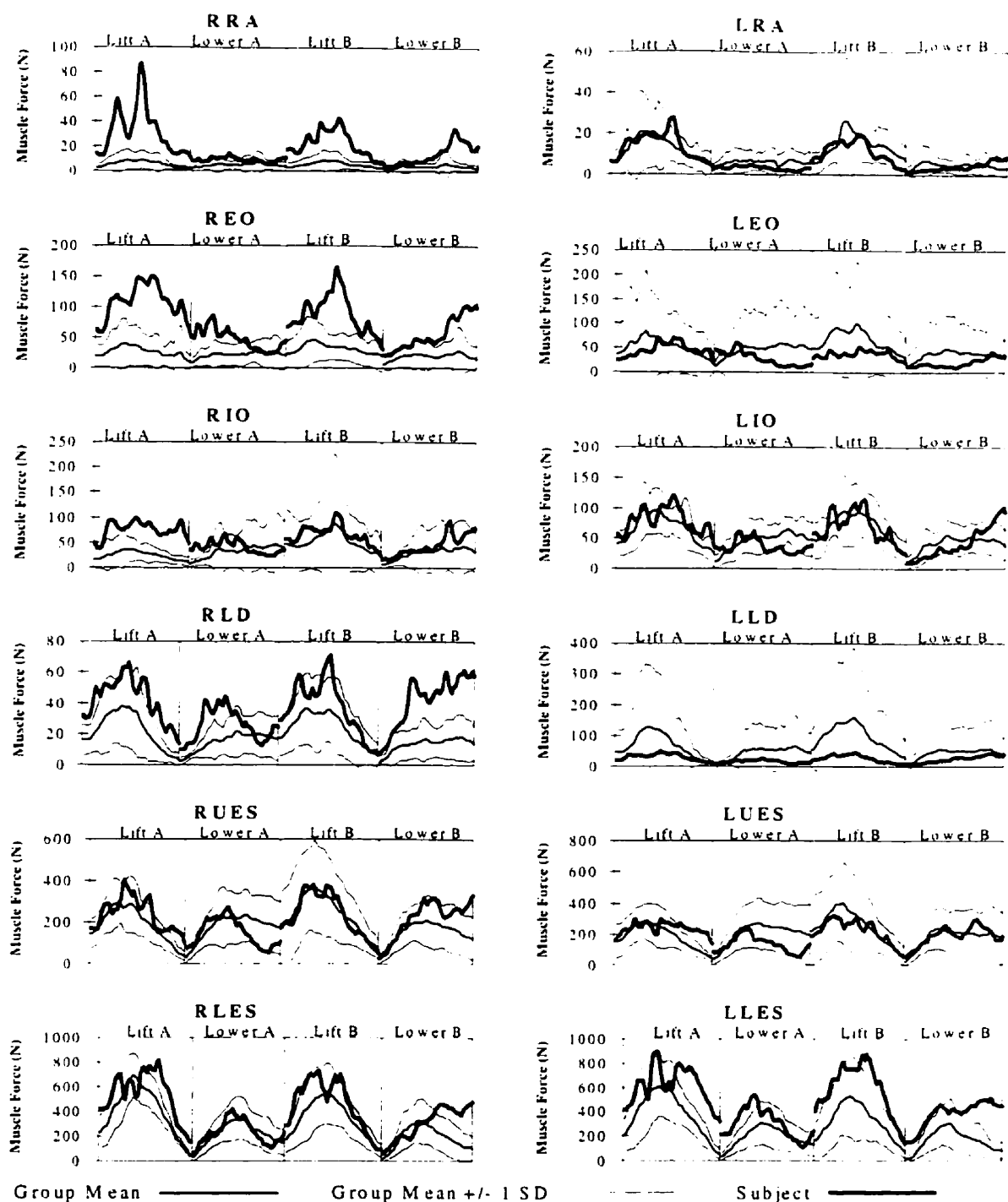


**Figure F18 B-200** summary performance for Case Study #4 (Good day), for dynamic lateral flexion at 50% resistance, day 2, test sequence #2.

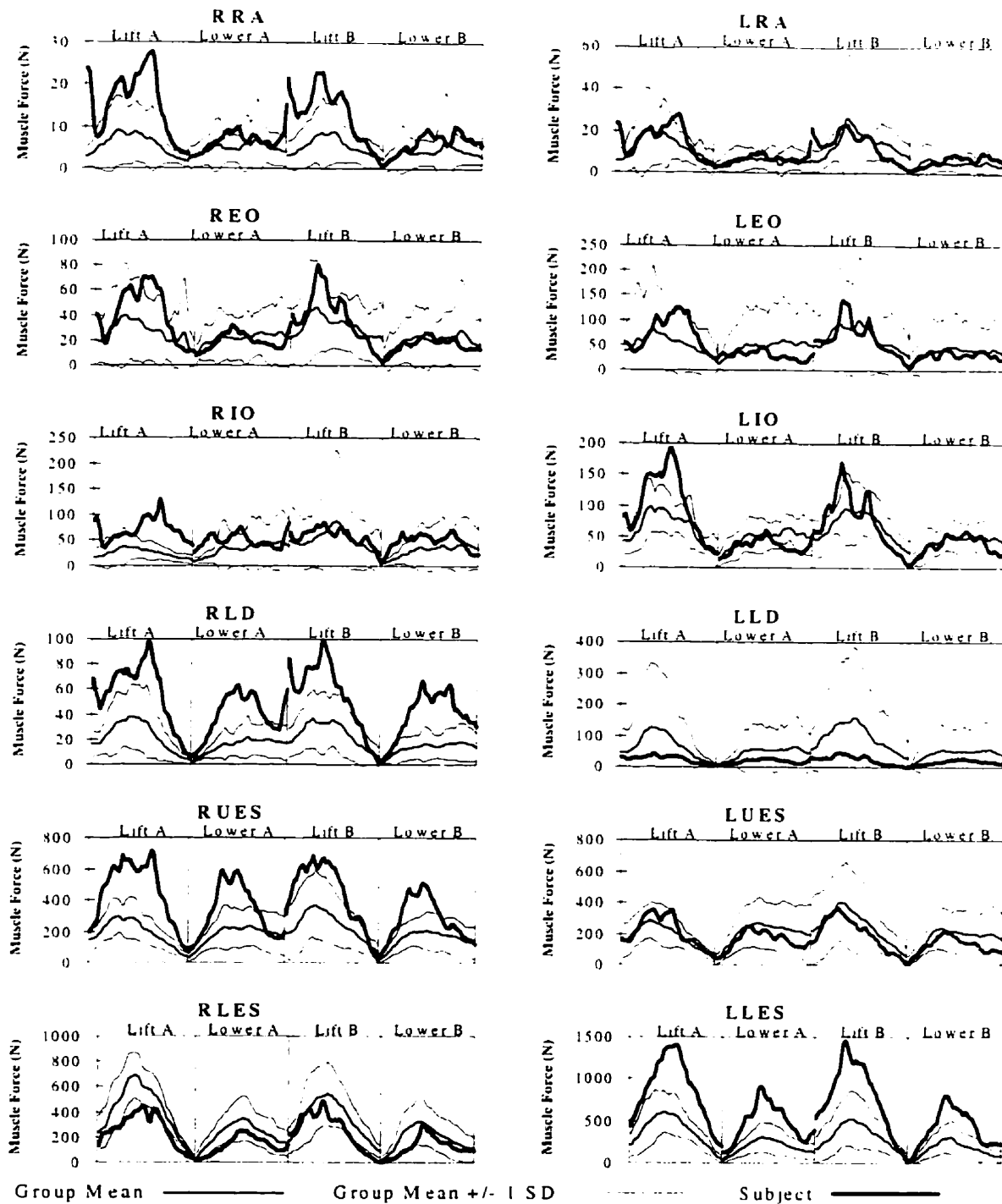




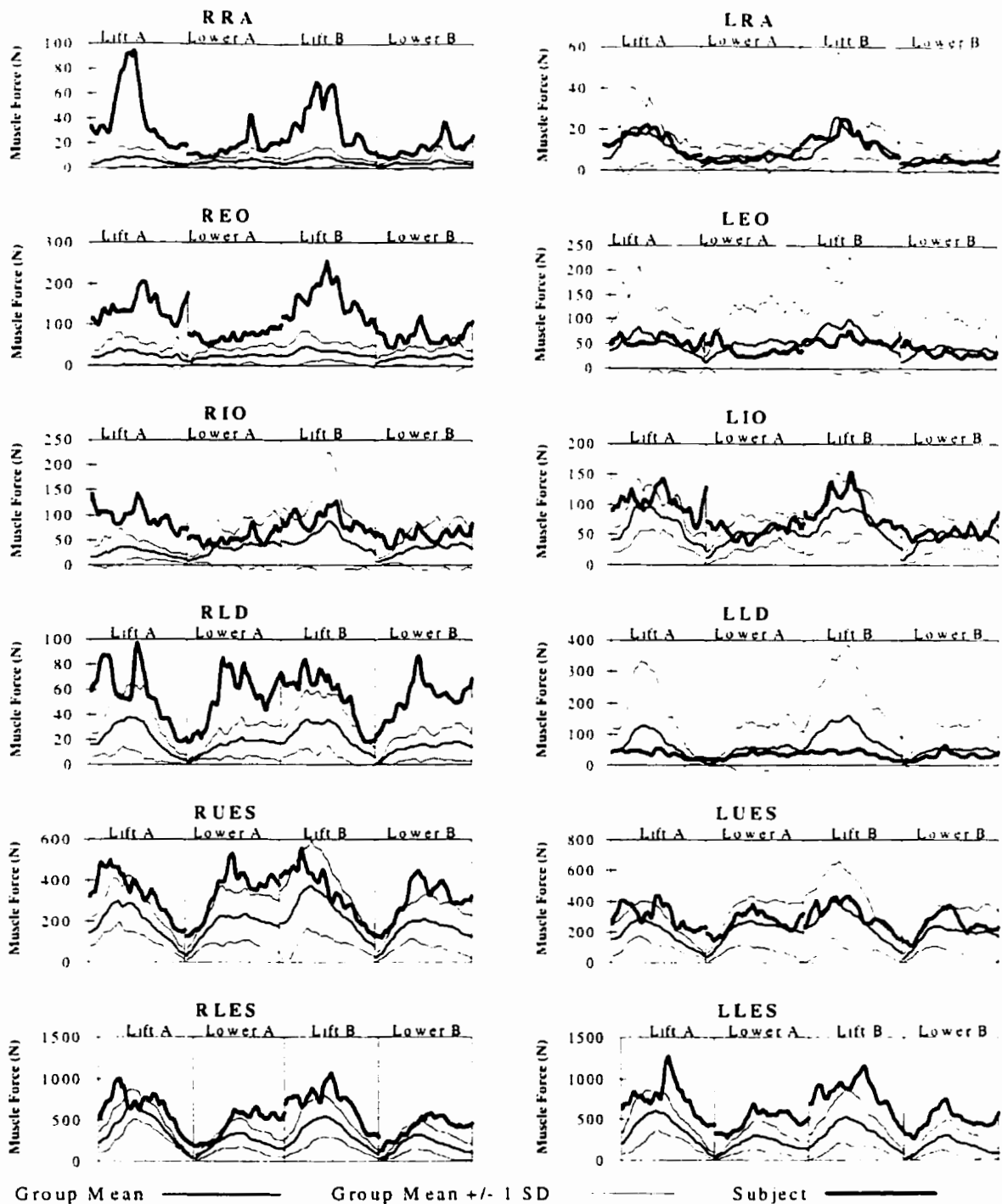
**Figure F19** Case Study #4 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) muscle force (N) for Day 1, 0 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L = left, RA = rectus abdominis, EO = external oblique. IO = internal oblique, LD = latissimus dorsi. UES = upper erector spinae, LES = lower erector spinae. Note: Day 1 = Bad Day.



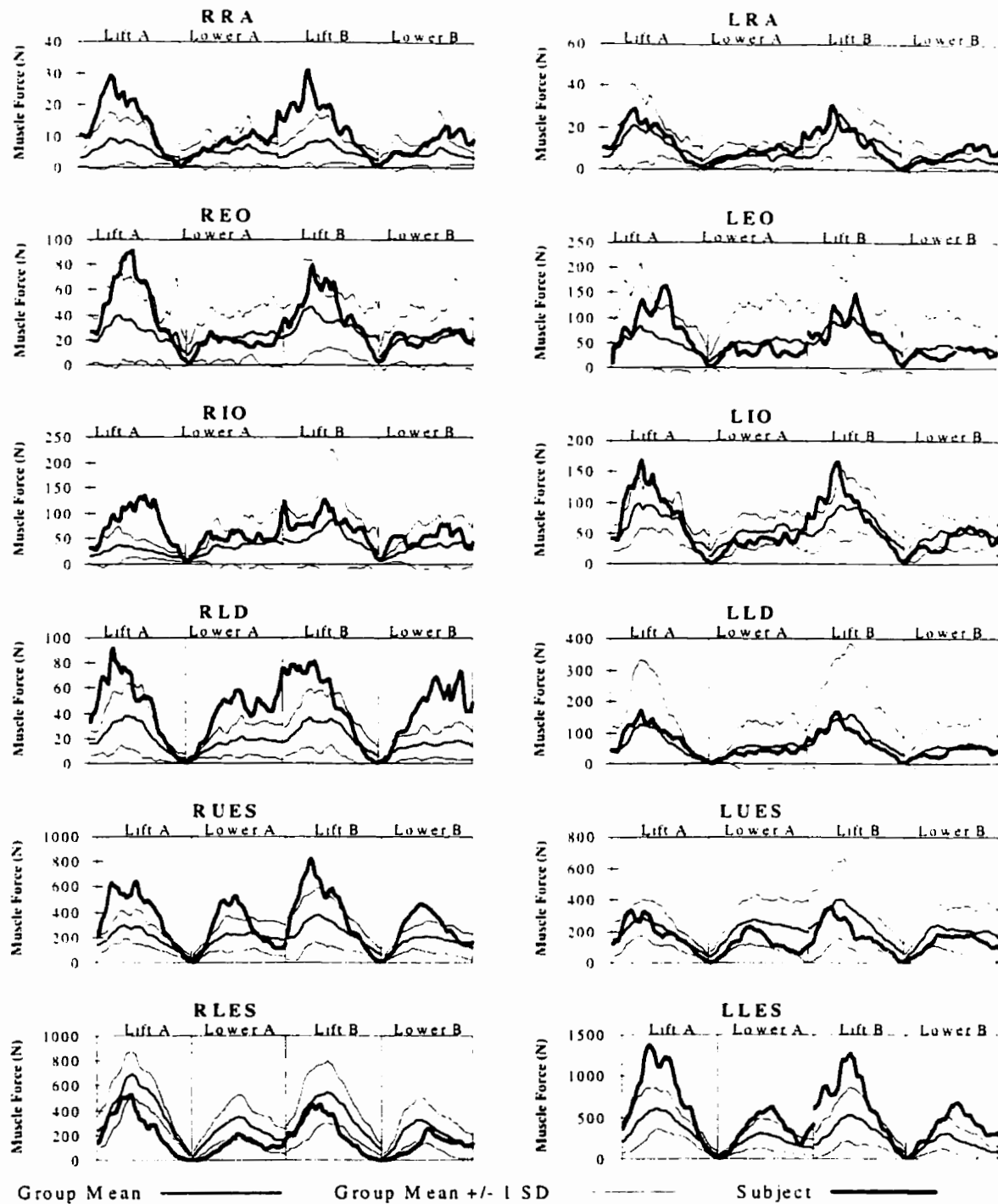
**Figure F20** Case Study #4 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) muscle force (N) for Day 1, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA= rectus abdominis, EO= external oblique, IO= internal oblique, LD= latissimus dorsi, UES= upper erector spinae, LES= lower erector spinae. Note: Day 1 = Bad Day.



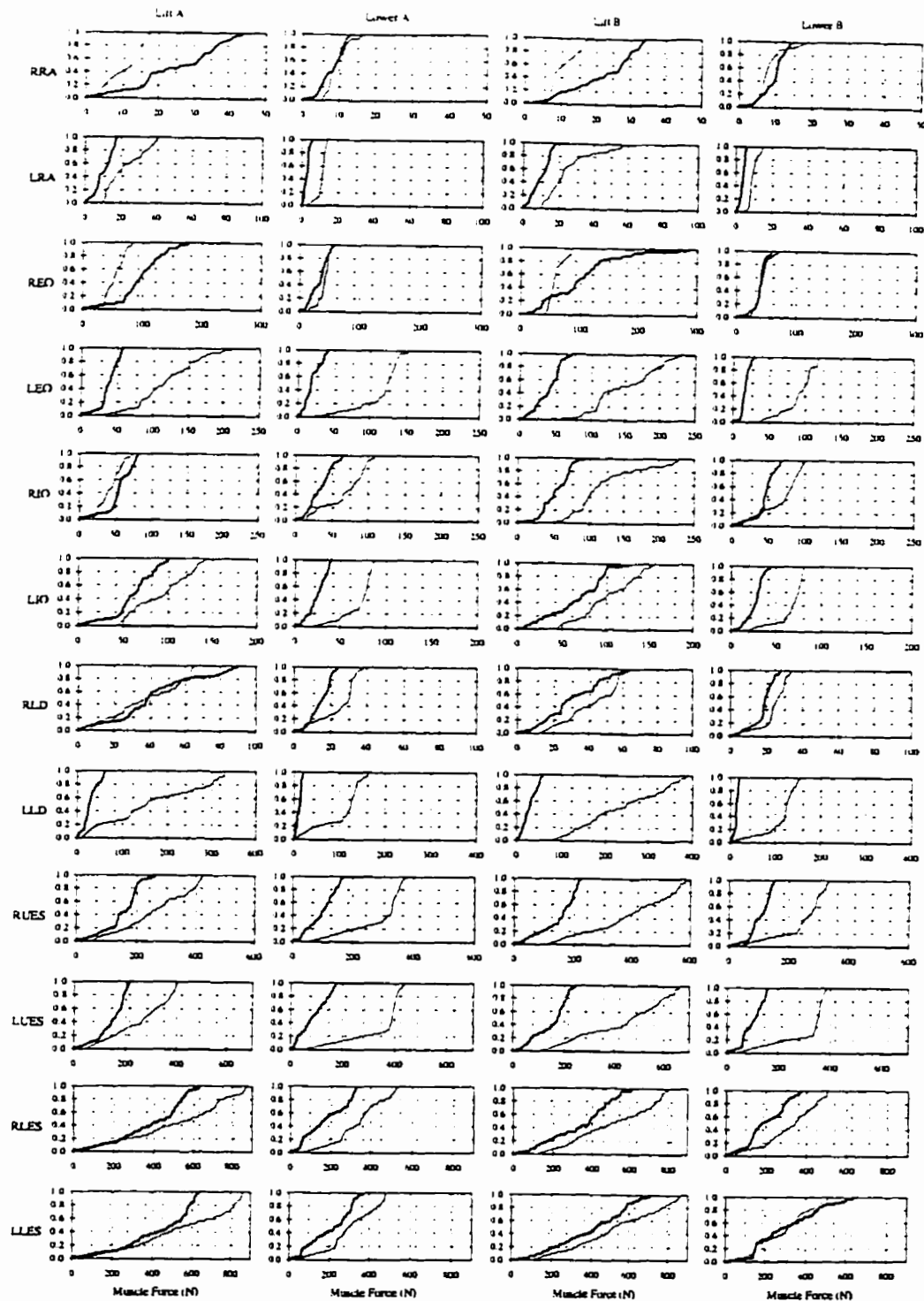
**Figure F21** Case Study #4 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (9 subjects) muscle force (N) for Day 2, 5 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R = right, L = left, RA = rectus abdominis, EO = external oblique, IO = internal oblique, LD = latissimus dorsi, UES = upper erector spinae, LES = lower erector spinae. Note: Day 2 = Good Day.



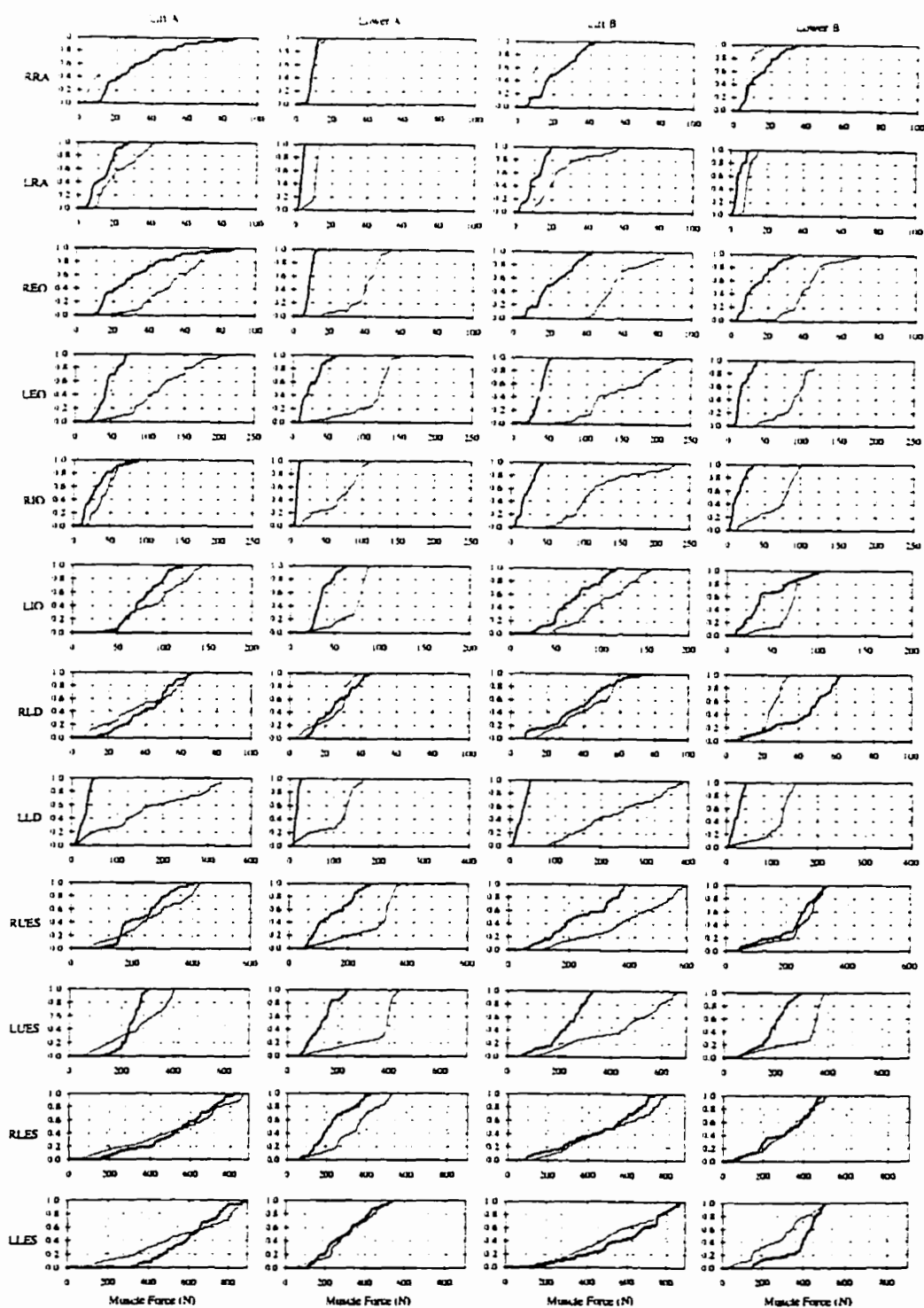
**Figure F22** Case Study #4 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (8 subjects) muscle force (N) for Day 1, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R=right, L=left, RA=rectus abdominis, EO=external oblique, IO=internal oblique, LD=latissimus dorsi, UES=upper erector spinae, LES=lower erector spinae. Note: Day 1 = Bad Day.



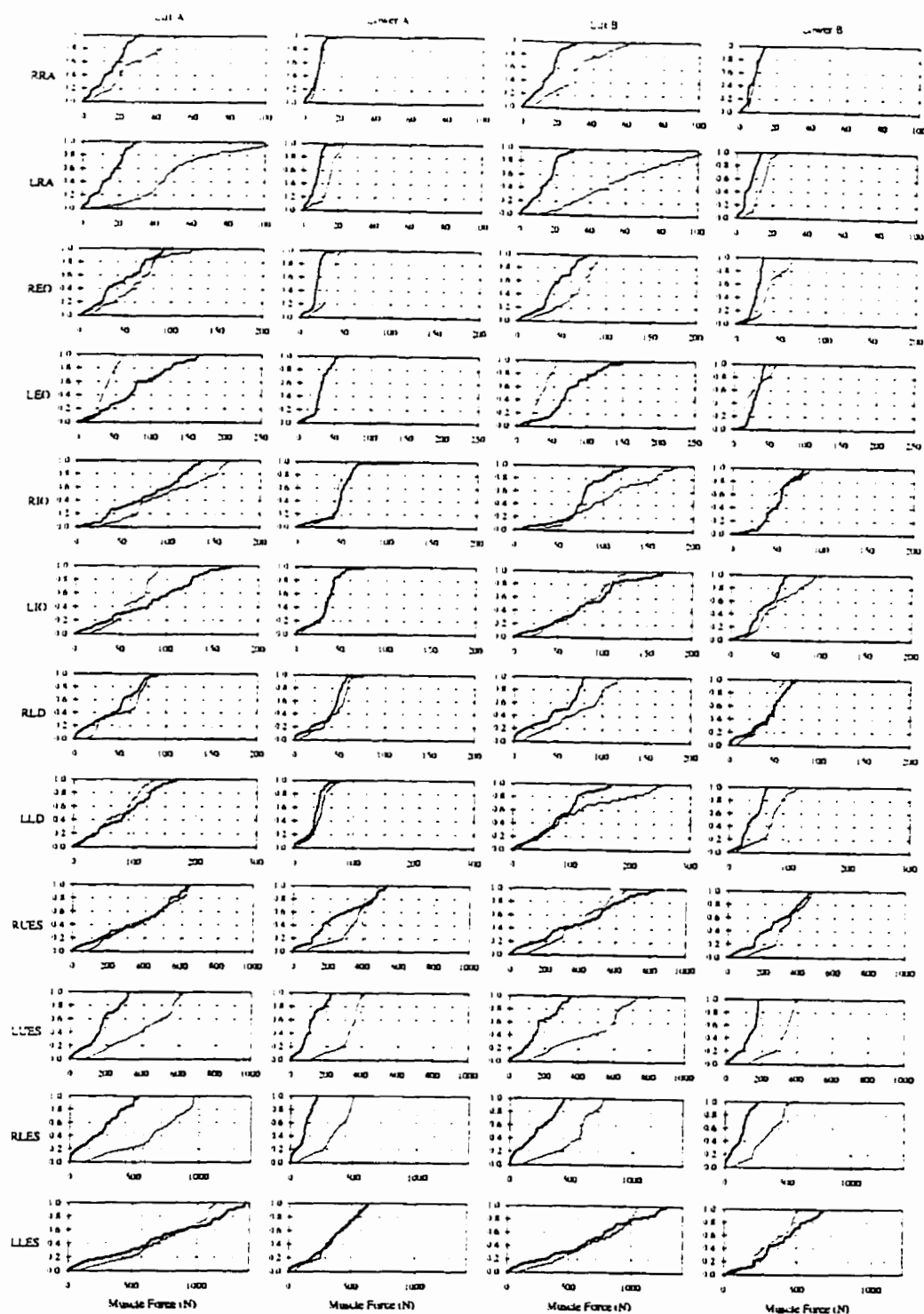
**Figure F23** Case Study #4 graphed against the summary profile of the mean ( $\pm 1$  SD) ASYMP (8 subjects) muscle force (N) for Day 2, 10 kg Load. Each graph identifies a specific electrode site. Within each graph the panels indicate specific lifts (A or B, extension) and lowers (A or B, flexion). Each panel is normalized from the start to the end of the lift/lower. Legend: R= right, L= left, RA= rectus abdominis, EO= external oblique, IO= internal oblique, LD= latissimus dorsi, UES= upper erector spinae, LES= lower erector spinae. Note: Day 2 = Good Day.



**Figure F24** Summary APDF profile of the muscle forces (N) by each electrode site for Day 1, 0 kg load and Case Study #4. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 9$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 1 = Bad Day.

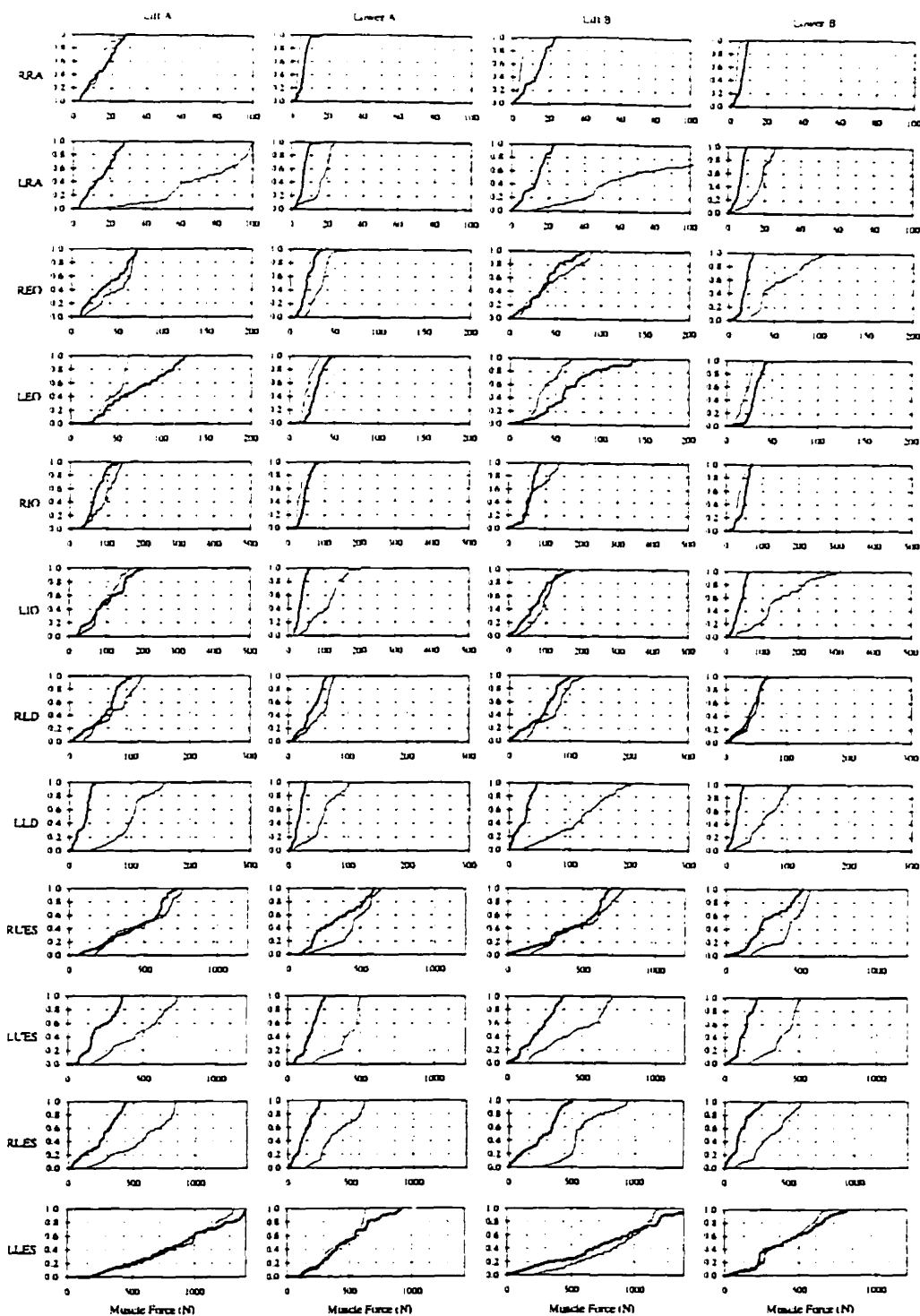


**Figure F25** Summary APDF profile of the muscle forces (N) by each electrode site for Day 1, 5 kg load and Case Study #4. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 9$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 1 = Bad Day.

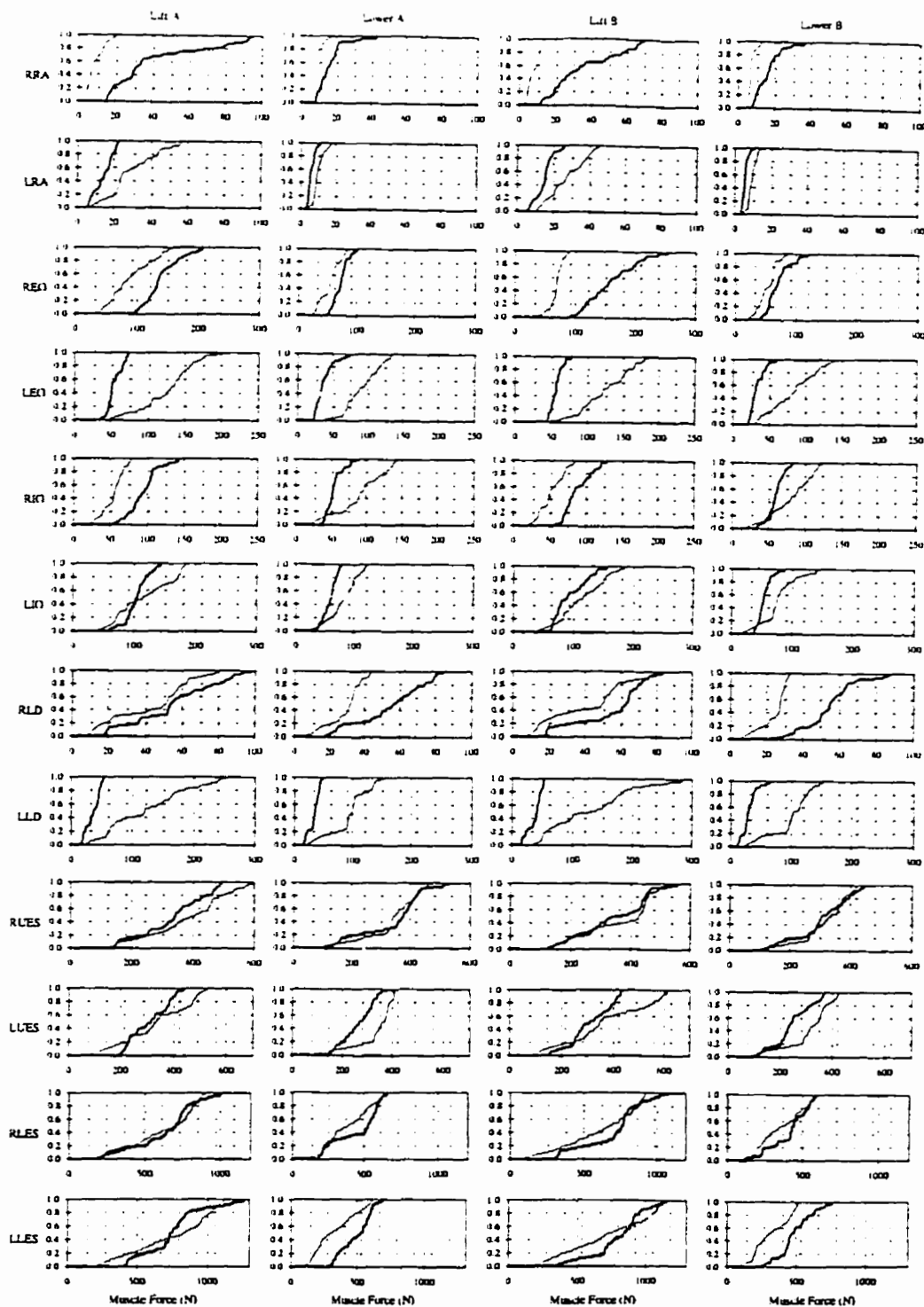


**Figure F26** Summary APDF profile of the muscle forces (N) by each electrode site for Day 2, 5 kg load and Case Study #4. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 9$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 2 = Good Day.





**Figure F27** Summary APDF profile of the muscle forces (N) by each electrode site for Day 1, 10 kg load and Case Study #4. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 8$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 1 = Bad Day.



**Figure F28** Summary APDF profile of the muscle forces (N) by each electrode site for Day 2. 10 kg load and Case Study #4. The thin line represents the upper boundary (mean + 1 SD) muscle force (N) of the ASYMP ( $n = 8$  subjects) group. The thick line represents the case study. Each figure represents a specific electrode site and task activity (i.e. lifting or lowering). The ordinate for each figure is Probability. Note: Day 2 = Good Day.

OOC Evaluation Results for

#2730747  
17-NOV-94

Demographic Data

Resistance Settings

Age : 40	Sex : M	Rotation 25% : 22 N-m
Height : 177.8 cm	Weight : 40.4 kg	Rotation 50% : 43 N-m
Diagnosis :		Flex/Ext 25% : 34 N-m
Surgical Category :		Flex/Ext 50% : 68 N-m
Activity Level Category :		Lat Flex 25% : 42 N-m
		Lat Flex 50% : 94 N-m

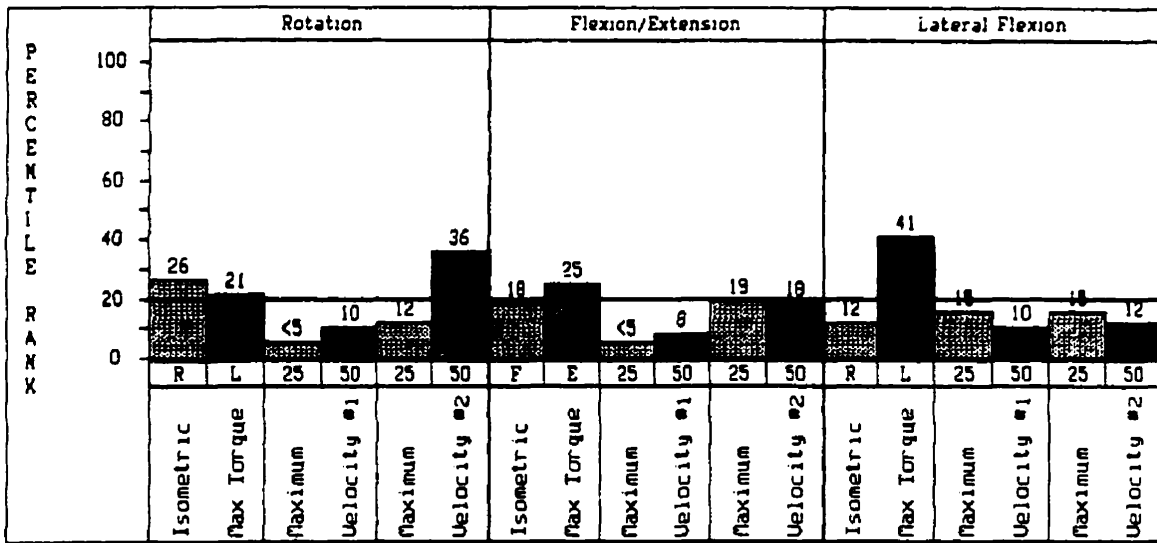
Abnormal Indicators 8

	Rotation		Flex/Ext		Lat Flex	
	25%	50%	25%	50%	25%	50%
Isometric Max Torque						
Max Velocity						
Rotation Sec Max Torque						
Flex/Ext Sec Max Torque						
Lat Flex Sec Max Torque						

Non-physiological Indicators 6

- 1) not observed
- 2) not observed
- 3) not observed
- 4) not observed
- 5) not observed
- 6) not observed

Baseline Rehabilitation Data



Test Administered By : Kardy Frazer      Signed : \_\_\_\_\_      Date : \_\_\_\_\_

Figure F29 Isostaion B-200 report (page 1) for Case Study #4 on Day 1 (Bad Day).

## OOC Evaluation Results for

#2730747  
17-KOV-94

OOC Test	Rotation	Flex/Ext	Lat Flex
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## Range of Motion

ROM (deg) #1	82.1	79.1	62.1
#2	88.0	85.4	78.8
ROM #2 (Δ%)	7.19	7.96	26.89†
ROM #1			

## Isometric

Max Torque (N-m)	97.4	135.3	206.1	168.0
------------------	------	-------	-------	-------

## Dynamic

		25%	50%	25%	50%	25%	50%
Resisted ROM (Δ%)	#1	8.56	6.76	4.24	4.24	19.72	7.75
	#2	7.14	6.30	5.06	-5.62	0.56	-17.22
Avg Vel 50% (Δ%)	#1	-4.17		6.19		-23.77	
	#2	-23.89		-35.92		-45.32	
Max Velocity (deg/sec)	#1	75.1‡	85.0	105.6‡	116.4	107.7	89.8
	#2	118.6	116.6	143.5	124.6	118.0	82.1

## Secondary Axes

Rot Secondary	#1	--	--	10.7	25.6	19.2	36.2
Max Torq (N-m)	#2	--	--	14.9	17.1	23.5	19.2‡
F/E Secondary	#1	21.5‡	21.5‡	--	--	21.5‡	21.5‡
Max Torq (N-m)	#2	40.0	36.9	--	--	36.9	61.5
L F Secondary	#1	30.1‡	43.1‡	8.6	21.5	--	--
Max Torq (N-m)	#2	51.7	71.1	25.8	25.8	--	--

## Key

‡ Subnormal, less than critical level  
 † Supernormal, greater than critical level  
 (see Statistical Review in OOC Back Evaluation System)

Version 3.0

Figure F30 Isostaion B-200 report (page 2) for Case Study #4 on Day 1 (Bad Day).

OOC Evaluation Results for

#2730747  
27-JAN-95

Demographic Data

Age : 40	Sex : M
Height : 177.8 cm	Weight : 40.4 kg
Diagnosis :	
Surgical Category :	
Activity Level Category :	

Resistance Settings

Rotation 25%	: 24 N-m
Rotation 50%	: 47 N-m
Flex/Ext 25%	: 46 N-m
Flex/Ext 50%	: 92 N-m
Lat Flex 25%	: 42 N-m
Lat Flex 50%	: 83 N-m

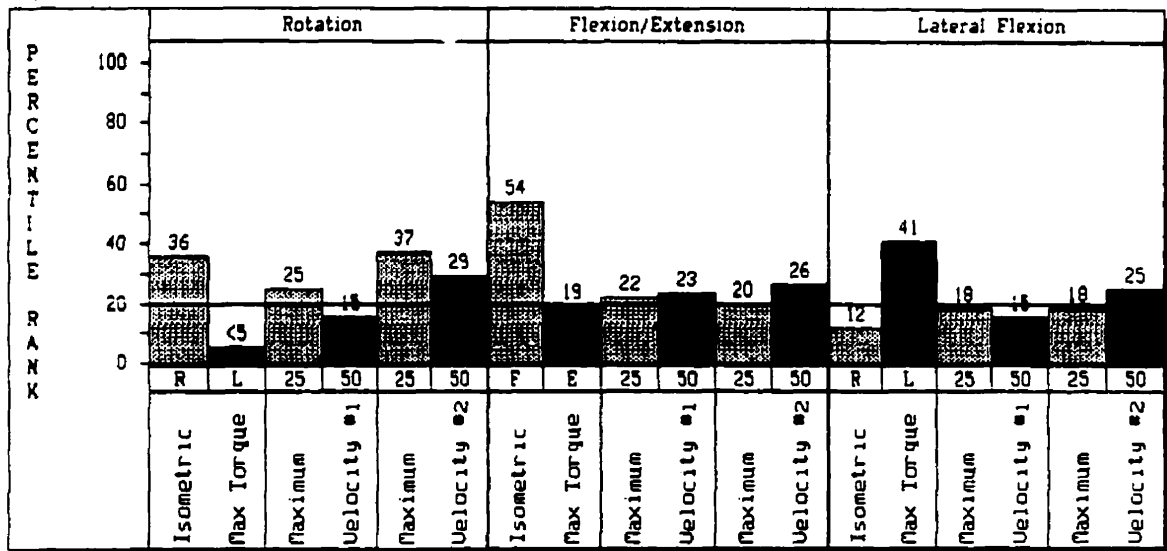
Abnormal Indicators 0

	Rotation		Flex/Ext		Lat Flex	
	25%	50%	25%	50%	25%	50%
Isometric Max Torque						
Max Velocity						
Rotation Sec Max Torque						
Flex/Ext Sec Max Torque						
Lat Flex Sec Max Torque						

Non-physiological Indicators 0

- 1) not observed
- 2) not observed
- 3) not observed
- 4) not observed
- 5) not observed
- 6) not observed

Baseline Rehabilitation Data



Test Administered By : Kardy Frazer      Signed : \_\_\_\_\_      Date : \_\_\_\_\_

Figure F31 Isostaion B-200 report (page 1) for Case Study #4 on Day 2 (Good Day).

## OOC Evaluation Results for

#2730747  
27-JAN-95

OOC Test	Rotation	Flex/Ext	Lat Flex
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## Range of Motion

ROM (deg) #1	89.5	88.7	77.0
#2	90.3	87.7	74.4
ROM #2 (Δ%)	0.89	-1.13	-3.38
ROM #1			

## Isometric

Max Torque (N-m)	96.0	184.5	187.6	165.8
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## Dynamic

		25%	50%	25%	50%	25%	50%
Resisted ROM (Δ%)	#1	5.37	3.72	7.03	2.16	0.00	-7.95
	#2	4.51	4.10	10.38	-3.83	14.12	-2.35
Avg Vel 50% (Δ%)	#1	-19.50		5.54		-15.08	
	#2	-26.19		-27.68		-21.84	
Max Velocity (deg/sec)	#1	130.5	100.8	138.1	138.1	115.4	100.0
	#2	138.4	110.7	148.9	138.1	130.8	107.7

## Secondary Axes

Rot Secondary	#1	--	--	23.5	12.8	29.9	38.4
Max Torq (N-m)	#2	--	--	19.2	17.1	34.1	29.9
F/E Secondary	#1	70.7	120.0	--	--	52.3	86.1
Max Torq (N-m)	#2	64.6	98.4	--	--	33.8	58.4
L F Secondary	#1	71.1	73.2	12.9	12.9	--	--
Max Torq (N-m)	#2	77.5	73.2	15.1	10.8	--	--

## Key

† Subnormal, less than critical level  
 ‡ Supernormal, greater than critical level  
 (see Statistical Review in OOC Back Evaluation System)

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Figure F32 Isostaion B-200 report (page 2) for Case Study #4 on Day 2 (Good Day).