DETECTION, PREVALENCE, AND CHARACTERISTICS OF BREAKAWAY WEAKNESS

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Sheila Schindler-Ivens
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APPROVAL

NAME: Sheila Schindler-Ivens

DEGREE: Master of Science

TITLE OF THESIS: Detection, Prevalence, and Characteristics of Breakaway Weakness

EXAMINING COMMITTEE:

Chair: Dr. D. Goodman

Dr. J.A. Hoffer
Senior Supervisor
Professor and Director
School of Kinesiology, S.F.U.

Dr. T. Milner
Associate Professor
School of Kinesiology, S.F.U.

Dr. R. Mathias
Professor, Department of Health Care and
Epidemiology, U.B.C.

Dr. T. Richardson
Associate Professor
School of Kinesiology, S.F.U.
External Examiner

Date Approved: 23 October 1996

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Detection, Prevalence, and Characteristics of Breakaway Weakness

Author.
(signature)

Sheila Schindler-Ivens
(name)

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ABSTRACT

This study was prompted by recent reports by an orthopedic surgeon who observed a number of patients with a peculiar muscle weakness called breakaway weakness (BAW) that had previously gone undetected. There appeared to be a pattern of similarities among individuals who manifest the weakness, including the observation that patients typically reside in the Lower Fraser Valley of British Columbia (LFV) and have musculoskeletal disorders of the extremities or spine.

BAW cannot be detected with the standard testing techniques that are routinely used in clinical evaluation. This physician developed a manual test to evaluate patients for BAW, whereby the muscles in question are fully activated and then challenged with a rapid, brief increase in load. A previous study (Archibald and Mathias, 1991) found that, although the manual test was neither quantitative nor reliable, the phenomenon itself deserved further study.

In this research, we 1. developed a novel and repeatable mechanical test for BAW, 2. developed a method to classify subjects as normal or BAW, 3. assessed the reliability of the test for producing consistent diagnoses on two test sessions, 4. estimated the prevalence of BAW in individuals with and without injuries, living within and outside the LFV, 5. described the characteristics of the electromyogram (EMG), torque, and position that were associated with

BAW, and 6. estimated the latency of BAW. A total of 71 subjects were tested at least one time over the course of 4 phases of experimentation.

BAW was found to be characterized by a dramatic decrease in pronation torque and pronator teres (PT) EMG. It was detected at least once in 69% of all subjects tested. The test/re-test reliability was poor, largely attributable to the sporadic occurrence of BAW. There was no evidence that BAW in PT was associated with musculoskeletal injuries in the lower limbs or back in subjects living outside the LFV. Injured subjects from the LFV were significantly more likely to display BAW on two consecutive test sessions than subjects from outside the LFV.

Our results showed that BAW was associated with reduced activation of PT and biceps (BI) EMG. However, mechanisms remain unclear. The short latency stretch reflex to PT and reciprocal inhibition to BI were normal in subjects with BAW. Possible mechanisms involved in BAW include an inhibitory clasp-knife reflex and/or inadvertent voluntary shutdown of motor output.

DEDICATION

To Herman and Ludwina Schindler

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I extend a sincere thank you to all the subjects who allowed me to "twist their arm", especially those who traveled from the Lower Fraser Valley. Furthermore, I would like to thank Dr. Richard Sweeting for bringing this issue to our attention and for referring dozens of patients over the past three years.

Finally, I would like to thank my family for supporting me in my endeavor the earn this degree and my husband Chris for believing in me and being my biggest fan.

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Introduction

This study was prompted by recent reports by an orthopedic surgeon that a population of individuals may display a unique form of muscle weakness, referred to as "breakaway weakness", that has previously gone undetected. The cause of this weakness is unknown, but it has been suggested that it may be associated with residence in the Lower Fraser Valley of British Columbia, and an increased risk for musculoskeletal injuries (Archibald and Mathias, 1991; Richard Sweeting, personal communication).

The type of individuals who have been suspected of having breakaway weakness (BAW) usually display a variety of signs and symptoms that may include joint pain, a feeling of limb weakness, knee buckling, dropping objects, recurrent ankle sprains, anterior knee pain, and whiplash injury. Physical examination by a physician usually reveals no muscle wasting, joint injury, or nerve conduction abnormalities. Patients' symptoms often do not fit into a recognized disease category (Archibald and Mathias, 1991) and do not respond well to treatments. Often, these problems are severe enough to prevent independence in activities of daily living, gainful employment, and participation in leisure activities. Patients are often young, active, athletic, and otherwise healthy.

It was the observation of Dr. Richard Sweeting, an orthopedic surgeon practicing in the Lower Fraser Valley (LFV), that individuals who have the previously mentioned signs and symptoms also share the common characteristic of BAW and that BAW may be associated with, if not responsible for, their high propensity to injury and pain. Sweeting hypothesized that this weakness was caused by an abnormality of the stretch reflex, or as he calls it, the "load compensation" reflex. He further theorized that people living in the LFV are affected by BAW because of exposure to environmental toxins in the air, food, and drinking water. This assumption is based on patient reports of possible exposure to toxins through food and water that may be contaminated and inhalation of agricultural sprays (herbicides and pesticides) that are used in the area. It is important to note that, in his medical practice, Sweeting mainly treats people who live in the LFV and has limited experience testing people from other geographic regions. Hence, there is no evidence that BAW is limited to the LFV.

Breakaway difficult to detect with traditional methods

It has been difficult to accurately diagnose people with BAW because this disorder is not detected with standard testing techniques that are routinely used in clinical evaluation. Rather, BAW only becomes apparent when an active muscle is challenged with a rapid increase in load or stretch. In traditional muscle testing, which is used to identify musculoskeletal disorders (Kendall and

McCreary, 1983; Bohannon, 1986; Miller *et al.*, 1988; Wadsworth *et al.*, 1987), the examiner evaluates strength by asking the subject to activate a muscle and then gradually applies external force that matches the subject's effort. The amount of force that the subject is able to produce determines muscle strength. An important principle in traditional muscle testing is that force must be applied **gradually**. Sudden application of force has been thought to result in an inaccurately low muscle grade because it may suddenly disrupt the pull of the muscle (Kendall and McCreary, 1983). However, this assumption may be inaccurate because a sudden stretch actually **increases** - not decreases - the muscle's ability to produce force. In this regard, active muscles behave like springs: when stretched quickly, a restoring force is generated that resists the stretch. This behavior is due to intrinsic elastic properties of muscles and is reinforced by the stretch reflex (Hoffer *et al.*, 1990; Hoffer and Andreassen, 1981; Nichols and Houk, 1976; Toft *et al.*, 1989). Traditional manual muscle testing techniques evaluate isometric strength but do not evaluate the spring-like properties of muscle. Therefore, traditional tests may not be capable of detecting subtle forms of weakness, particularly those characterized by the inability to resist sudden, unexpected loads.

Manual test for BAW

A manual test for BAW was introduced and is used by Sweeting as a diagnostic tool. The test is performed by asking the patient to activate a specific muscle with maximum effort while the examiner matches the patient's effort with his or her hand. (See Figure 0.1.) When the patient has achieved maximum effort, the examiner quickly and suddenly rotates the patient's limb in the direction opposite of the patient's effort to determine whether or not the patient is capable of resisting the examiner. In all cases, the immediate response is to "give" or "yield" slightly in response to the sudden muscle stretch. If after the initial yield, the patient is able to continue to resist the examiner, the response is considered normal. If the patient is overcome by the rotation and "gives out" or "breaks", the response is considered abnormal, and the patient is diagnosed with BAW. In some patients, BAW is revealed more easily by several test rotations performed in quick succession.

Sweeting reports that BAW is most frequently detected in the following muscle groups: neck flexors, shoulder external rotators, wrist pronators, hip abductors, and ankle evertors. He considers wrist pronation, specifically pronator teres (PT) BAW, to be a good overall indicator of BAW in other muscle groups. If BAW is present in PT, it is likely to be present in other muscles.

Conversely, if BAW is not present in PT, he reports that it is unlikely to be present in other muscle groups (Archibald and Mathias, 1991).

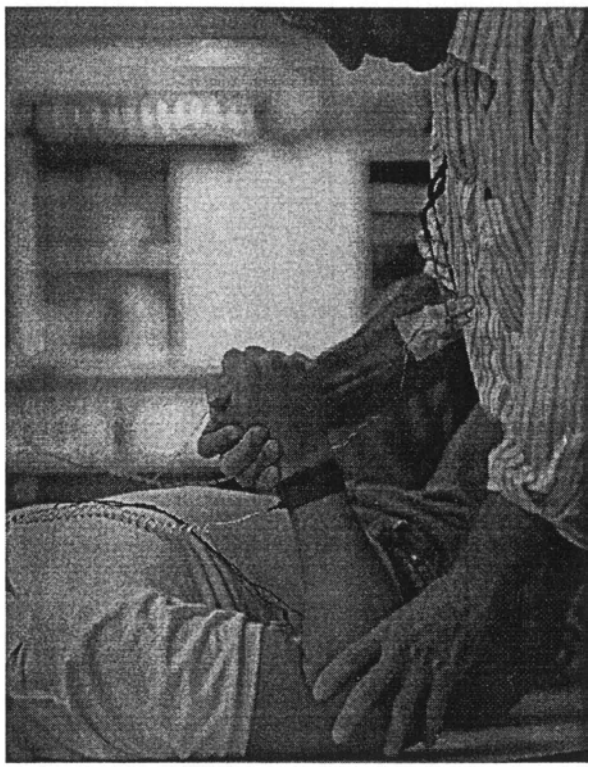

Figure 0.1. Manual Test for BAW. The examiner is testing the right PT for BAW. The subject activates PT maximally after which the examiner rotates the limb quickly in the opposite direction, overpowering the subject. This evaluation was done in the laboratory to examine the types of manual rotations that are typically performed. In the clinical setting EMG electrodes are not used, and the examiner relies on subjective "feel" to determine whether BAW is present.

Aside from Sweeting's observations and an unpublished study from the University of British Columbia (1991) that will be discussed in the next section, we are aware of only one other report of BAW. Baker (1986) found that patients who experienced eccentric, muscle injuries due to rapid deceleration from motor vehicle collisions displayed painful trigger points (i.e. areas of muscular hypersensitivity) and BAW. The muscles most likely to have pain and BAW

were those that "braked" most forcibly during the impact of the collision. These muscles were otherwise very strong and, when tested with a traditional manual muscle test, were given a normal strength grade of 5/5. Baker proposed that a new classification of $5_b/5$ be employed to identify muscles that are otherwise strong but unable to resist rapid increases in load due to pain. Baker's observations differed from Sweeting's in that Baker suggested that BAW was due to muscle pain and injury, whereas Sweeting proposed that BAW leads to muscle injury and pain.

Evaluation of manual test

In an unpublished study conducted by Drs. C. Archibald and R. Mathias at the University of British Columbia (1991) and reported to the British Columbia Ministry of Health, the intra- and inter-tester reliability of Sweeting's test for BAW were evaluated. Sweeting and two physiotherapists, who he trained in the testing technique, blindly tested 78 subjects for presence or absence of BAW. Among the subjects were 21 confirmed cases (as per Sweeting's diagnosis), 16 subjects who were on a waiting list to see Sweeting, and 42 subjects from the general LFV community. Results of the study indicated that the test had only fair intra-observer reliability (Kappa=0.48), poor inter-observer reliability (Kappa=0.13), and was unable to differentiate between people who have BAW and people from the general community. We feel that this study took an appropriate approach to the issue under investigation.

Because they found problems with the reproducibility of the clinical test for BAW, these investigators were unable to proceed with further hypothesis testing. Despite this fact, these investigators also concluded that Sweeting was an unusually keen observer and that the issue of BAW deserved further study.

In addition to these observations, other shortcomings of the manual test for BAW can been identified. It relies on the subjective "feel" of the examiner to determine whether or not BAW is present. The procedure is difficult for other examiners to learn, making it hard to confirm or refute Sweeting's observations. It is impossible to ensure that the examiner is not biasing the result by inadvertently cueing the subject verbally or non-verbally to make a particular response more likely. It provides no method for quantifying the severity of BAW, or monitoring improvement or regression of the condition.

Possible mechanism of BAW

In our view, the sudden collapse in muscle force that is associated with BAW may be caused by an abnormally powerful clasp-knife reflex. This reflex has previously been observed only in spastic humans (Burke *et al.*, 1971a, b) and reduced animal preparations (Sherrington, 1909 in Burke *et al*; 1972; Burke *et al.*, 1972; Rymer *et al.*, 1979; Cleland and Rymer, 1990; Cleland *et al.*, 1990) and is traditionally defined as a sudden decrease in muscle force and EMG that occurs when a spastic limb is moved quickly and forcibly (Rymer, 1979). Although

textbooks often describe clasp-knife reflex as an autogenic, force limiting reflex that is probably mediated by Golgi tendon organs (GTOs) or secondary spindle afferents, more recent investigations have revealed that it may be a version of flexion-withdrawal reflex that is mediated by stimulation of Group II, III, or IV muscular free nerve endings (FNE) (Rymer, 1979).

Characteristics of clasp-knife reflex

The characteristics of clasp-knife reflex have been studied in spastic humans and reduced animal preparations. The decerebrate cat with dorsal hemisection of the spinal cord has been used as a model of human clasp-knife reflex because stretch induced inhibition of EMG and force that closely resembles clasp-knife inhibition in the spastic condition is observed in this preparation (Burke *et al.*, 1972; Rymer *et al.*, 1979; Cleland *et al.*, 1990). The animal studies that are discussed below utilized this preparation.

In the decerebrate spinalized cat, Rymer *et al.* (1979) demonstrated the autogenic nature of clasp-knife reflex by stretching the functionally isolated soleus and observing an inhibitory (clasp-knife) response in soleus. (See Figure 0.2.) This group also confirmed previous observations in cat and human subjects that clasp-knife reflex displayed a clear length threshold (Burke *et al.*, 1972, 1971). Small stretches of soleus were purely excitatory while large stretches produced excitation followed by inhibition of EMG and force. The

extent of inhibition was influenced by initial muscle length and initial motor output, as measured by background EMG. As these 2 parameters increased, so did the extent of clasp-knife inhibition. This relationship was presumably due to a dependence of the reflex on muscle force.

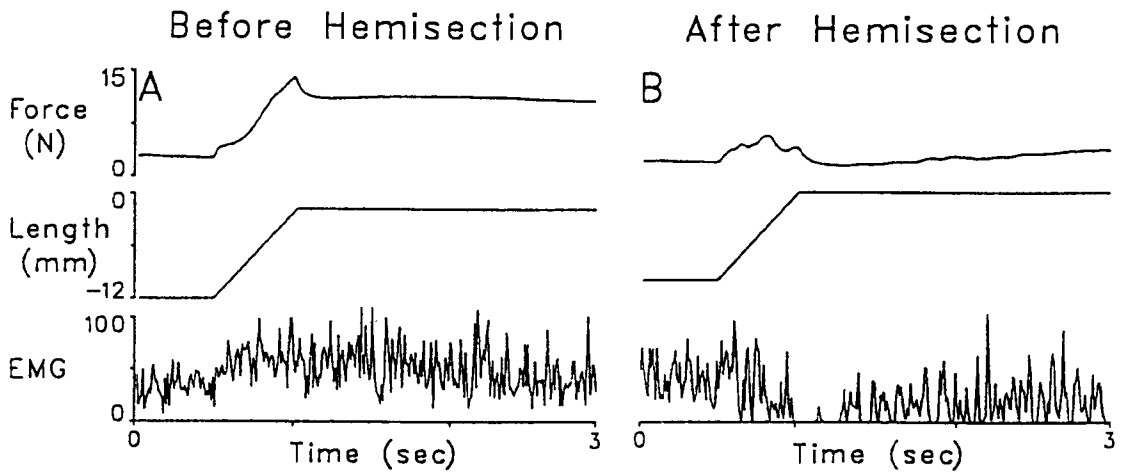

Figure 0.2. Example of Clasp-Knife Reflex in Reduced Cat Preparation. (From Cleland, *et al.*, 1990) A. Stretch of the soleus muscle in decerebrate cat produced an increase in force and EMG. B. After dorsal hemisection at T12, a similar stretch caused brief excitation followed by inhibition. This is clasp-knife reflex. Similar preparations have revealed clasp-knife in synergistic muscles and homonymous muscles.

In the decerebrate hemispinalized animal preparation, Cleland and Rymer (1990) elaborated on the description of clasp-knife reflex by studying the effects of muscle stretch and contraction on homonymous extensors, synergistic extensors, and flexors in the hindlimb. They found that stretch of one extensor (e.g. soleus) produced, not only homonymous inhibition, but also synergistic inhibition to other extensor muscles (e.g. medial gastrocnemius, lateral gastrocnemius, and plantaris). The threshold and intensity of synergistic clasp-

knife responses were affected by amplitude of stretch, level of initial motor output, and initial muscle length. Other features of homonymous and synergistic clasp-knife reflex that were identified by this group included an average latency of 106-138 ms (slightly longer in the synergist), inhibition before the stretch was complete, segmentation (or irregular peaks) in EMG during the stretch, decay of inhibition during maintained stretch, adaptation to repeated stretch, and inhibition that persisted beyond termination of stretch.

Because muscle stretch evoked increases in both muscle length and muscle force, it was unclear which of the two stimuli contributed to clasp-knife responses, change in length or change in force. Using the synergistic model of clasp-knife reflex, Cleland and Rymer (1990) electrically stimulated one muscle to increase force only and measured the extent of inhibition of a synergistic muscle. In fact, electrical stimulation of one muscle produced inhibition of the synergist, indicating that force alone may contribute to clasp-knife reflex. But contraction alone produced less inhibition than stretch that produced similar levels of force. This result suggested that change in length may also contribute to clasp-knife inhibition. The clasp-knife response was most robust when contraction and stretch were applied together, indicating that a combination of increased length and increased force contributed to clasp-knife reflex.

Investigation of the spatial divergence of clasp-knife reflex, demonstrated that stretch of extensors produced, not only inhibition of homonymous and synergistic extensors, but also excitation of flexors (Cleland and Rymer, 1990). Furthermore, stretch of flexors excited flexors and inhibited extensors. This spatial pattern of EMG suggested that clasp-knife reflex may be a version of flexion-withdrawal reflex. To further investigate this hypothesis, flexion withdrawal reflex was evoked in cat hindlimb by application of noxious stimulation to the bottom of the foot (Cleland and Rymer, 1990). The pattern of muscle activity produced by stretch of the extensors closely resembled the pattern of activity produced by noxious stimulation of the foot. Based on these observations, it seemed likely that clasp-knife reflex was not a simple autogenic force limiting reflex, but rather a version of flexion-withdrawal reflex that is evoked by muscular stimuli.

Receptors mediating clasp-knife reflex

For many years, controversy has surrounded the question of which receptors mediate clasp-knife reflex. Until recently, GTO and secondary spindle endings had been the two most commonly indicated receptors.

GTOs, first suggested by Matthews (1933), had been implicated because they have inhibitory connections onto homonymous motor neurons and were thought to be activated by high force and large stretches. Recent evidence has

suggested that GTOs alone could not be responsible for clasp-knife inhibition. GTOs have been shown to be very sensitive to contraction and to be active over a normal range of force gradation, not just high force. Cath and Crago (1982) found that in the decerebrate hemispinalized cat the strength of GTO force feedback was low. Rymer and coworkers (1979) showed that the threshold for GTO activation was much lower than the threshold for clasp-knife inhibition. Burke *et al.* (1971) found that ischemic block of Group I afferents did not abolish clasp-knife reflex in spastic humans. Cleland and Rymer (1990) showed that stretch of a flexor muscle produced homonymous excitation; whereas, GTOs in flexor muscles produced homonymous inhibition. They further found that clasp-knife inhibition declined during maintained stretch, was not affected immediately by release of stretch, and adapted to repeated stretch. One the other hand, GTOs were excited by static stretch, quickly turned off on release of stretch, and did not adapt to repeated stretch. Clasp-knife inhibition also persisted after force had declined due to the inhibitory process; whereas, Ib firing declined in parallel with decreases in muscle force (Rymer *et al.*, 1979).

Burke *et al.* (1970) suggested that Group II spindle endings were responsible for clasp-knife reflex because both clasp-knife reflex and Group II endings were thought to display a discrete length threshold and Group II endings were believed to evoke flexion reflexes (Matthews, 1972). Furthermore, clasp-knife inhibition in spastic humans was obliterated when procaine was

used to selectively block small diameter fibers in triceps surae of spastic humans (Burke *et al.*, 1971). Recently, these arguments have been contradicted. Rymer (1979) showed that the length threshold for clasp-knife reflex was poorly correlated with the length threshold for secondary spindle afferents. Iles (1989) showed that stimulation of secondary spindles did not evoke homonymous inhibition to extensor muscles. Hence, secondary spindle endings would have to decrease firing to cause clasp-knife reflex. Rymer *et al.* (1979) showed that both secondary and primary spindle endings displayed no decrease in discharge during clasp-knife. It is entirely possible that Burke's (1971) observation with the procaine block, was due to blockage of small diameter afferents other than Group II fibers that may have been responsible for the inhibition. Also, unlike clasp-knife reflex, secondary spindle afferents were excited by static stretch, turned off by release of stretch, and did not adapt to repeated stretch.

As early as 1979, Rymer *et al.* proposed that Group II, III and IV non-spindle afferents arising from muscular FNE (free nerve endings) may mediate clasp-knife reflex. They found that the stretch response of FNE was consistent with clasp-knife inhibition. Later, Cleland *et al.* (1990) compared the firing characteristics of mechanically sensitive muscular FNE in cat hindlimb to the characteristics of clasp-knife reflex and found that neither was excited by small stretches, both were excited by large stretches near maximum physiological length, both had an onset latency of approximately 118 ms, both displayed

segmentation of responses during the ramp, both had overshoot at the end of ramp, both decayed during maintained stretch, both were excited by isometric contraction, and both had a larger response to stretch than contraction at matched force levels. Furthermore, gentle tendon manipulation was a powerful stimulus for both.

The known properties of muscular FNE also suggested that they may be the receptors involved in clasp-knife reflex. FNE are located throughout the muscle and tendon, in the connective tissue of muscle, between intrafusal muscle fibers, between extrafusal muscle fibers, in the adventitia of arterioles and venules, in the capsule of the GTO, and in the tendon tissue of the neuromuscular junction (Stacey, 1969). Mense and Meyer (1987) identified four different types of slowly conducting afferents arising from cat skeletal muscle that were selectively sensitive to pain, light pressure arising from gentle tissue indentation, moderately high force from muscle contraction and stretch, and changes in temperature. Abrams *et al.* (1984) identified Group III muscular afferents in cat cervical dorsal rami that had small receptive fields and responded to localized pressure and stretch. Furthermore, FNE have been shown to be sensitive to metabolic byproducts, ionic alterations, fatigue, and inflammation (Hayward *et al.*, 1991; Grigg *et al.*, 1986). Hayward and co-workers (1991) have shown that muscle fatigue increases spontaneous discharge of Group III and IV muscle afferents and increases their sensitivity to stretch,

contraction and mechanical stimulation. This group has also shown that fatigue induced excitation of slowly conducting afferents causes inhibition of motor neuron output.

These observations taken together suggested that clasp-knife reflex is a version of flexion-withdrawal reflex and is characterized by homonymous and synergistic inhibition of extensors and simultaneous excitation of flexors. These data also suggested that the reflex actions were caused by stimulation of mechanically sensitive muscular FNE. The function of this reflex in intact humans remains unknown. It seems unlikely that it functions to protect the muscle from damaging forces because it is elicited, at least in pathological and animal preparations, in response to innocuous stimuli within the physiological range. It may be more likely to function as a protective response, signaling local stress or stain in the muscle and producing limb unloading.

Relationship of clasp-knife reflex to BAW

From the beginning of our investigation into BAW, it had been suggested that muscles with BAW displayed a substantial decrease in force when the muscle was highly activated (near maximum voluntary contraction) and then underwent a large stretch. It has been suggested that BAW may become evident after several trials performed at high levels of muscular effort, suggesting that fatigue may contribute to BAW (Archibald and Mathias, 1991; personal

communication Richard Sweeting). These observations suggest that clasp-knife reflex or a similar FNE mediated pathway may be responsible for BAW.

Another possible explanation for the influence of repeated trials on BAW is that repetition is necessary to provide a conditioning input to activate interneurons in a polysynaptic pathway. It is possible that the BAW response may be observed only after the pathway has been activated. This explanation is consistent with clasp-knife reflex because it is known to be mediated by a polysynaptic pathway with identifiable interneurons (Cleland and Rymer, 1993). What is unknown, however, is whether or not the "clasp-knife" interneurons require conditioning input to "turn-on" the pathway.

Although clasp-knife reflex has never been identified in non-neurologically impaired humans, it is likely that FNE play a role in normal movement. It has been shown that up to 75% of the sensory fibers innervating muscle in cat terminate in FNE (Stacey, 1969). If similar numbers of FNE exist in humans, it is conceivable that that they may have a protective role, functioning to protect muscles and tendons from unexpected, rapid increases in force and length by causing limb collapse (Rymer *et al.*, 1979). If FNE-mediated reflexes were functioning at an abnormally high gain or low threshold, we would expect that the muscles may shut down at inappropriate times producing responses that resemble BAW.

Project Objective and Plan

This project was divided into 4 phases that are discussed in detail in the sections to follow. All 4 phases were designed with respect to this background and introduction to achieve the following objectives:

1. To develop a quantitative testing technique that can reliably detect BAW.

2. To determine whether or not BAW is associated with musculoskeletal injury and/or residence in the Lower Fraser Valley.

3. To describe and quantify the characteristics of BAW.

4. To determine whether or not clasp-knife pathways could be responsible for BAW.

Phase I

Background for Phase I

Based on information that patients who are potentially affected with BAW often have disabling musculoskeletal disorders that are difficult to diagnose and that the manual test for BAW has a number of inadequacies, it became evident that a systematic approach for identifying BAW may aid in diagnosing this population. Furthermore, the development of a quantitative test may enhance the understanding of the mechanisms that are responsible for BAW and may lead to new approaches for treatment.

Phase I of this project was dedicated to developing a preliminary version of a diagnostic test that was reproducible, quantitative, and capable of detecting BAW.

Goals of Phase I

1. To develop a preliminary version of a diagnostic test that was capable of detecting BAW.

2. To determine whether or not patients referred by Sweeting displayed a shutdown in PT EMG that would suggest that BAW was present.

3. To determine whether or not a shutdown in PT EMG occurred early enough that it could be attributed to an abnormal stretch reflex.

4. To evaluate the behavior of the biceps (BI) and to determine whether or not it may play a role in BAW.

Methodology for Phase I

Subjects Tested

A total of 14 subjects (8 controls and 6 patients) were tested over a period of 2 weeks. The control group consisted of 5 male and 3 female volunteers who lived outside the LFV and ranged in age from 25-45 years. The patient group consisted of 2 male and 4 female volunteers who were patients of Sweeting and had been manually diagnosed with BAW. Each of the patients had been treated recently for musculoskeletal disorders, and some had disabilities that prevented them from working or participating in leisure activities. All the patients lived in the LFV and ranged in age from teens to adults in their forties. All subjects gave verbal consent prior to participating in the experiment.

Test Apparatus and Protocol

The testing method that was used included the cardinal features of the manual test but was more objective and quantitative. The muscle that was tested was PT for reasons stated in the Introduction. The initial plan was to rotate the wrist with a computer controlled motor attached to a rigid handle

gripped by the subject. The motor configuration that was available was incapable of producing rotations that were fast and strong enough to overpower the subjects' maximum pronation effort; therefore, a modified manual technique was used.

Subjects stood and gripped a steel handle that was mounted on a rigid, immovable stand. The upper arm was positioned next to the torso with the shoulder at zero degrees of flexion, abduction and external rotation. The elbow was positioned in approximately 90 degrees of flexion, and the wrist was neutral with respect to flexion/extension and pronation/supination.

The subject was asked to produce maximum voluntary contraction in the direction of pronation (MVC) while the examiner held the handle stationary, resisting the subject's effort. When the examiner sensed that maximum effort was achieved, the handle was manually rotated in the direction opposite of the subject's effort, overpowering the subject. As soon as the rotation was complete, the handle was returned to the start position. Although information about the velocity and amplitude of the rotation was not analyzed in detail, it was estimated that the rotations were approximately 20-40 degrees in amplitude delivered in 100-200 ms. The instruction to the subject was "Maintain maximum pronation effort despite any rotation of the handle."

In most cases the procedure was repeated several times on each limb. In some cases only one rotation was delivered after which the subject was given a brief rest (referred to as single trials). In other cases 2, 3, or 4 rotations were delivered in quick succession with no rest in between (referred to as multiple trials).

Data Collection and Processing

After the skin was cleaned with alcohol, a bipolar surface EMG electrode was placed on the PT and on the BI to record muscle activity throughout the trials. A potentiometer mounted on the handle measured the handle and limb displacement. The raw EMG signals and position traces were recorded, plotted on paper, and visually inspected.

Subject responses were evaluated based on the following criteria:

1. Increase in PT EMG during the rotation. If during handle rotation, subjects displayed a visually detectable increase in PT EMG compared to baseline, they were given the classification of YES for this criterion. If no increase in PT EMG during the rotation could be detected, subjects were given the classification of NO for this criterion. An increase in PT EMG during handle rotation was considered evidence of intact short latency stretch reflex pathways.

2. Increase in BI EMG during the rotation. If during handle rotation, subjects displayed a visually detectable increase in BI EMG compared to baseline, they

were given the classification of YES for this criterion. If no increase in BI EMG during the rotation could be detected, subjects were given the classification of NO for this criterion.

3. Evidence of BAW. If during or after handle rotation, but not before the handle began to return to the start position, subjects displayed a decrease in PT EMG that was below background levels, they were given the classification of YES for this criterion. If no decrease in PT EMG was visually detectable, and PT EMG remained $\geq$ background levels, then subjects were given the classification of NO for this criterion.

4. Sweeting's manual diagnosis. All patients and some control subjects were manually tested by Sweeting in addition to undergoing the handle rotation method. In these cases, the results of Sweeting's diagnosis were compared to the results of the handle rotation test. If Sweeting's test revealed BAW, subjects were classified as positive (+) for BAW according to Sweeting's manual diagnosis.

Results of Phase 1 Trials

In all 8 control subjects, there was no evidence of BAW with the newly developed handle rotation technique. An example of a typical control response is depicted in the left panel of Figure 1.1. In response to both single and

multiple rotations, control subjects typically displayed a visually detectable increase in PT EMG during handle rotation. After this initial rise in activity, PT EMG returned to near-background levels. During the test, PT EMG never fell below background levels, as would be expected if BAW were present. Control subjects also displayed a transient increase in BI EMG during the rotation that was followed by a return to background levels. One control subject (SS) was diagnosed with BAW according to Sweeting's manual evaluation.

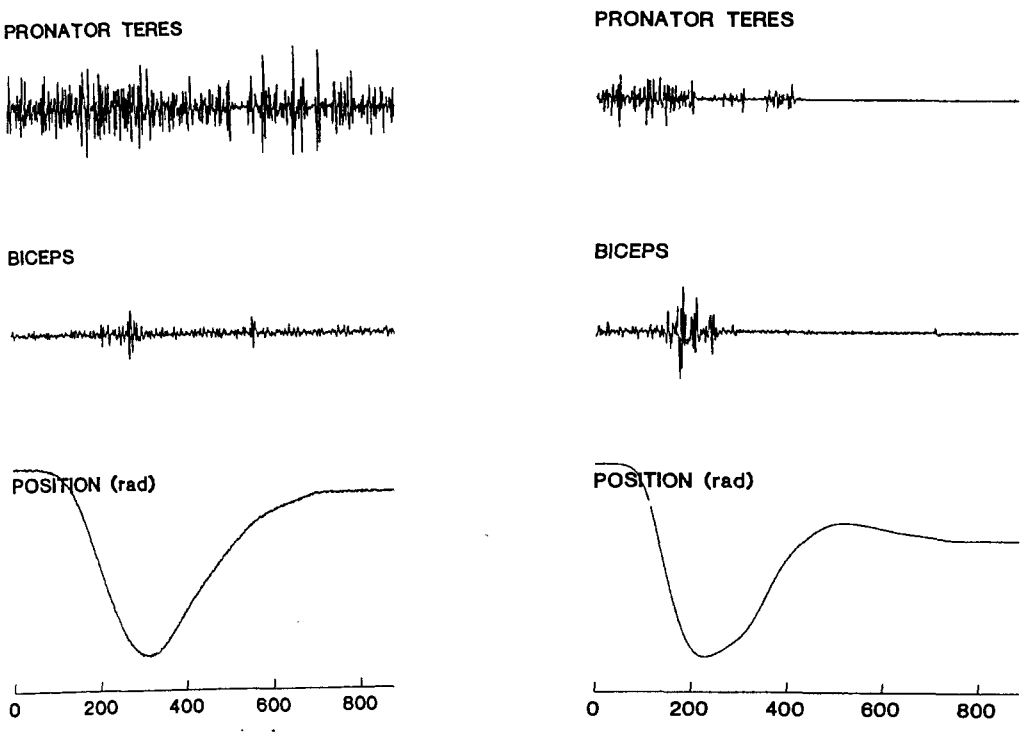

Figure 1.1. Normal and BAW Responses. Top two traces represent raw EMG from PT and BI. Bottom traces represent relative displacement of limb from start position of approximately 0° of pronation/supination. Displacement is represented in downward direction because the rotation was counterclockwise, supinating the left wrist. Time scale is in milliseconds. Left Panel: Typical Control Responses. Data from TS, left limb, single rotation. PT EMG increased after the onset of the rotation and remained near initial values for the duration of the rotation. PT EMG did not decrease until the handle returned to the start position and the trial was complete. BI displayed a characteristic increase in activity after the onset of the rotation, but was otherwise quiet throughout the trial. There was no evidence of BAW in this trial. Right Panel: BAW Response. Data from MA, left limb, single rotation. There was a slight increase in PT and BI EMG after the onset of handle rotation, followed by a dramatic and sustained decline in PT EMG that remained near zero for the duration of the trial. This response suggested that BAW may have been present.

In 1 of 6 patients tested, a response that was different from the control response and consistent with BAW was observed. In this patient (MA), there was a decline in PT EMG that began approximately 100-120 ms after the onset of the rotation and remained low for the duration of the trial. The subject displayed this response in the left limb in 1 out of 4 trials. (See Figure 1.1 right

panel.) Aside from this one observation, all other patient responses resembled control responses. During handle rotation, patients displayed an increase in PT and BI EMG followed by a return to initial background levels. Even the subject who displayed evidence of BAW, first displayed an initial, transient rise in PT and BI EMG after the onset of handle rotation. All patients were diagnosed with BAW according to Sweeting's manual test. Phase I data is summarized in Table 1.1.

Subject Code	*L or R Limb*	*Type of Rotation*	*Increase PT EMG*	*Increase BI EMG*	*Evidence of BAW*	*Sweeting's Diagnosis*
Controls						
MT	R	Single	Yes	Yes	No	Not Tested
MT	R	Triple	Yes (2/3)	Yes (3/3)	No	Not Tested
MT	L	Single	Yes	Yes	No	Not Tested
MT	L	Triple	Yes (3/3)	Yes (3/3)	No	Not Tested
WH	R	Single	Yes	No	No	Not Tested
WH	R	Single	Yes	Yes	No	Not Tested
WH	R	Triple	Yes (3/3)	Yes (3/3)	No	Not Tested
WH	L	Single	Yes	No	No	Not Tested
WH	L	Single	Yes	Yes	No	Not Tested
WH	L	Triple	Yes (3/3)	Yes (2/3)	No	Not Tested
LA	L	Single	Yes	Yes	No	Not Tested
LA	L	Quad	Yes (4/4)	Yes (3/4)	No	Not Tested
LA	L	Quad	Yes (4/4)	Yes (3/4)	No	Not Tested
SS	L	Single	Yes	Yes	No	(+)
SS	L	Single	Yes	Yes	No	(+)
TS	L	Single	Yes	Yes	No	(-)
TS	L	Double	Yes (2/2)	Yes (2/2)	No	(-)
TS	L	Triple	Yes (3/3)	Yes(2/3)	No	(-)
CP	R	Single	Yes	Yes	No	Not Tested
CP	R	Triple	Yes (3/3)	Yes (2/3)	No	Not Tested
CP	L	Single	Yes	Yes	No	Not Tested
CP	L	Triple	Yes (2/3)	Yes (2/3)	No	Not Tested
EM	R	Single	Yes	Yes	No	Not Tested
EM	R	Triple	Yes (2/3)	Yes (3/3)	No	Not Tested
EM	L	Single	Yes	Yes	No	Not Tested
EM	L	Triple	Yes (3/3)	Yes (3/3)	No	Not Tested
SR	R	Single	Yes	Yes	No	Not Tested
SR	R	Triple	Yes (3/3)	Yes (3/3)	No	Not Tested
SR	L	Single	Yes	Yes	No	Not Tested
Patients						
MA	R	Single	Yes	Yes	No	(+)
MA	R	Triple	Yes (3/3)	Yes (3/3)	No	(+)
MA	R	Triple	Yes (3/3)	Yes (3/3)	No	(+)
MA	L	Single	Yes	Yes	No	(+)
MA	L	Single	No	Yes	No	(+)
MA	L	Single	Yes	Yes	Yes	(+)
MA	L	Triple	Yes (3/3)	Yes (3/3)	No	(+)
LE	R	Single	Yes	Yes	No	(+)
LE	R	Single	Yes	Yes	No	(+)
LE	R	Triple	Yes (3/3)	Yes (3/3)	No	(+)
LE	R	Triple	Yes (3/3)	Yes (3/3)	No	(+)
LE	R	Triple	Yes	Yes	No	(+)
KA	L	Triple	Yes (3/3)	Yes (3/3)	No	(+)
RA	R	Single	Yes	Yes	No	(+)
RA	L	Single	Yes	Yes	No	(+)
RA	L	Single	Yes	Yes	No	(+)
RA	L	Triple	Yes (1/3)	Yes (3/3)	No	(+)
DO	R	Single	Yes	Yes	No	(+)
DO	R	Single	Yes	Yes	No	(+)
DO	R	Triple	Yes (2/3)	Yes (3/3)	No	(+)
DO	R	Triple	Yes (2/3)	Yes (1/3)	No	(+)
DO	L	Single	Yes	Yes	No	(+)
DO	L	Single	Yes	Yes	No	(+)
DO	L	Triple	Yes (2/3)	Yes (3/3)	No	(+)
JE	R	Single	Yes	Yes	No	(+)
JE	R	Single	Yes	Yes	No	(+)
JE	R	Single	Yes	Yes	No	(+)

Table 1.1. Summary of Data Phase I. Eight control subjects and 6 patients were tested. One patient (MA) displayed a response that was consistent with BAW. For multiple rotation trials, the numbers in parentheses indicate the number of times out of the number of trials that a response was observed.

Discussion of Phase I Results

Based on these data, there was a hint that BAW may exist in the selected patient population and not in control subjects. However, these preliminary experiments suggested that BAW was clearly unrelated to abnormal short latency stretch reflex in PT, as both patients and control subjects displayed a rise in PT EMG during handle rotation when the PT was presumably undergoing stretch. Furthermore, the latency at which the decrease in PT EMG was observed (100-200 ms) was too long to be attributed to an abnormality of short latency reflexes. Hence, we concluded that both patients and control subjects displayed a normal short latency stretch reflex.

It is unlikely that BAW was caused by an unusually large increase in BI activity as would be expected if the subject had actively supinated the wrist. Patients and controls displayed similar bursts in BI activity during the rotation. The burst in BI EMG may have been caused by a vibration-induced shortening reaction that is thought to be caused by an increase in Ia firing when a limb is moved quickly enough to create vibration (Angel, 1982; Angel, 1983). Alternative explanations include a decrease in GTO inhibition to BI or increased co-contraction to increase joint stiffness in the face of the rotation.

Despite the fact that the new handle rotation test was only able to identify one case of BAW, we felt that the issue deserved further study. It

seemed possible that, with his manual test, Sweeting may have been able to detect something that our test was not sensitive enough to reveal. Subsequent experimentation was dedicated to developing a reproducible and quantitative test that was sensitive enough to detect BAW in individuals who had been diagnosed manually with the disorder.

Phase II

Background for Phase II

The results of Phase I suggested that BAW may exist in some patients and not in control subjects. The testing method that was used, however, lacked reproducibility because it was performed manually and did not provide a method for quantifying responses to a controlled stimulus. Furthermore, our ability to detect only one case of BAW suggested that the test may not have been as effective or as sensitive as Sweeting's manual test. Phase II was dedicated to developing a computer-controlled, motorized device that was capable of producing reproducible stretches of desired velocity, amplitude and duration, suitable for detecting BAW in subjects referred by Sweeting.

Goals of Phase II

1. To develop and refine a motorized, computer-controlled diagnostic test that was sensitive, objective, quantitative, reliable, and capable of detecting BAW.

2. To describe the characteristics of BAW.

3. To investigate whether or not clasp-knife reflex pathways could be responsible for BAW.

4. To investigate whether or not BAW was more prevalent in injured patients who live in the LFV than control subjects who live outside the LFV.

Methodology for Phase II

Apparatus

A new computerized motor apparatus was designed to test the PT for presence or absence of BAW in 18 subjects. The test apparatus consisted of a motor (PMI ServoDisc ™ DC Motor, model number 00-J1644-002) that was operated by position feedback and controlled by a computer (PC 386). The motor was attached to a vertical handle via a horizontal shaft and pulley arrangement and was sufficiently stiff to resist movement of the handle caused by wrist pronation and supination. (See Figure 2.1.) The motor could also rotate the handle to displace the wrist in the direction of supination. The position control system was designed so that each rotation was a specific amplitude and velocity regardless of differences in subject strength. The motor was capable of producing enough torque to overpower even the strongest subject at velocities up to 500°/second.

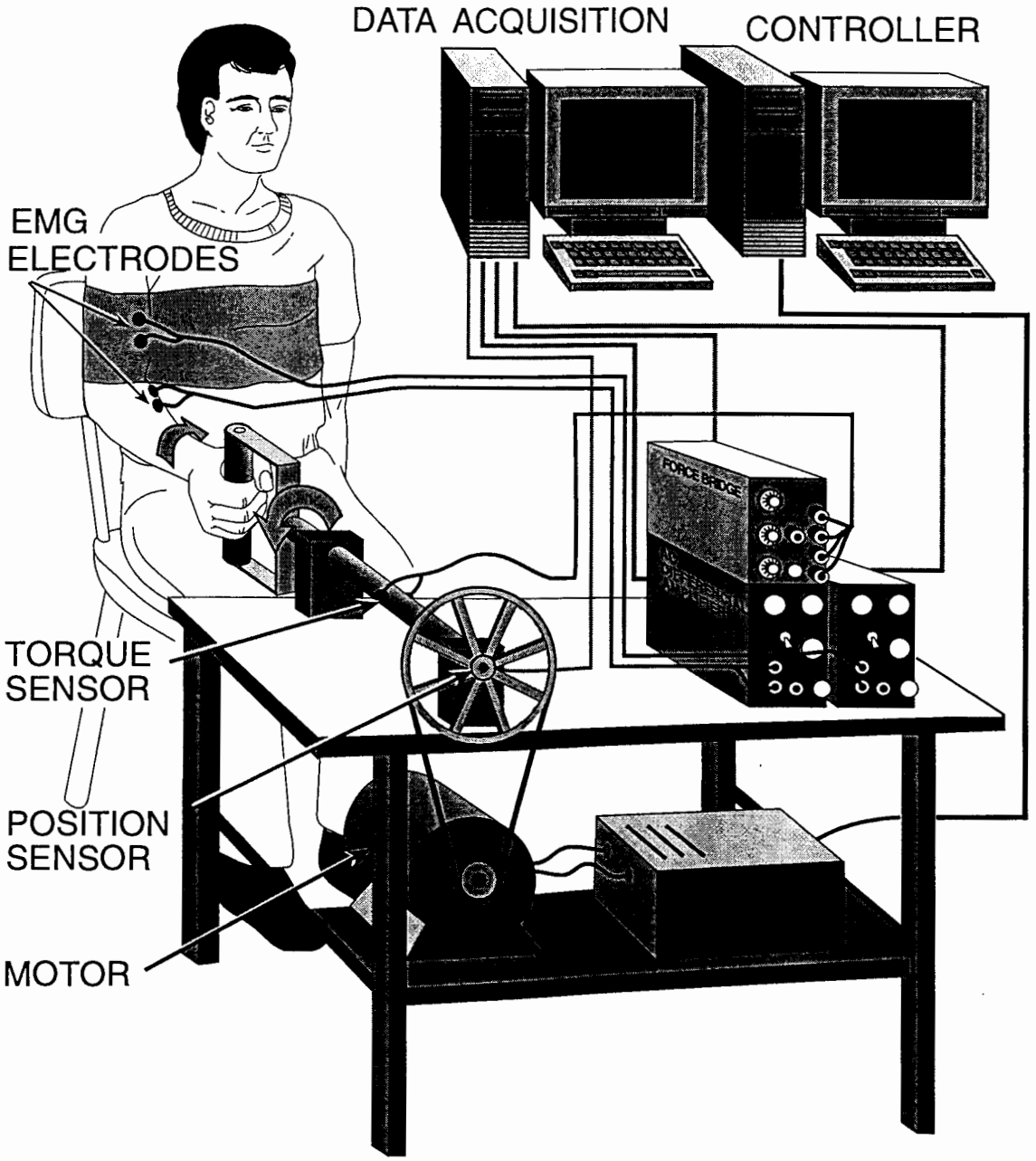

Figure 2.1. Diagram of Subject Sitting at the Breakaway Testing Apparatus.

Test Procedure

Establishing MVC

While seated, subjects were asked to hold the handle vertically and isometrically pronate against the resistance of the motor. The arm was positioned as in Phase I with the addition of a wide Velcro strap placed snugly around the arm and torso to prevent substitution from shoulder muscles. The subject gradually increased pronation effort against the handle to determine maximum voluntary isometric pronation torque (MVC). Verbal cues, visual cues, and encouragement were given to ensure that subjects were exerting maximum effort. The highest torque that was achieved in 2-3 trials was recorded as MVC.

Rotation Trials

After MVC had been established, the subject was asked to pronate against the handle and maintain a specified level of effort (either 0%, 50% or 100% of MVC). Subjects were given a visual target on an oscilloscope to help achieve the specified level of effort. Subjects were told that after the specified level of effort was achieved, the handle would rotate in the direction opposite their effort. The instruction to subjects was: "Maintain your level of effort regardless of any movement of the handle." After the specified level of effort was achieved, the examiner triggered the rotation of the handle which resulted

in a rapid displacement of the limb, overpowering the subject. Mechanical safety stops were placed at ±45° of wrist rotation to prevent the subject's wrist from being supinated beyond normal limits.

Nine types of test modes were used, each repeated 3 times. Rotations were delivered in the form of a single brief pulse, a single rectangular step, or three brief pulses delivered 400 ms apart. (See Figure 2.2.) Pulse rotations measured approximately 22° in amplitude and had a risetime of 100 ms. Step rotations measured approximately 25° in amplitude and had a risetime of 100 ms. Pulse rotations ramped down immediately after the rise was complete, but the step rotations were held at the 25° displacement for 400 ms and then ramped down. Trials were performed at three levels of background pronation torque: 0%, 50% and 100% of MVC.

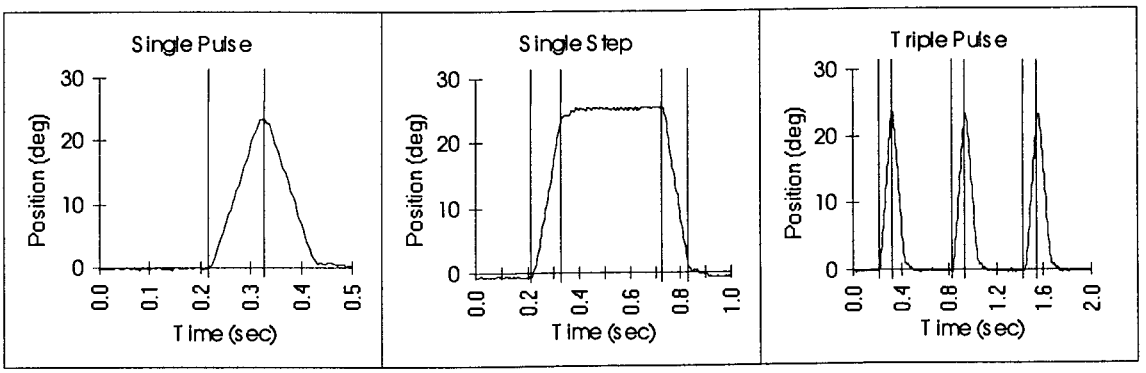

Figure 2.2. Three types of rotations were delivered to all subjects: single pulse, single step, and triple pulse. Each rotation was 22°-25° in amplitude and had a risetime of 100 ms. The single pulse ramped down immediately after the rise was complete. The single step was held at the 25° displacement for 400 ms and then ramped down. The triple pulses were identical to the single pulses except that 3 were administered in succession with a 400 ms pause in between. Note the difference in time scales for each of the figures. Vertical lines bracket the "ramp phase" of rotations.

Data Collection

EMG

Bipolar surface electrodes (Sensormedics, 16 mm, silver-silver chloride) filled with conductive gel (Teca Corporation) were used to record EMG from BI and PT. After the skin was cleaned with alcohol, one pair of electrodes was placed over the BI muscle belly in a position that was best for recording EMG associated with wrist supination. The second pair of electrodes was positioned over the belly of the PT. Care was taken to place the electrodes in such a way that the signals were largest when either wrist pronation or supination was performed and smallest when other movements, such as wrist and elbow flexion, were performed. A wrist-band ground electrode that was fabricated in the lab was used for noise reduction and to ground the subject. Analog EMG signals were amplified 1000 times and band pass filtered at 50-500 Hz using an AC differential amplifier (BAK electronics).

Torque

Torque was measured by semiconductor strain gauges that were mounted on the handle of the apparatus in such a way that they were most sensitive to pronation and supination torque and less sensitive to torque produced by movements in other planes such as wrist flexion/extension and elbow flexion/extension. The strain gauges were connected to a force bridge amplifier (BAK Electronics). The torque sensor was manually calibrated prior to

data collection by fastening a broomstick of known length to the handle and hanging known weights from the stick by way of a pulley. The torque applied to the handle was calculated and recorded along with the voltage reading from the torque sensor. After several reading were obtained, the torque value corresponding to 1 volt was calculated.

Position

Handle position was monitored by a potentiometer mounted on the wheel of the apparatus that was connected to the handle by a rigid shaft (15 mm diameter with a length of 55 cm). The position of the wheel accurately reflected the handle position to within ± 1 degree.

Trigger and Command Signals

Both the data acquisition trigger signal that initiated data collection and the command signal to the motor that controlled how and when the handle turned were recorded as a timing reference for other events.

Data Recording and Digital Processing

Analog EMG, torque, position, command, and trigger signals were simultaneously recorded on-line on FM tape and later sampled with digital data acquisition software (Datasponge) at a rate of 1000 samples/sec/channel. Digital smoothing of EMG was completed in Matlab and consisted of full wave rectification, bin integration (3 ms bins) and averaging (15 point moving

average). EMG, position, and torque traces were then displayed using graphical display software (Microsoft Excel) and visually inspected.

Subjects

A total of 18 subjects (7 controls and 11 patients) were tested bilaterally in Phase II. The control group consisted of 3 male and 4 female volunteers who were free of musculoskeletal injury and lived outside the LFV. The control subjects were students from the School of Kinesiology who were very athletic and motivated. They ranged in age from 23-28 years. The patient group consisted of 5 male and 6 female volunteers who were patients of Sweeting and had been manually diagnosed with BAW. Each of the patients had recently been treated for musculoskeletal disorders. Some patients had disabilities that prevented them from working or participating in leisure activities. All the patients lived in the LFV and ranged in age from teenagers to adults in their fifties. All subjects gave written informed consent, according to Simon Fraser University Ethics regulations prior to participation in the study.

Subject Coding

At the time of admission to the study, all subjects were given a code letter and number. Patients were identified by the letter *P* and controls were

identified by the letter C. After a subject was given a "subject code", the same code was used to identify the same subject in subsequent phases.

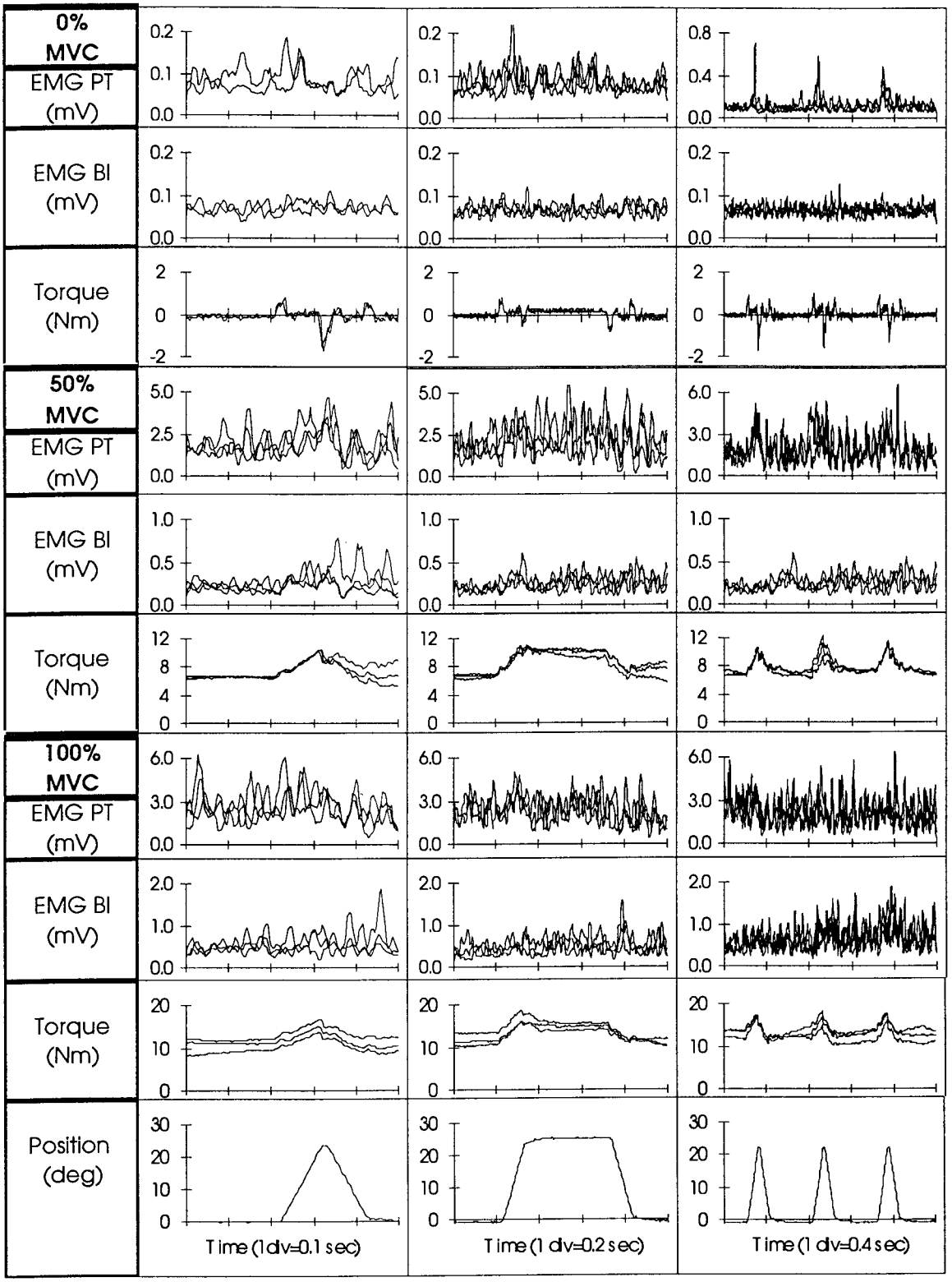

Figure 2.3. Summary of 9 Test Modes. Data from C4 Right. Each of the three types of rotations was delivered at 0%, 50%, and 100% MVC. Three repetitions were performed for each condition. These traces show typical normal responses.

Results of Phase II

Of the nine test modes utilized (Figure 2.3), two modes best revealed a response in patients that was clearly and systematically different from the control subjects. These modes were the step rotation administered at 100% and 50% MVC.

Normal Response: A typical control response to a step rotation, as displayed in Figures 2.3 (center) and 2.4 (left column), was characterized by an increase in torque during the handle rotation, followed by a slight decrease in torque when the rotation ended, and a plateau in torque that was approximately maintained for the duration of the "hold phase" of the step rotation. These changes in torque were accompanied by increases in BI and PT EMG immediately after the onset of the rotation and maintenance of elevated BI and PT EMG during the "hold phase". This response was considered normal because it is consistent with known muscle and reflex properties and was characteristic of all 7 control subjects.

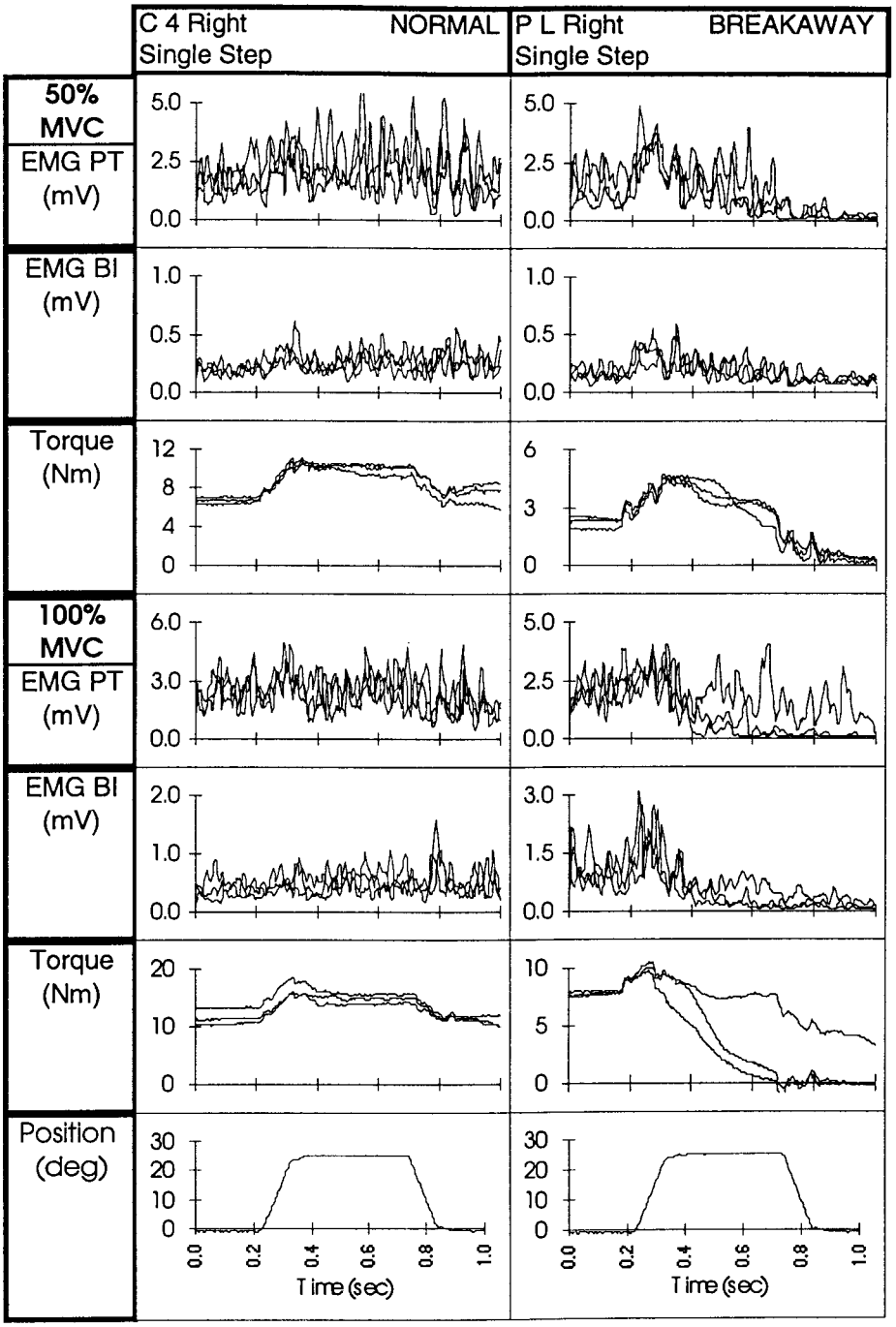

Figure 2.4. Normal vs. Breakaway Responses. Data from C4 and P7 Right. In the left column is a typical normal response. Torque and EMG increased during handle rotation. During "hold phase", torque and EMG declined and then returned to a level that was lower than peak value but above background. This response was the same for 50% and 100% MVC trials. In the right column is a typical BAW response. Torque and EMG increased during handle rotation, as in the normal case. During the "hold phase" there was a decline in torque and EMG that is characteristic of BAW. In this example and others, the first rotation resulted in a fairly normal response, but the next 2 rotations revealed BAW.

Breakaway Response: In 4 of the 11 patients (P2, P6, P7, P8), we observed a response to the step rotation applied against a background torque of 100% MVC that was clearly different from the control response. The initial portion of responses in these 4 patients looked identical to normal responses. They displayed an initial increase in torque and EMG of PT and BI during handle rotation. However, at approximately 120-150 ms after the onset of the step rotation, these patients displayed a large, rapid decline in pronation torque and EMG. (See figure 2.4, right column.) In patients, pronation torque and EMG in the PT and BI decreased simultaneously. We considered this response BAW.

In two of the patients (P6 and P7), the response was present bilaterally, and in the other 2 patients (P2 and P8), it was present unilaterally. Patients 6, 7, and 8 also displayed a weaker version of the same response unilaterally when a step rotation was applied at 50% MVC. This response is referred to as marginal BAW. In contrast, none of the 7 control subjects demonstrated a similar, sudden decline in torque or EMG after the rotation. Patients who did not display BAW produced responses indistinguishable from the normal responses.

As mentioned earlier, each of the test modes was repeated three times. BAW was never observed in the first of the three rotations. It only became evident on the second and third rotations. This issue was addressed further in

subsequent phases of experimentation. Table 2.1 summarizes the results of Phase II.

Subject Code	*Sex*	*Presence of BAW with Step Rotation at 50% MVC*	*Presence of BAW with Step Rotation at 100% MVC*
All Controls	3M/4F	No BAW	No BAW
P1	M		
R		No BAW	No BAW
L		No BAW	No BAW
P2	F		
R		No BAW	BAW (1/3)
L		No BAW	No BAW
P3	F		
R		No BAW	No BAW
L		No BAW	No BAW
P4	F		
R		No BAW	No BAW
L		No BAW	No BAW
P5	F		
R		No BAW	No BAW
L		No BAW	No BAW
P6	F		
R		Marginal BAW (1/3)	BAW (2/3)
L		No BAW	BAW (2/3)
P7	F		
R		Marginal BAW (1/3)	BAW (2/3)
L		No BAW	BAW (1/3)
P8	M		
R		Marginal BAW (1/3)	BAW (2/3)
L		No BAW	No BAW
P9	M		
R		No BAW	No BAW
L		No BAW	No BAW
P10	M		
R		No BAW	No BAW
L		No BAW	No BAW
P11	M		
R		No BAW	No BAW
L		No BAW	No BAW

Table 2.1. Summary of Phase II Results. Numbers in parentheses indicate the number of times out of the number of rotations that BAW was observed.

In these trials we never observed an *immediate* decline in torque or EMG in either patients or control subjects. In fact, during the "ramp phase", patients resembled controls in that they all typically exhibited initial increases in EMG and torque. The rapid decline in muscle activation and torque in the patients started at a latency longer than 100 ms. Conceivably this could have occurred if these patients were voluntarily "quitting" or reducing their effort upon detection of the start of the rotation, against our instruction.

To investigate this possibility, we asked one of the control subjects (C4) to repeat the test, except now with the instruction: "As soon as you feel the handle begin to rotate, relax all muscle activity as quickly as you can." In response to this instruction, the control subject started to reduce his pronation torque approximately 120 ms after initiation of the rotation. (See Figure 2.5.) However, the pattern that he used for decreasing torque voluntarily was different from the strategy exhibited by the patients. The control subject decreased pronation torque by decreasing EMG in PT and at the same time *maintaining* EMG in the antagonist, BI. This result may have been due to the instruction that was given to the subject which was to "relax all muscle activity *as quickly as you can.*" This instruction may have inadvertently cued the subject to do anything he could to stop producing pronation torque, including maintaining BI activity while

decreasing PT to "put the brakes on" pronation torque production, which was not the desired response.

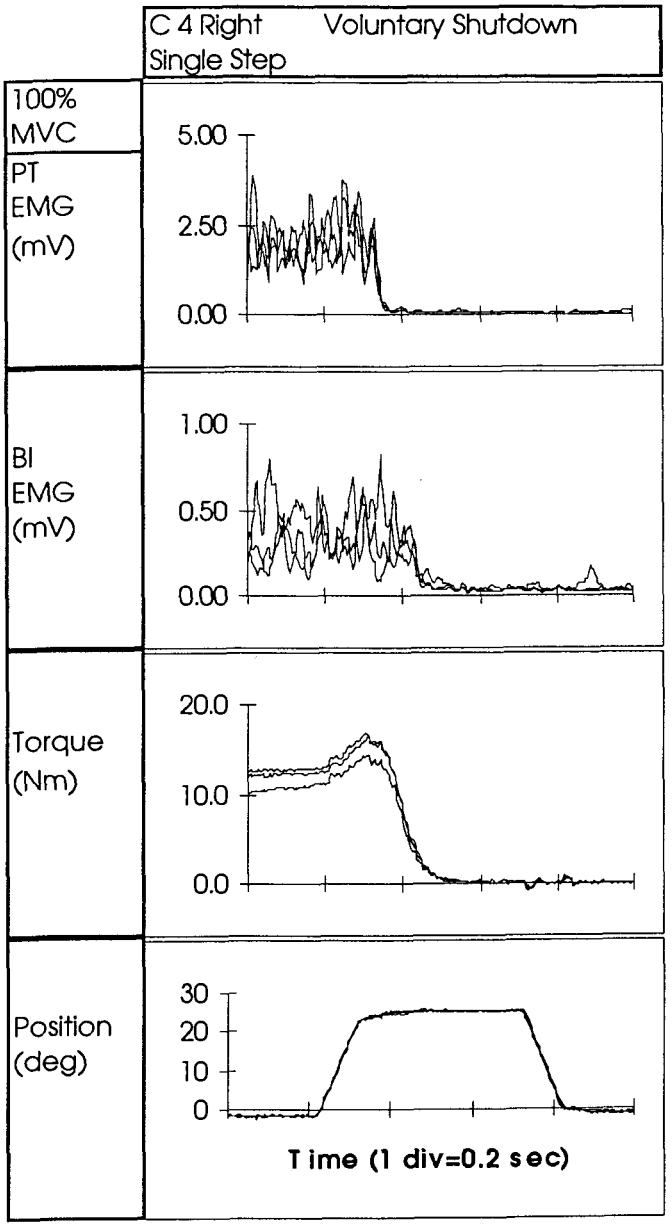

Figure 2.5. Voluntary shutdown trials. One control subject (C4) was asked to shutdown all muscle activity as quickly as possible as soon as handle movement was detected. The latency of shutdown of torque and EMG was similar to that of BAW trials. However, the strategy employed by this control subject was different in that he maintained BI EMG while decreasing PT EMG to "put the brakes on" pronation torque. This strategy is evidenced by the longer persistence of BI EMG.

Discussion of Phase II Results

When subjects were evaluated with the new motorized, computer-controlled diagnostic test, two distinct responses were observed: a normal response and a BAW response. Based on visual observation of the data, we defined a normal response as one in which torque and EMG during the "hold phase" of the step rotation remained equal to or greater than background values. BAW was defined as a response in which torque and usually EMG during the "hold phase" fell below initial values. Marginal cases of BAW were defined as cases in which torque and EMG during the "hold phase" declined but did not drop below background. Based on these definitions, BAW was detected only in the patient group. These results are consistent with Sweeting's clinical description of BAW and his observation that BAW is present in subjects that he has referred to the laboratory. These findings are also consistent with our results from Phase I.

The step rotation (consisting of a 25° rotation delivered in 100 ms, followed by a 400 ms "hold phase") was the only condition that clearly revealed BAW. The other two types of rotations produced identical responses in control subjects and patients. The ability of the step rotation to detect BAW was probably due to the 400 ms "hold phase" which may have allowed the muscles time to respond to the perturbation. The pulse rotations lasted only 100 ms and

may have ended before the muscle had an opportunity to respond. The observation that BAW only became apparent at high levels of voluntary effort (near 100% MVC) is consistent with Sweeting's report that BAW becomes evident when a muscle is producing a great deal of force.

In both control subjects and patients, pronation torque and PT EMG increased during handle rotation. This observation strongly suggests that the stretch reflex was normal in both groups. The time at which torque and EMG began to decline in BAW patients suggests that BAW was *not* caused by an immediate shutdown of the short-latency stretch reflex pathways. Also, the strategy used by one control subject to voluntarily shutdown his pronation torque as quickly as he could suggests that the patients did not intentionally turn off their pronation effort. Rather, our data suggest that the shutdown occurred involuntarily at a latency that is consistent with long-latency reflexes such as clasp-knife reflex. The fact that BAW was never observed on the first of three rotations suggests that a conditioning input may be required to activate interneurons in a polysynaptic pathway or that muscle fatigue may contribute to BAW.

Despite the fact that the test procedure successfully detected 4 of the 11 potential cases of BAW, 7 other possible cases were not detected. This result suggests that the test may not have been aggressive or sensitive enough to

detect the other, possibly less severe, cases of BAW. Alternatively, the condition of these patients could have changed since their manual diagnosis of BAW, or Sweeting could have misdiagnosed them. These observations prompted us to conduct Phase III in which we altered the test parameters to converge on a test that was optimal for detecting BAW.

Phase III

Background for Phase III

In Phase II, step rotations administered at either 100% or 50% of MVC led to detection of BAW in 4 out of 11 patients. There were 7 other patients, however, who were expected to have BAW but did not display it with this test. The inability to detect BAW in these subjects could be explained in a number of ways including misdiagnosis by Sweeting, insensitivity of the new test for detecting subtle cases of BAW, or inability to accurately replicate the manual test. Phase III was designed to optimize the test for BAW by increasing its ability to identify BAW in all patients who were thought to have it while minimizing detection in control subjects. By altering the amplitude, velocity, duration, and shape of the rotation as well as the handle position, background level of pronation effort, and limb position, an improved version of the test was identified.

Goals of Phase III

1. To converge on a test that was optimal for confirming manual diagnoses of BAW while minimizing BAW responses in normal control subjects.

2. To select control subjects matched in age and activity level to the patient pool to determine whether or not non-injured control subjects living outside the LFV display BAW.

Methodology for Phase III

The same test apparatus that was used in Phase II was used for this phase. The test protocol was similar to the one used in Phase II with the following improvements.

MVC trials

At the onset of every testing session, maximum voluntary isometric pronation torque (MVC) was measured. Subjects attempted to achieve MVC by increasing their effort at producing isometric pronation torque while following a computer generated target displayed on an oscilloscope screen. The target moved slowly either up or down the screen (depending on which limb was being tested) to a level that exceeded MVC. The instruction to the subject was "follow the target slowly and carefully by producing pronation force until no additional force can be produced, then hold that level of force until the trial is complete and the target line returns to the start position." Verbal encouragement was given to ensure that subjects produced their best effort.

Each trial was 5 sec in duration. Four repetitions of this task were conducted on each limb, and MVC was calculated by averaging the torque of the four trials at a point where torque reached a plateau.

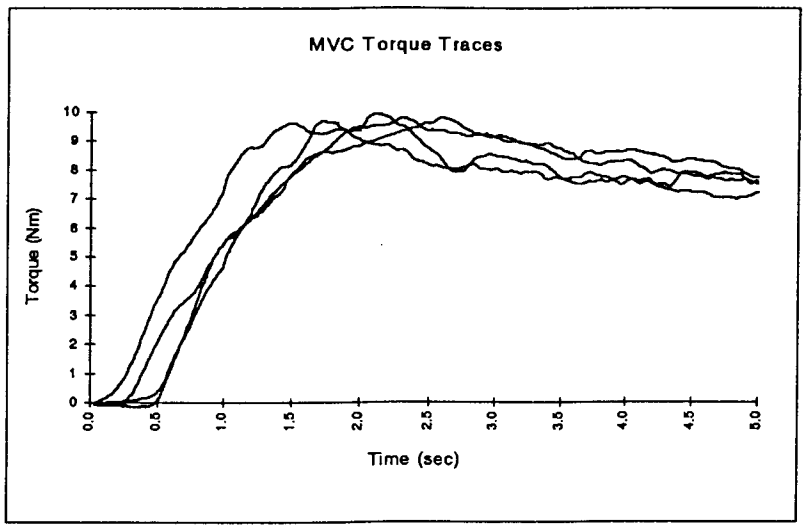

Figure 3.1. MVC Trials. Subject C1 right. Typical torque traces for pronation MVC trials.

Rotation trials

Subjects were asked to produce a specific amount of pronation torque by matching a computer generated target on an oscilloscope screen that represented a percentage of their MVC. After the subject reached the target, there was a random time delay of 0-3 seconds after which the rotation was delivered. The delay helped ensure that subjects achieved the specified level of

background torque prior to the rotation and that the exact time of onset of the rotation could not be anticipated. Rotations were delivered only if subjects were within $\pm$5% of the target.

Subjects were diagnosed with BAW if pronation torque during the "hold phase" of the rotation went below background torque. The most robust cases of BAW were the ones in which the torque plummeted to zero during the "hold phase". Less robust cases were those in which pronation torque during the "hold phase" went below background torque but did not reach zero before the end of the trial.

Catch trials and Mini-Rotation trials

In addition to rotation trials, we administered "catch trials" in which the handle did not rotate when the subject was anticipating a rotation and "mini-rotation" trials in which the handle rotated 3°-6° when the subject was anticipating a much larger rotation. The mini-rotation trials were large enough for subjects to feel, but should have been too small to elicit BAW. These two types of trials were designed to identify people who had a tendency to display inappropriate responses in anticipation of the rotation.

Subjects Tested

In previous testing, all controls subjects were athletic graduate students from the School of Kinesiology who were between the ages of 23 and 28, and may have been familiar with the anticipated results. In contrast, patients were from a diverse population consisting of a variety of age groups and differing levels of physical fitness. In Phase III, the pool of control subjects was expanded to include, not only subjects who were previously tested in Phases I and II, but also individuals who were more closely matched to patients in age and level of physical fitness. Control subjects who were added to the study were healthy, moderately active but not athletic, similar in age to many of the patients, and lived outside the LFV. Many of the controls were not from the School of Kinesiology and were naive to the experiment.

A total of 10 controls and 7 patients were tested in Phase III. Many subjects were used for more than one phase of optimization. Seven control subjects and 1 patient were added to the subject pool in phase III. The other 9 subjects had been tested previously in Phase II.

Subject Coding Convention

Subjects were assigned a code letter and number at the time of admission to the study. The letter *P* represented patients who were referred to the study by Sweeting and were manually diagnosed with BAW. The letter *C* represented control subjects. After a subject was assigned a "subject code" (i.e. *C1* or *P12*) the same code was always used for the same subject throughout Phases II and III. For example, the subject with the code *C1* in Phase II is the same individual as *C1* in Phase III. Subjects who were added to the experiment in Phase III have subject codes that do not appear in Phase II.

Changes to methods that were specific to a particular stage of optimization are described in detail prior to the results of the corresponding phase of optimization.

Results of Phase III

Changes to "ramp phase" of rotations

To determine whether variations in amplitude, duration, or velocity of the "ramp phase" of step rotations would enhance detection of BAW, subjects were re-tested with 4 different types of step rotations that are presented in Table

3.1 and Figure 3.2. Each rotation was a step rotation with a "hold phase" of 400 ms. Rotations differed from one another in the "ramp phase" parameters only. Subjects performed tests at 0%, 50%, and 85% of MVC. For each condition and at each level of MVC, one block of trials was delivered. A block of trials consisted of 3 rotation trials and 1 catch trial.

Five patients (P5, 6, 8, 9, 10) and 3 control subjects (C1, 2, 5), all of whom had been tested in Phase II, were re-tested in this phase. A period of 11 months separated the Phase II and Phase III testing.

Rotation Code	*Amplitude of "Ramp Phase"/Limb Displacement*	*Duration of "Ramp Phase" of Rotation*	*Velocity of Ramp*
A	25°	100 ms	250°/sec
B	25°	50 ms	500°/sec
C	40°	100 ms	400°/sec
D	40°	200 ms	200°/sec

Table 3.1. Four Types of Step Rotations. Subjects were tested with each of the 4 types of step rotations at 0%, 50% , and 85% of MVC. All rotations had a "hold phase" of 400 ms and differed only in "ramp phase" parameters.

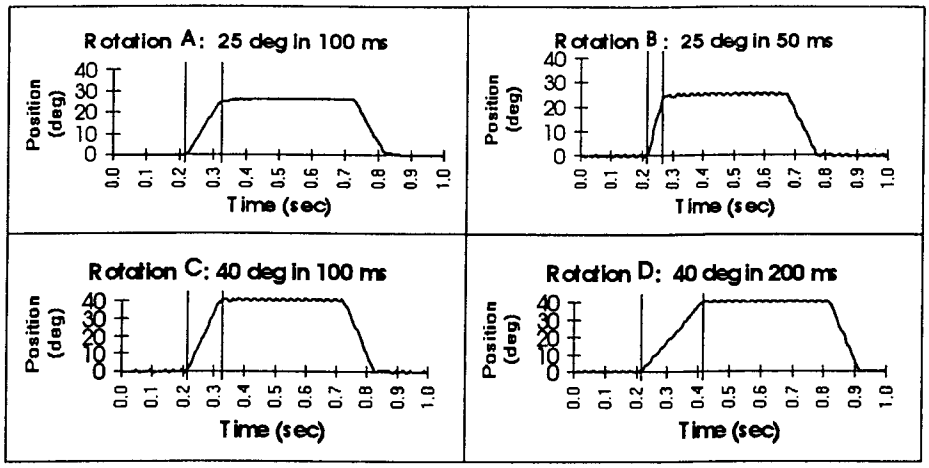

Figure 3.2. Four Types of Step Rotations. Four types of rotations that differed in "ramp phase" only were used in Phase III: (A) 25° in 100 ms, (B) 25° in 50 ms, (C) 40° in 100 ms, and (D) 40° in 200 ms. Note that the rotations were identical except for the "ramp phase" which is indicated by vertical lines. The oscillations that can been seen in these traces were due to high feedback gain to the motor that was necessary to produce the sharp onsets of rotation. One block of trials was delivered at 0%, 50%, and 85% of MVC. A block of trials consisted of 3 rotation trials and 1 catch trial.

Of the 5 patients and 3 controls who were tested with the new rotations, previous testing in Phase II revealed 1 case of bilateral BAW (P6), 1 case of unilateral BAW (P8), and 6 normal responses (C1, C2, C5, P5, P9, P10). Our objective was to identify a single test that reproduced bilateral BAW in P6, reproduced normal responses in control subjects, and detected bilateral BAW in the 4 patients who tested normally in one or both limbs. Furthermore, it was expected that one rotation might emerge as capable of producing the most robust cases of BAW.

Responses to changes in "ramp phase" of rotations

With the increase to 4 types of rotations, the following results were obtained:

a. Four Phase II diagnoses of NO BAW bilaterally remained unchanged (P10, C1, C2, C5).

b. One Phase II diagnosis of NO BAW became bilateral BAW (P9).

c. One Phase II diagnosis of NO BAW became unilateral BAW (P5).

d. One Phase II diagnosis of unilateral BAW became bilateral BAW (P8).

e. One Phase II diagnosis of bilateral BAW became unilateral BAW (P6).

In summary, 4 subjects were unaffected by the changes to the test protocol and never showed BAW; 3 subjects were more prone to show BAW with the new test rotations; and one subject was less prone to show BAW with the new protocol. Results are summarized in Table 3.2.

Subject Code	Sex	Sweeting Diagnosis Manual	Diagnosis Phase II 25°/100 ms	Diagnosis Phase III Rotation A 25°/100 ms		Rotation B 25°/50 ms		Rotation C 40°/100 ms		Rotation D 40°/200 ms		Change in Detection of BAW
				R	L	R	L	R	L	R	L	
P5	F	BAW	No BAW	No BAW	BAW (1/3)	No BAW	No BAW	No BAW	No BAW	No BAW	BAW (1/3)*	Increase
P6	F	BAW	BAW Bilat	BAW (2/3)*	No BAW	BAW (1/3)	No BAW	No BAW	No BAW	No BAW	No BAW	Decrease
P8	M	BAW	BAW (R)	No BAW	No BAW	BAW (1/3)	No BAW	No BAW	No BAW	BAW (1/3)*	BAW (2/3)*	Increase
P9	M	BAW	No BAW	No BAW	BAW (1/3)	No BAW	BAW (2/3)	BAW (3/3)*	BAW (2/3)	BAW (1/3)	No BAW	Increase
P10	M	BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No Change
C1	M	NA	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No Change
C2	F	BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No Change
C5	F	NA	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No BAW	No Change

Table 3.2. Summary of Changes in Diagnosis from Phase II to Phase III with New Test Rotations. Numbers in parentheses indicate the number of times out of the number of rotations delivered that BAW was detected. For example in phase III, P6 displayed BAW in 1 out of 3 trials for rotation B. The * indicates most robust examples of BAW. The last column indicates whether there was an increase, decrease, or no change in the detection of BAW with the introduction of the new test rotations.

Effectiveness of individual rotations for detecting BAW

Rotation A: The step rotation that was 25° in amplitude and 100 ms in duration closely resembled the rotation that was used in Phase II where it identified 4 out of 11 potential cases of BAW. In Phase III, it was capable of detecting 3 unilateral cases of BAW (P5, P6, P9). If it had been the only rotation used, 1 out of 4 cases of BAW would have gone undetected (P8). If this rotation had been omitted, no cases of BAW would have been overlooked, but one "best example" would have gone undetected.

Rotation B: The step rotation that was 25° in amplitude and 50 ms in duration was capable of detecting 3 unilateral cases of BAW (P6, P8, P9). If given alone, it would have missed 1 out of 4 cases of BAW (P5). This rotation did not detect any cases of BAW that were not detected with one of the other tests.

Rotation C: The step rotation that was 40° in amplitude and 100 ms in duration was capable of detecting 1 bilateral case of BAW (P9). If it had been the only rotation delivered, 3 out of 4 patients who displayed BAW with other rotations would have gone undetected. However, if this rotation had never been administered, the single case that it detected (P9) would have been detected by one of the other 3 tests. This rotation provided the fewest BAW diagnoses and did not provide any information that had not been provided

with other tests.

Rotation D: The step rotation that was 40° in amplitude and 200 ms in duration was capable of detecting 3 out of 4 cases of BAW, 1 bilateral and 2 unilateral (P5, P8, P9). It also produced the best examples of BAW in 2 out of 3 subjects (or 3 out of 4 limbs) which exceeded any other rotation. If given alone, it would have missed 1 unilateral case of BAW (P6).

Using four types of step rotations in Phase III, detection of BAW was enhanced in 3 patients, decreased in 1 patient, unchanged in 1 patient, and unchanged in 3 controls. The 3 control subjects who never showed BAW in either Phase II or Phase III support the hypothesis that BAW does not occur in control subjects. However, these control subjects were from an athletic, student control group and may not have been well matched to the patient group. The fact that we never observed BAW in P10 may indicate that either our methods are not sensitive enough to detect subtle cases of BAW or that Sweeting's manual test for BAW produces false positives. The 3 cases in which detection of BAW increased or decreased in Phase III are of greatest interest to us and are discussed in the paragraphs to follow.

Patient 5, who was diagnosed with NO BAW in Phase II, was diagnosed with unilateral BAW in Phase III when rotations A and D were applied. Because rotation A was almost identical to the rotation used in Phase II, it is

unclear why this rotation would produce BAW in the second test session and not the first. Perhaps the subject's physical condition changed from one session to the next, or the subject's level of motivation differed on the two occasions. Likewise, P9 who went from NO BAW in Phase II to bilateral BAW in Phase III, displayed BAW on the left with rotations A, B and C. On the right, this subject displayed BAW only with the larger rotations, C and D. Again, it is curious as to why rotation A elicited BAW on the left in Phase III, but a nearly identical rotation in Phase II did not. Patient 8 went from unilateral BAW in Phase II to bilateral BAW in Phase III but never displayed BAW in either arm with rotation A in Phase III. Instead, BAW was revealed only with rotations B and D.

Also of interest is P6 who went from bilateral BAW to unilateral BAW. In Phase III, rotations A and B were capable of eliciting BAW in the left but no rotation, not even rotation A which was similar to the one used in Phase II, detected BAW on the right. One possible explanation for the loss of detection of BAW is that this subject produced less pronation torque in Phase III than she did in Phase II, despite the fact that she was instructed to produce a strong MVC. If BAW only becomes apparent when muscles are activated at very high levels of force, then slightly less effort resulting in a smaller MVC could contribute to the decrease in detection. It is also possible that the subject's condition improved or that she became better at resisting the rotations.

The cases in which enhanced detection of BAW corresponds with the

addition of a new rotation suggests that the new rotation may have contributed to the improved ability to detect BAW. However, the cases in which enhanced detection was associated with the same rotation used in Phase II but did not detect BAW suggests that the changes in diagnosis may be due to changes in patient conditions, the inconsistent occurrence of the disorder, or subject motivation.

In summary, nearly identical conditions did not always produce the same responses in the same subjects on different test occasions. For example, rotation A which was nearly the same rotation that was used in Phase II, failed to produce the same responses in Phases II and III. This result suggests that detection of BAW may not depend on the type of rotation that is used as long as the rotation meets minimum criteria for amplitude, velocity, and duration. It also suggests that the condition may be labile, changing from test session to test session. Nearly one year passed before the subjects were re-tested, so it is possible that their condition could have changed during that time. However, patients who were tested in Phase III were under the care of Sweeting and had not fully recovered from the disorder for which they initially sought medical attention, suggesting that they were not fully recovered. Another important finding that emerged from Phase III was that the ability to detect BAW increased when the number of rotations delivered to each subject increased. The more opportunities that the subject was given to show BAW, the more likely it

was that BAW was detected.

Our results suggest that detection of BAW may be influenced by factors other than the rotation parameters. Such parameters may include level of background torque, the instructions to the subject, the number of repetitions of the test protocol, the subject's level of motivation, and the patient's state of health and/or recovery. One rotation did not emerge as superior to others for detecting BAW in all subjects. However, rotation D (40° on 200 ms) detected one more case of BAW than any other rotation and detected the greatest number of "best cases" of BAW. Based on these findings, we selected Rotation D for subsequent testing. We also concluded that our protocol should include multiple repetitions of the rotation to ensure optimal detection.

Qualitatively different rotations

To determine whether a type of rotation other than a single step rotation was more effective for identifying BAW, two different types of rotations were administered to several subjects. (See Table 3.3 for list of subjects.) Sweeting reported that if BAW was not detected with a single rotation, it may become apparent with multiple rotations performed in quick succession. In this phase, we attempted to replicate Sweeting's results by introducing triple step and triple stair-step rotations. A description of each follows, and examples can be found in Figure 3.3.

Triple Step Rotation

This rotation consisted of 3 identical step rotations delivered in quick succession. Each of the three rotations had a "ramp phase" that was approximately 40° in amplitude and 250 ms in duration and a "hold phase" that was 400 ms in duration. After the "hold phase", the handle returned to the initial position. Each of the three rotations was separated by a 100 ms pause. The duration of one trial was 3 seconds. Subjects were expected to maintain the specified level of torque for the duration of the trial, regardless of the rotations of the handle.

Triple Stair-Step Rotation

This rotation consisted of 3 consecutive step rotations, each displacing the limb further than the previous. The system was designed to displace the limb either 10° in 50 ms or 20° in 100 ms after which there was a 400 ms "hold phase". Because of an imperfectly servo-controlled motor, actual displacements and velocities deviated slightly from these ideal values. After the "hold phase", the limb did not return to the initial position; instead, it was displaced by 2 more rotations resulting in a 30-40° total displacement. At the end of all 3 rotations, the handle returned to the start position. The total duration of these trials varied from 1.8-2.0 seconds, depending on the total limb displacement. Subjects were expected to maintain the specified level of effort for the duration

of the trial, regardless of the rotations of the handle.

All rotations started from a wrist position of 20° of pronation. Many subjects that were tested in this phase did so in conjunction with evaluating the effects of limb position on BAW. If BAW was detected in a subject, the limb position was noted in Table 3.3. Refer to Phase III *Changes to limb position* for details.

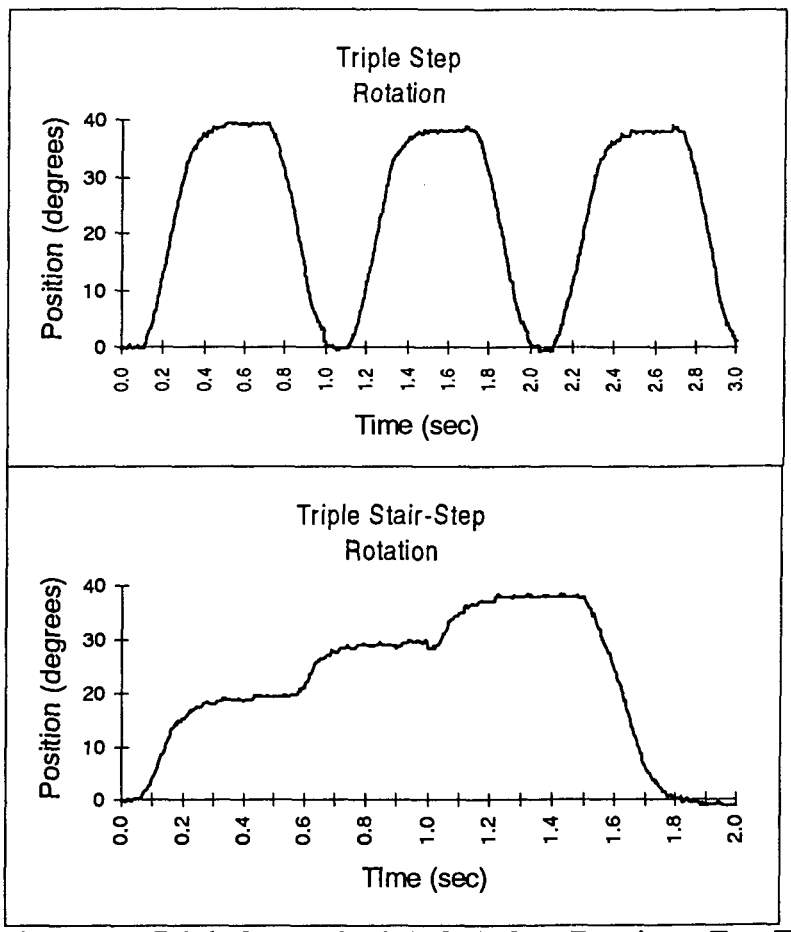

Figure 3.3. Triple Step and Triple Stair-Step Rotations. Top: Triple Step Rotation consisting of 3 identical rotations that were approximately 40° in amplitude and 200 ms in duration. Bottom: Triple Stair-Step Rotation consisting of 3 consecutive handle rotations that were approximately 20° in 100 ms, 10° in 50 ms, and 10° in 50 ms.

Effectiveness of triple step and stair-step rotations for detecting BAW

Triple Step Rotation: The triple step rotation was tested on three control subjects (C1, C2 and C14) and 1 patient (P12). It elicited unilateral BAW in C14 and P12. The control subjects on whom it was tested reported that the rotations were too long and too vigorous to resist repeatedly. Subjects complained of hand and muscle soreness, and, in one case, finger numbness (C14). Despite good motivation and verbal cueing, even subjects who did not break away had difficulty maintaining the specified level of effort for the duration of the trial and often displayed a gradual decline in pronation torque before the trial was complete. This was probably due to muscle fatigue caused by the long duration of the trial (3 sec) and insufficient time between rotations to recover from the previous rotation and prepare for the next rotation. It was concluded that this type of rotation was very aggressive and might be difficult for subjects to perform repeatedly. Therefore, despite the fact that it elicited BAW in one patient, this rotation was discarded.

Triple Stair-Step Rotation: The triple stair-step rotation was tested on 3 control subjects and 4 patients (C2, C14, C15, P1, P6, P8, and P12). In control subjects it never elicited BAW, but produced results similar the triple step rotation with respect to discomfort and gradually declining torque.

This rotation elicited unilateral BAW in 2 out of 4 patients (P6 and P8).

Both of these patients had been tested previously, once in Phase II and once in a different portion of Phase III. (See Table 3.2.) Interestingly, both subjects displayed a less robust BAW response with the triple stair-step rotation than they had in previous testing with single step rotations. This result may have been due to the ineffectiveness of the triple stair-step rotation for detecting BAW. Perhaps, the amplitude of each rotation was too small to elicit BAW. Alternatively, with repeated testing, subjects may have become better at performing the tests, or the subject's condition may have changed over time. This rotation was discarded because there was no evidence that it was superior to single step rotations for detecting BAW.

Subject Code	*Sex*	*Triple Step Rotation*	*Triple Stair-Step Rotation*
C 1	M	NO BAW	NO BAW
C 2	F	NO BAW	NO BAW
C 14	M	BAW Right (elbow flexed and extended)	NO BAW
C 15	M	Not Tested	NO BAW
P 1	M	Not Tested	NO BAW
P 6	F	Not Tested	BAW Right (elbow extended)
P 8	M	Not Tested	BAW Left (elbow flexed)
P 12	M	BAW Right (elbow flexed and extended)	NO BAW

Table 3.3. Effectiveness of Triple Step and Triple Stair-Step Rotations for detecting BAW. In some cases, these rotations were tested in conjunction with evaluating the effectiveness of different limb positions on detection of BAW. Information in parentheses indicates the limb position in which BAW was elicited.

Changes to limb position

The effect of elbow and shoulder position on detection of BAW was evaluated. One possible explanation for BAW is that, instead of being an involuntary reduction of pronation torque caused by deactivation of the PT, BAW may be caused by an increase in supination torque caused by activation of BI, either voluntarily or involuntarily. To address this issue, attempts were made to reduce the ability of BI to produce supination torque by modifying shoulder and elbow positions. Extended and flexed elbow positions were evaluated on the basis of their ability to generate high levels of pronation torque, to minimize supination torque, to optimize the ratio of pronation to supination torque, and to detect BAW.

The two arm positions that were evaluated are described below:

A. Flexed elbow: Elbow flexed to 90°, shoulder neutral in all planes, upper arm secured to torso with Velcro strap, forearm resting on rigid arm rest. This position placed the PT and BI in a relatively shortened position across the elbow and placed the BI in a relatively lengthened position across the shoulder.

B. Extended elbow: Elbow positioned in full extension, shoulder positioned at 90 ° of flexion and neutral with respect to internal/external rotation and abduction/adduction. Forearm, elbow, and half of upper arm supported on a rigid arm rest and secured in place with a Velcro strap to help

eliminate substitution from the shoulder. Compared to the flexed elbow position, this position lengthened the PT and BI across the elbow and shortened the BI across the shoulder.

Nine control subjects and 4 patients were tested in each of the two positions for maximum pronation torque, maximum supination torque, and presence or absence of BAW. In many cases, the effect of limb position on BAW was tested in conjunction with the triple step and stair-step rotations. These rotations resembled the ones depicted in Fig. 3.2. In other cases, single step rotations were used. These rotations varied in amplitude from 10° to 54° due to calibration errors during data collection. The last column in Table 3.4 contains information about the type of rotation that was used for each subject. Results are summarized in Table 3.4.

Subject Code	*Sex*	*Pronation MVC in Nm*		*Supination MVC in Nm*		*Ratio of Pronation to Supination Torque*		*Presence of BAW*		*Type of Rotation*
		Flexed	Extended	Flexed	Extended	Flexed	Extended	Flexed	Extended	
C1 Right	M	6.5*	8.7	8.2	1.6	0.80	5.44	No BAW	No BAW	single step 25 °/ 100ms
C2 Right	F	5.2*	5.6	NA	NA	NA	NA	No BAW	No BAW	single step 54 °/ 200ms
C2 Left	F	5.2	6.9*	NA	NA	NA	NA	No BAW	No BAW	single step 54 °/ 200ms
C9 Right	M	7.4*	11.5	NA	NA	NA	NA	NA	NA	NA
C10 Right	F	7.9*	7.4	5.9	1.5	1.34	4.93	No BAW	BAW (1/3)	single step 15 °/ 100ms
C11 Right	M	5.6*	9.2	NA	NA	NA	NA	No BAW	No BAW	single step 54 °/ 200ms
C11 Left	M	7.5	10.1*	NA	NA	NA	NA	No BAW	BAW (2/3)	single step 54 °/ 200ms
C12 Right	F	5.3	4.8*	NA	NA	NA	NA	No BAW	BAW (3/3)	single step 54 °/ 200ms
C12 Left	F	4.8*	3.8	NA	NA	NA	NA	No BAW	BAW (3/3)	single step 54 °/ 200ms
C13 Right	M	10.1*	15.3	NA	NA	NA	NA	BAW (1/3)	BAW ((3/3)	single step 54 °/ 200ms
C13 Left	M	7.6	11.5*	NA	NA	NA	NA	BAW (1/3)	BAW (2/3)	single step 54 °/ 200ms
C14 Right	M	8.9*	9.2	NA	NA	NA	NA	No BAW BAW (2/3)	BAW (2/3) BAW (2/3)	single step 40°/200 ms triple step
C14 Left	M	8.9*	10	NA	NA	NA	NA	No BAW No BAW	No BAW No BAW	single step 40°/200 ms triple step
C15 Right	M	8.5	6.6*	9.1	4.3	0.93	1.53	No BAW	No BAW	triple stair-step
P1 Right	M	11.3*	12.1	12.4	7.5	0.9	1.6	No BAW	No BAW	triple stair-step
P1 Left	M	12.5	12.9*	11.1	6	1.1	2.2	No BAW	No BAW	triple stair-step
P6 Right	F	2.5*	2.4	2.8	2.2	0.9	1.1	No BAW	No BAW	triple stair-step
P6 Left	F	2	2.6*	2.7	1.9	0.7	1.4	No BAW	BAW (1/3)	triple stair-step
P8 Right	M	10.7*	9.2	10.6	7.6	1.0	1.2	No BAW	No BAW	triple stair-step
P8 Left	M	11	12.7*	10.1	5.6	1.1	2.3	BAW (1/3)	No BAW	triple stair-step
P12 Right	M	7	9.6*	NA	6.9	NA	1.4	No BAW BAW (2/2)	No BAW BAW (1/3)	triple stair-step triple step
P12 Left	M	7.6*	6.6	NA	NA	NA	NA	No BAW No BAW	No BAW No BAW	triple stair-step triple step

Table 3.4. Relationship between limb position, torque output, and detection of BAW. NA indicates that data was not collected or not applicable. Asterisks indicates the MVC trial that was performed first.

Pronation torque

The ability to produce maximum voluntary pronation torque in the extended and flexed elbow positions was evaluated in all 13 subjects. The order of the MVC trials (extended vs. flexed) alternated between arms. For example, if the flexed elbow position was tested first on the left arm, then the extended elbow position was tested first on the right arm. This order alternated with each subject.

The majority of the controls (6 out of 9) produced a larger pronation MVC with the elbow extended than with the elbow flexed, regardless of which position was tested first. The other 3 controls generated a larger pronation MVC with the elbow flexed, regardless of which position was tested first. For control subjects who were tested bilaterally, the strongest elbow position was always the same for both arms.

Of the patients that were tested, one displayed a mixed response. This subject displayed his strongest MVC on each arm in a different limb position. The other 3 patients' responses depended on which position was tested first. The elbow position that was tested first always resulted in the strongest MVC. This result suggested that patients may be more vulnerable than control subjects to muscle fatigue, and it interfered with our ability to determine which limb position in patients was capable of producing the strongest MVC.

Supination torque

The ability to generate supination torque with the elbow flexed and extended was evaluated in 3 control subjects and 3 patients. In all 6 cases, subjects produced more supination torque with the elbow flexed than with the elbow extended. This effect was robust with supination torque being up to 5x larger with the elbow flexed.

Ratio of pronation to supination torque

In all cases, the most favorable ratio of pronation to supination torque existed with the elbow extended. On average, in control subjects the ratio of pronation to supination torque was over 4 times greater with the elbow extended than with the elbow flexed, and in patients it was 1.6 times greater. This relationship of pronation and supination torque to elbow position has been reported previously. Consistent with our findings, Winters and Kleweno (1993) reported that BI activity was minimized when the elbow was extended and the shoulder was flexed

In short, these data indicated that the extended elbow position was most effective for enhancing pronation MVC, reducing supination MVC, and optimizing the ratio between pronation and supination torque.

Ability to detect BAW

Of the 12 subjects who were tested for BAW in the elbow extended and flexed positions, 2 displayed bilateral BAW (C12, C13), and 6 displayed unilateral BAW (C10, C11, C14, P6, P8, P12). In 3 cases, (C13, C14, P12) BAW occurred in both the flexed and extended elbow positions. In one case (P8) BAW only occurred when the elbow was flexed. In most of cases, BAW only occurred if the elbow was extended (C10, C11, C12, P6).

For many subjects, it appeared that elbow position did not affect detection of BAW because BAW was detected regardless of limb position. For other subjects, however, elbow position was important. We found that, in the majority of the subjects for whom elbow position mattered, the extended elbow position was more effective than the flexed elbow position for identifying BAW. It is difficult to know whether the cases of BAW that were detected in controls in the extended elbow position were false positives because the test for BAW lacks a "gold standard". The only thing that can be said with certainty is that, in subjects whose response depended on limb position, the extended elbow position identified more cases of BAW than the flexed elbow position.

In conclusion, the extended elbow position was chosen over the flexed position for its ability to generate higher levels of pronation MVC in most cases where fatigue was not an issue, to generate smaller levels of supination MVC, to

optimize the ratio of pronation to supination torque, and to detect more cases of BAW than the flexed elbow position. One possible pitfall of this position is that it may generate false positives in control subjects. There is no way of determining this because our test lacks a "gold standard". All but one control subject who displayed BAW did so with a step rotation that was 54° in amplitude. This rotation was administered inadvertently when a calibration error occurred in the apparatus. It may have been the large amplitude or combination of amplitude and elbow position that resulted in BAW in control subjects.

Background torque

An objective of Phase III was to identify the minimum level of background pronation torque at which BAW could be detected. In Phases I and II, BAW was detected in trials where subjects were asked to produce and hold 100% of MVC, and less severe cases were occasionally detected in the same subjects at 50% of MVC. This observation suggested that it is not necessary for subjects to produce 100% of MVC in order to detect BAW. Furthermore, it is known that human subjects are not usually capable of generating true maximum voluntary contraction, so it has been assumed that subjects were producing torques close to MVC but not at 100% MVC.

After completing MVC trials to determine maximum voluntary

pronation torque, subjects were tested for BAW at 80%, 85% and 90% of MVC. At 90% of MVC, subjects tired very quickly and often could not complete the entire protocol. Also at 90% of MVC, control subjects who had never before shown BAW, began displaying signs of marginal BAW (C1, C14). At 80% of MVC, one subject (P6) who had displayed BAW in Phase II at 100% of MVC, failed to do so. This result may have been due to the lower level of torque because it seems that BAW may become evident only when muscles are producing high levels of force. The value of 85% of MVC was chosen for use in subsequent experiments because subjects were adequately challenged at this level without experiencing the fatigue that was present at 90% of MVC. Subjects were as likely to display BAW at 85% of MVC in Phase III as they were in Phase II at 100% MVC.

Handle position

The starting position of the forearm with respect to pronation/supination angle was determined by reviewing the literature on force-angle relationships and considering subject safety. Isometric pronation torque is largest at joint angles from 30-60° of supination (Klewona and Winters, 1993; Caldwell and VanLeemputte, 1991). It is unclear, however, the extent to which passive properties contribute to torque generation in these positions. In this study, it was not feasible to position the limb in 30-60° of supination to start because the limb was rotated an additional 40° in the same direction. This combination of

start position and limb displacement would place the limb in 70-100° of supination at the end of the rotation, which approaches end range of motion and moves the limb away from its strongest position. Instead, 20° of pronation, a position that is less favorable for generating isometric pronation torque, was chosen as the start position because it allows the limb to move into a "stronger" position as it is rotated and remains within a safe range of motion. This starting position, used in conjunction with the 40° displacement, resulted in nearly the same end position as the Phase II step rotations that were 25° in amplitude but started at 0° of pronation/supination.

Catch trials and mini-rotation trials

No subjects displayed BAW in response to catch trials or mini-rotation trials. These data indicate that even subjects with BAW had adequate endurance to maintain the specified level of torque for the duration of the trial and that subjects did not have a tendency to shut-down muscle activity in anticipation of rotations.

Test/Re-test

An important finding of Phase III was that patients who were tested on many occasions became less likely to display BAW. For example, P8 and P6 went from bilateral BAW in Phase II to unilateral BAW in Phase III. Both subjects displayed less frequent and less severe cases of BAW in Phase III

compared to Phase II. Perhaps, their condition improved over time or they learned to perform the test properly. Although, an improvement in their condition is questionable because they were still seeing Sweeting for their musculoskeletal injuries at the time of Phase III testing. It is also possible that subjects became better at resisting the rotation or learned how to respond appropriately. If the second explanation was correct, it would suggest that BAW may be under voluntary control and may respond to training. This issue of repeatability will be addressed in the reliability portion of Phase IV.

Diverse control subjects

An important finding that emerged from this phase was that, as the control population became more diverse, a higher prevalence of BAW in control subjects was detected. This result could have been due to the new tests that were administered or may suggest that BAW is not limited to the population identified by Sweeting. A portion of Phase IV was designed to investigate this issue.

Discussion of Phase III Results

Based on data collected in Phase III, it became evident that no single test was clearly superior for detecting BAW in all subjects, but certain parameters seemed to be somewhat more effective than others. The test that was selected was a single step rotation with a "ramp phase" of 40° in amplitude and 200 ms

in duration and a "hold phase" of 400 ms. The level of background pronation torque was chosen to be 85% of MVC. The arm position that was selected was the elbow extended position. The chosen protocol consisted of several (2-6) blocks of trials, giving the subject ample opportunity to display BAW. One block of trials would consist of 3 rotation trials, one catch trial, and one mini-rotation trial.

An important finding that emerged from this phase was that BAW was detected in control subjects, which did not occur in previous phases. It seems that when the control population became more diverse, BAW was detected in control subjects. This result could have been due to the new tests that were administered or may have been because many of the new control subjects were not highly athletic, exceptionally motivated, and knowledgeable about the outcome of the test as they had been in previous phases. The new control subjects were more closely matched to patients with respect to age and activity level. This result suggests that BAW is not limited to the population identified by Sweeting. A portion of Phase IV was designed to investigate this issue.

Phase IV

Background for Phase IV

Phases I through III were dedicated to developing a test for detecting BAW and to obtaining preliminary data on prevalence of BAW in residents of the LFV. Quantitative examination of torque and EMG traces seemed to suggest that BAW was not caused by an abnormal stretch reflex, but the mechanisms underlying BAW remained unclear. In Phase IV, we used our newly developed test to examine 5 hypothesis that arose from the initial observations and the data that was collected thus far. Note: There is a Glossary of Terms to assist with this section located in the Appendix.

Goals of Phase IV

1. To develop an objective and quantitative method of classifying responses as normal or BAW.

2. To determine whether or not testing and classification techniques can reliably detect normal and BAW responses on two consecutive test sessions.

3. To determine whether or not BAW is more likely to be detected in injured people who live in the LFV than injured and non-injured people who live in the Lower Mainland of British Columbia but outside the LFV.

4. To quantify torque, timing, joint position, and EMG data of normal and BAW responses in order to describe and understand differences and similarities between normal and BAW responses and to identify which physiological mechanisms may contribute to the two conditions.

5. To estimate the latency at which BAW occurs and assess whether or not an inhibitory clasp-knife reflex may mediate BAW.

Methods for Phase IV

Test apparatus

The same test apparatus that was used in Phases II and III was used for this phase.

Test protocol

The test protocol that was used was the one that was selected from the Phase III optimization experiments. (See *Discussion of Phase III Results.*) All subjects were tested bilaterally. The right limb was always tested first.

MVC Trials

At the onset of every testing session, maximum voluntary isometric pronation and supination torque (MVC) were measured according to the same methods described in Phase III. The measurement of supination torque was added in this phase, but the methods were the same as those used to record pronation MVC. Supination MVC was always collected before pronation MVC.

Either 2 or 3 MVC trials that were 5 sec in duration were collected for each limb in each direction.

Rotation Trials

We administered large rotation trials to test for presence or absence of BAW, mini-rotation trials to evaluate the possible tendency to breakaway in anticipation of a rotation, and catch trials to evaluate subjects' ability to maintain the specified torque output for the duration of the trial.

Large rotation trials: Large step rotations were administered to detect presence or absence of BAW. The command signal to the motor for these trials specified a rotation that was 40° in amplitude, 200 ms in duration, and was held for 400 ms. In reality, the rotations were slightly slower than what was specified because the motor was not capable of overpowering subjects instantaneously. Hence, 90% of total handle displacement or 36° was achieved on average in 244 ms (std=5), and the "hold phase" of the rotation was approximately 350 ms after which the handle returned to the start position. All large rotations were 40° in amplitude, began from the position of 20° of pronation, and ended at 20° of supination. Figure 4.1 displays a typical position trace from a large rotation trial.

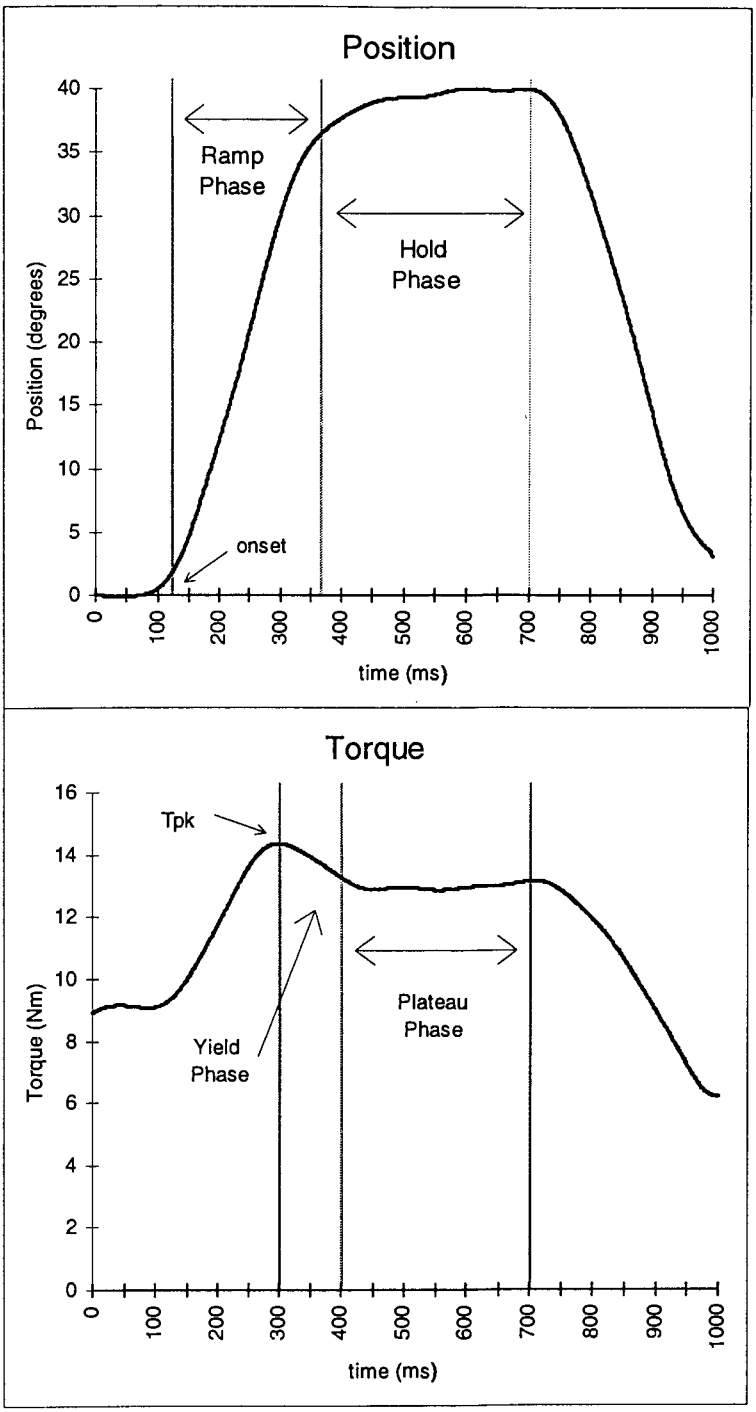

Figure 4.1. Components of Position and Torque Trace.

Mini-rotation trials: Small step rotation trials (4° in amplitude) were administered to subjects to detect inappropriate responses in anticipation of a rotation. The 4° displacements were 20 ms in duration and had a 400 ms "hold phase". The mini-rotation trials were large enough for subjects to feel, but were presumed to be too small to elicit BAW.

Catch trials: Trials in which the handle did not rotate were administered to evaluate subjects' ability to maintain a specified level of torque for the duration of the trial in the absence of a rotation.

Both mini-rotation trials and catch-trials were designed to identify people who had a tendency to display inappropriate responses in anticipation of the rotation.

Blocks of Trials

Rotation trials were administered in blocks of 5 trials, consisting of 3 large rotation trials, 1 catch trial, and 1 mini-rotation trial. Within a block, trials were administered in random order except that the first trial was always a large rotation trial. The instructions to the subject prior to each type of rotation were always the same. Neither the subject nor the examiner knew what type of rotation would be administered from trial to trial. All subjects performed at least 2 blocks of trials bilaterally, and some subjects were able to complete up to 5 blocks of trials.

Rest

To minimize fatigue, all subjects were given as much rest between trials as was necessary to maintain a high level of performance. Usually, subjects rested from 5-20 sec between trials, but this time was not recorded. If a subject performed more than 2 blocks, he/she often took a slightly longer rest (30-120 sec) before proceeding with the third block. Subjects were told that optimal performance was necessary to achieve proper test results; therefore, they should feel free to ask for rest breaks whenever necessary.

Instructions

The instructions to the subject were similar to the ones used in Phase III. Subjects were told that after they achieved the specified level of force, the handle may or may not turn. They were not told that trials were arranged in blocks or that there were a certain number of large, mini, and catch trials in each block. Verbal encouragement and coaching were used to help ensure that subjects gave a strong effort and were motivated to perform well.

Subjects tested

A total of 32 subjects were tested bilaterally. Thirty of these subjects returned on a different occasion for a second test session. All 32 subjects were, or had been until prevented by injury, involved in a fitness program that required moderate to vigorous exercise for at least 30 minutes, 3 times per week.

If a subject was unable to participate in physical activity because of injury, he/she was admitted as long as the period of sedentary lifestyle had not exceeded 3 months. Subjects were free of known neurologic, orthopedic or other disorders of the upper extremity or neck, and had no recent history (last 2 years) of such disorders. Subjects were free of systemic disorders of any origin that may have affected multiple muscle groups (i.e. uncontrolled diabetes, multiple sclerosis, muscular dystrophy). Subjects in Phase IV were non-overlapping with the subjects used in Phases I-III of the study and had not been tested with our apparatus on any previous occasion. Subjects were willing and able to follow instructions and to give informed consent. Subjects were unfamiliar to the examiner by name and by sight.

In addition to these global requirements, subjects from within and outside the LFV were selected as well as subjects with and without known musculoskeletal injury. Based on these criteria, at least 10 subjects from each of the following categories were selected.

Group 1: (Enriched Sample)

1. Subjects had an orthopedic disorder of the lower back or lower extremity for which they were **currently** being treated by Sweeting.

2. Subjects exhibited bilateral of unilateral BAW in PT when manually tested by Sweeting.

3. Subjects currently resided in the LFV and had for at least 2 years.

Group 2 (Semi-Enriched Sample)

1. Subjects had an orthopedic disorder of the lower back or lower extremity for which they were **currently** being treated by a physician or physiotherapist.

2. Subjects currently resided in the Lower Mainland of British Columbia but **outside** the LFV.

3. Subjects had never been treated by Sweeting.

Group 3: (Control Sample)

1. Subjects had no orthopedic disorders of the lower extremity or back and had not had such an injury for at least 6 months.

2. Subjects were not under the care of a physician or physiotherapist for any disorders of the neck, back, or extremities.

3. Subjects currently resided in the Lower Mainland of British Columbia but **outside** the LFV.

An equal number of male and female subjects were tested, but the proportion of male and female subjects in each group varied slightly. Subjects ranged in age from 16 to 33. The mean ages of Groups 1, 2 and 3 were 23, 23, and 20 respectively. There was no significant difference in the mean age of the subjects in each of the 3 groups ($p \leq 0.05$). There were a variety of injuries

represented in Groups 1 and 2, including back, hip, knee, and lower leg injuries. Subjects from the LFV resided in Abbottsford, Mission, Langley, Maple Ridge, and Clearbrook. Subjects from outside the LFV lived in Coquitlam, North Vancouver, Vancouver, Burnaby, and Surrey. Table 4.1 summarizes the information about each of the subjects that was tested.

Group 1 *Enriched*						*Group 2* *Semi-Enriched*						*Group 3* *Control*					
1st Test Code	Re-test Code	sex	age	injury	resi-dence	1st Test Code	Re-test Code	sex	age	injury	resi-dence	1st Test Code	Re-test Code	sex	age	injury	resi-dence
C61	P93	M	17	back	Abbts	C60	P92	M	23	leg	Coquit	C62	P90	F	26	none	Bnby
C64	P72	M	27	back	Mission	C67	P82	M	31	knee	N. Van	C63	P74	M	18	none	N. Van
C65	P80	M	22	knee	Lang	C69	P76	M	19	na	Van	C72	P83	F	18	none	Bnby
C68	P68	F	21	hip	Abbts	C70	P91	M	21	na	Bnby	C74	P87	F	21	none	Bnby
C73	P67	M	31	back	Abbts	C71	P60	M	19	back	Coquit	C76	P62	F	22	none	Bnby
C79	P69	M	19	back	Abbts	C75	P66	F	24	knee	Bnby	C78	P65	M	19	none	Bnby
C80	P63	F	33	hip	Lang	C77	P86	F	24	back	Van	C81	P64	F	17	none	Bnby
C83	P77	F	16	knee	M. Rdg	C82	P73	F	23	back	W. Van	C87	P84	M	22	none	Bnby
C88	P85	F	25	leg	Mission	C84	P71	M	20	back	Bnby	C89	P61	M	20	none	Bnby
C98	P98	M	23	knee	Clbk	C85	P78	F	33	back	Coquit	C93	P70	F	18	none	Van
						C86	P79	F	18	back	Surrey						
						C99	P75	F	22	na	Bnby						
Total		*10*				*Total*		*12*				*Total*		*10*			
males		*6*				*males*		*6*				*males*		*4*			
females		*4*				*females*		*6*				*females*		*6*			
Mean Age		*23*				*Mean Age*		*23*				*Mean Age*		*20*			

Table 4.1. Summary of Phase IV Data. Summary of information about all 32 subjects tested in Phase IV. Codes, sex, age, type of injury, and place of residence are outlined for each subject.

Subject recruitment

Subjects from Group 1 were patients of Sweeting and were recruited by him. When he encountered a patient who fit the selection criteria, he discussed with them the possibility of participating in the study, and with their permission, gave the prospective subject's name and phone number to the principal examiner. When the principal examiner contacted the prospective subjects by phone, they were told that participation was strictly voluntary and that it was unlikely that there would be any immediate benefit from participating.

Subjects from Groups 2 and 3 were recruited through written advertisements located throughout the university, at the university physiotherapy clinic, and in gyms throughout the Burnaby/Coquitlam area. Volunteers either left their name and phone number on a sign-up sheet to be contacted by the examiner or telephoned the examiner to volunteer for the study.

Blinding technique

Subject blinding

To avoid potential biasing of responses, subjects were told that the purpose of the experiment was to test muscle responses to stretch at high levels of force. They were not told that the goal of the research was to design a test for

muscle weakness. Subjects from Groups 2 and 3 were aware that they were selected partially on the basis of their residence and their injury status, but they were unaware of the relevance of these issues. Subjects from Group 1 probably had more knowledge about the goal of the experiment because Sweeting had manually tested them for BAW, and was likely to have associated their presence of BAW with a musculoskeletal disorder. The people from Group 1 knew that they had been asked to volunteer for the study because they were thought to have a unique disorder that may be responsible for their pain or injury. Nevertheless, they were given the same instructions and background as the other subjects.

Examiner blinding

To avoid potential biasing of responses on the part of the examiner, all subjects were unfamiliar to the experimenter. When subjects were recruited, a list of names and telephone numbers was given to the principal examiner. The principal examiner contacted prospective subjects by phone to screen them for admission to the study. If a suitable and willing candidate was identified, the subject's name and phone number were given to a research assistant for scheduling. When the "scheduler" had received several names of subjects, he contacted each subject to schedule an appointment and recorded the appointment on a calendar along with a subject code that concealed the identity of the subject from the principal examiner. Most weeks, the principal examiner

would speak to 5-25 prospective subjects and give 1-5 names to the "scheduler". It was not possible for the examiner to remember the voices of the subjects or to associate the code with a particular subject.

Upon arrival in the lab, the subject was greeted by the "scheduler", and the identity of each subject was withheld from the principal examiner. Subjects were asked to reveal no information about themselves and to refrain from conversation during testing. The examiner remained unaware of the subject classifications, even after all testing was complete. Identities were made known only after all subjects had been classified as BAW or normal.

As a result of these blinding techniques, subjects from Group 1 were single blind, and subjects from Groups 2 and 3 were double blind.

Test and re-test

All 32 subjects who were recruited for the study underwent one complete bilateral test session, referred to as the first test session. Of these 32 subjects, 30 returned for a second bilateral test session, referred to a the re-test session. The time between the first test and re-test session ranged from 3 hours to several weeks but was never long enough that a subject's injury status could have changed substantially. The re-test session was conducted in exactly the same way as the first test session with respect to subject blinding and instructions. At the re-test session, subjects were treated as if it was the first time that they had

undergone testing. At the time of re-test, the examiner had no knowledge of previous performance except that which could not be avoided by memory. During testing, it was not possible for the examiner to know whether or not a subject would be classified as BAW or normal. Each test session lasted approximately 1 hour.

Subject Coding Convention

Subjects were assigned a code letter and number at the time of admission to the study. The letter *C* was used for all subject codes in the first test session. The letter *P* was used for all subject codes in the re-test session. There was no relationship between the number that followed the letter *P* or *C* for the same subject who was tested 2 times. In this phase, the letters *C* and *P* did not indicate whether or not the subject was injured.

Data Collection

EMG

Bipolar surface electrodes (Sensormedics, 16 mm, silver-silver chloride) filled with conductive gel (Teca Corporation) were used to record EMG from BI and PT. After the skin was cleaned with alcohol, one pair of electrodes was placed over the BI muscle belly in a position that was best for recording EMG associated with wrist supination. The second pair of electrodes was positioned over the belly of the PT. Care was taken to place the electrodes in such a way that the signals were largest when either wrist pronation or supination were

performed and smallest when other movements, such as wrist and elbow flexion, were performed. Electrodes were held in place with adhesive tape. A wrist-band electrode that was fabricated in the laboratory was used for noise reduction and to ground the subjects. Analog EMG signals were amplified 1000 times and band pass filtered at 50-500 Hz using an AC differential amplifier (BAK electronics).

Torque

Torque was measured by semiconductor strain gauges (Entran) that were mounted on the shaft of the apparatus in such a way that they were most sensitive to pronation and supination torque and less sensitive to torque produced by movements in other planes such as wrist flexion/extension and elbow flexion/extension. The strain gauges were connected to a force bridge amplifier (BAK Electronics). Torque readings from the apparatus were manually calibrated several times over the course of 2 months to demonstrate the consistency of force bridge output.

Position

Handle position was monitored by a potentiometer mounted on the wheel of the apparatus that was connected to the handle by a rigid shaft (15 mm diameter steel with a length of 55 cm). The potentiometer was manually

calibrated at the onset of experimentation. The position of the wheel accurately reflected the handle position to within ±1 degree.

Trigger and Command Signals

Both the data acquisition trigger signal that initiated data collection and the command signal to the motor that controlled how and when to turn the handle were recorded as a timing reference for other events.

Data Recording and Digital Processing

Analog EMG, torque, position, command, and trigger signals were simultaneously recorded on-line with digital data acquisition software(DataSponge) sampled at 1000 samples/sec/channel. Digital smoothing was completed with Labview graphical programming software to obtain the measured and calculated variables that will be discussed in the sections to follow.

Variables measured

In order to understand the variables that were measured from the torque, position, and EMG traces, it is essential to define the characteristics of the position and torque traces. See Figures 4.1 and 4.2.

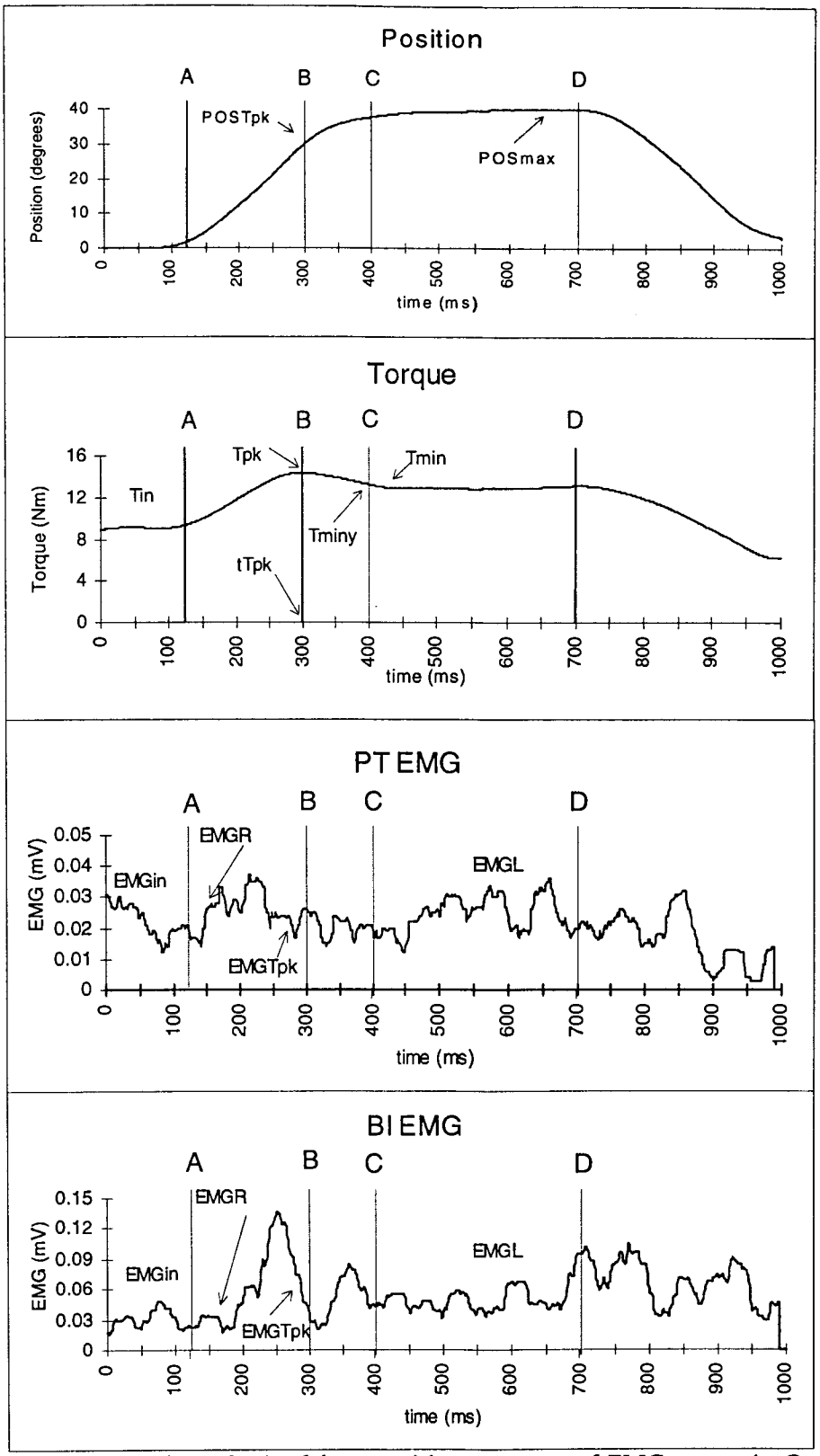

Figure 4.2. Values obtained from position, torque, and EMG traces. A. Onset of rotation, B. Time of T_{pk} and beginning of "yield phase", C. End of "yield phase" and beginning of "plateau phase", and D. End of "hold phase".

Position Trace (Figure 4.1, top.)

The position trace represented the rotation that was administered to each subject and consisted of a "ramp phase" and "hold phase".

Ramp phase of rotation: This portion of the position signal was defined as the period of time during which the motor was turning the handle to displace the limb 40°. It began at the onset of handle rotation and ended at 90% of total handle displacement (36°). The "ramp phase" duration was approximately 244 ms.

Hold Phase of rotation: This portion of the position signal was defined as the period of time during which the motor held the handle (and limb) in displaced position. It began at the end of the "ramp phase" and ended at t=700 ms from the beginning of data collection. The hold phase duration was approximately 350 ms.

Onset of Rotation: The onset of handle rotation was identified in the position trace as the point at which there was a substantial deviation in the position signal from baseline. The onset of handle rotation occurred gradually because the motor was not capable of overpowering subjects instantaneously. The following method was used to estimate the time of onset of handle rotation:

Position signals were recorded from the apparatus during large rotation trials in a no-load condition (i.e. no subject was grasping the handle). Five

traces were recorded for each of the 2 directions of handle rotation, clockwise and counter-clockwise. A line of best fit was created for the middle portion of the "ramp phase" of the position trace where velocity was presumed to be constant. The onset of rotation was defined as the point in time that the best fit line crossed the horizontal axis. In the clockwise direction, this value was 122 ms (std 1.0) from the beginning of data collection, and in the counter-clockwise direction, the value was 125 ms (std 1.4) from the beginning of data collection. The value of 123 ms (approximately midway between the 2 values) was selected as the definition of time of onset of rotation.

This value represented the earliest possible time that handle rotation may have begun. In the loaded condition, the actual time of onset of handle rotation may have been a few ms later because the motor required more time to overpower the subject.

Torque trace (Figure 4.1, bottom.)

The torque trace represented the change in subjects' torque output due to the rotation and had 3 main components that were characteristic of normal responses: peak torque, "yield phase", and "plateau phase".

Peak torque: Peak torque was defined as the single point at which torque reached maximum. It typically occurred near the end of the "ramp phase" of the position trace.

Yield phase: After achieving peak torque, there would typically be a small transient decrease in torque that was referred to as a "yield". The "yield phase" of the torque trace was defined as the 100 ms window from time of peak torque to 100 ms after time of peak torque.

Plateau phase: Following the "yield phase", torque traces typically displayed a plateau that, if BAW did not occur, lasted for the duration of the "hold phase". This phase was referred to as the "plateau phase" of the torque trace and was defined as the window of time from the end of the "yield phase" of torque to the end of the "hold phase" of the rotation or t=700 ms after the beginning of data collection.

See figure 4.1 for graphical a depiction of each of the phases of the position and torque traces.

Variables measured

All measured and calculated variables from the EMG, position, and torque traces are listed in Tables 4.2-4.6 (pp.100-104) along with their abbreviations, units of measure, methods of calculation/measurement, and data processing prior obtaining the value. Most variables are depicted graphically in Figure 4.2.

Torque: Four different torque values (T_{in}, T_{pk}, T_{miny}, T_{min}) were measured from each large rotation trial. All torque values were measured at a single

instant in time from the torque trace sampled at 1000 samples/sec and low pass filtered at 20 Hz. Two additional values (T_{incr} and $\%T_{incr}$) were calculated from the measured values. Values for $\%T_{incr}$ and T_{miny} were both normalized to T_{in} according to the formulas stated in Table 4.3 in order to account for differences in strength among subjects. See Tables 4.2 and 4.3 for descriptions of absolute and normalized torque values.

Rate of Change of Torque: Four values for rate of change of torque (RTavey, RTmaxy, RTavep, RTmaxp) were measured from the first derivative of the 20 Hz low pass filtered torque trace. Both the maximum and average rate of change of torque during the "yield" and "plateau" phases of the torque trace were measured. Each of the 4 values was normalized to T_{in} according the formula given in Table 4.3 to control for individual differences in strength. See Tables 4.2 and 4.3 for information about absolute and normalized rate of change of torque values.

Time and position: Four time and handle position variables (POS_{Tpk}, onset, t_{Tpk}, $t_{POS90\%}$) were measured from the appropriate traces after low pass filtering at 20 Hz to remove noise. All time values were presented with respect to onset of handle rotation. For descriptions of time and position variables see Table 4.4.

EMG : The root mean square (RMS) values of EMG for various time windows were calculated in Labview from the raw data according to the following formula:

Equation 4.1 Root Mean Square (RMS).

$$\sqrt{\frac{1}{n}\sum_{i=1}^{n} x_i^2}$$

where x_i is the i^{th} sample of EMG and n is the total number of EMG samples in the window.

The specific windows from which RMS values were obtained are shown in Table 4.5 and Figure 4.2 and include EMG_{max}, EMG_{in}, EMG, EMG_{Tpk}, and $EMG_{last100}$. EMG_{in} was normalized to EMG_{max} according to the formula presented in the Table 4.6. Other EMG values were presented as a net change from EMG_{in}, normalized to EMG_{max} as per the formula in the Table 4.6. See Tables 4.5 and 4.6 for information about absolute and normalized EMG values.

Spectral analysis of EMG: Power spectral analysis for PT and BI was conducted on each EMG trace for the 123 ms period prior to onset of handle rotation to determine whether or not fatigue occurred from the first to the sixth trial. The median frequency of the signal was calculated in Labview.

Statistical Analysis

Statistical analysis was done with BMDP Statistical software package. Differences were considered significant for $p \leq 0.05$.

Table 4.2. Torque Values.

Value	Abbreviation	Unit of Measure	Method of Measurement or Calculation	Processing of Data Prior to Obtaining Value
Initial Torque	T_{in}	Nm	Measured at a single point in time. 20 ms before onset of handle rotation.	Torque trace low pass filtered 20 Hz
Peak Torque	T_{pk}	Nm	Measured at a single point in time. Maximum value of torque for the entire trace.	Torque trace low pass filtered 20 Hz
Torque Increase	T_{incr}	Nm	Tpk-Tin	Torque trace low pass filtered 20 Hz
Minimum Torque during Yield	T_{miny}	Nm	Measured at a single point in time. Minimum value of torque during "yield phase".	Torque trace low pass filtered 20 Hz
Minimum Torque	T_{min}	Nm	Measured at a single point in time. Minimum value of torque for the entire trace.	Torque trace low pass filtered 20 Hz
Average Rate of Change of Torque during Yield	RTavey	Nm/sec	Average value for rate of change of torque during the "yield phase".	Torque trace low pass filtered 20 Hz then differentiated.
Maximum Rate of Change of Torque during Yield	RTmaxy	Nm/sec	Measured at a single point in time. Maximum value of rate of change of torque during the "yield phase".	Torque trace low pass filtered 20 Hz then differentiated.
Average Rate of Change of Torque during Plateau	RTavep	Nm/sec	Average value for rate of change of torque during the "plateau phase".	Torque trace low pass filtered 20 Hz then differentiated.
Maximum Rate of Change of Torque during Plateau	RTmaxp	Nm/sec	Measured at a single point in time. Maximum value of rate of change of torque during the "plateau phase".	Torque trace low pass filtered 20 Hz then differentiated.

Table 4.3. Normalized Torque Values.

Value	Abbreviation	Unit of Measure	Method of Measurement or Calculation	Processing of Data Prior to Obtaining Value
Percent Torque Increase	$\%T_{incr}$	% of T_{in}	Tincr/Tin*100.	Torque trace low pass filtered 20 Hz
Minimum Torque during Yield Normalized	T_{minyN}	% of T_{in}	Tminy/Tin.	Torque trace low pass filtered 20 Hz
Average Rate of Change of Torque during Yield Normalized	RTaveyN	1/sec	RTavey/Tin	Torque trace low pass filtered 20 Hz then differentiated.
Maximum Rate of Change of Torque during Yield Normalized	RtmaxyN	1/sec	RTmaxy/Tin	Torque trace low pass filtered 20 Hz then differentiated.
Average Rate of Change of Torque during Plateau Normalized	RTavepN	1/sec	RTavep/Tin	Torque trace low pass filtered 20 Hz then differentiated.
Maximum Rate of Change of Torque during Plateau Normalized	RtmaxpN	1/sec	RTmaxp/Tin	Torque trace low pass filtered 20 Hz then differentiated.

Table 4.4. Position and Timing Values.

Value	Abbreviation	Unit of Measure	Method of Measurement or Calculation	Processing of Data Prior to Obtaining Value
Onset of Handle Rotation	Onset	ms (from onset of data collection)	Ramp portion of position trace in no-load condition was fitted with line of best fit. Point in time that line of best fit crossed time axis.	Position trace sampled at 1000 samples/second.
Time of Peak Torque	t_{Tpk}	ms (from onset of handle rotation)	Single time value at which Tpk occurred.	Torque trace low pass filtered 20 Hz.
Handle Position at Peak Torque	POS_{Tpk}	degrees	Single point measured from position trace. Measured handle position at tTpk.	Position trace low pass filtered 20 Hz.
Time that Handle Reached 90% of Maximum Displacement	$t_{POS90\%}$	ms (from onset of handle rotation)	Single time value at which handle position reached 90% of maximum displacement.	Position trace low pass filtered 20 Hz.

Table 4.5. EMG Values.

Value	Abbreviation	Unit of Measure	Method of Measurement or Calculation	Processing of Data Prior to Obtaining Value
Maximum value of PT EMG	$PTEMG_{max}$	mV	Obtained from every pronation MVC trial. RMS value for PT EMG during 123 ms moving window. The RMS with the largest value was PTEMGmax.	Calculated from raw EMG data sampled at 1000 samples/sec.
Maximum value of BI EMG	$BIEMG_{max}$	mV	Obtained from every supination MVC trial. RMS value for BI EMG during 123 ms moving window. The RMS with the largest value was BIEMGmax.	Calculated from raw EMG data sampled at 1000 samples/sec.
Initial value of PT EMG	$PTEMG_{in}$	mV	Obtained from rotation trials. RMS value of PT EMG during 123 ms window. Window=onset of data collection to onset of handle rotation.	Calculated from raw EMG data sampled at 1000 samples/sec.
Initial value of BI EMG	$BIEMG_{in}$	mV	Obtained from rotation trials. RMS value of BI EMG during 123 ms window. Window=onset of data collection to onset of handle rotation.	Calculated from raw EMG data sampled at 1000 samples/sec.
Value of PT EMG during stretch reflex latency	$PTEMG_{R}$	mV	Obtained from rotation trials. RMS value of PT EMG during 60 ms window. Window=20 ms after onset of rotation to 80 ms after onset of rotation.	Calculated from raw EMG data sampled at 1000 samples/sec.
Value of PT EMG during stretch reflex latency	$BIEMG_{R}$	mV	Obtained from rotation trials. RMS value of BI EMG during 60 ms window. Window=20 ms after onset of rotation to 80 ms after onset of rotation.	Calculated from raw EMG data sampled at 1000 samples/sec.
Value of PT EMG prior to Tpk	$PTEMG_{Tpk}$	mV	Obtained from rotation trials. RMS value of PT EMG during 60 ms window. Window=20 ms prior to Tpk to 80 ms prior to Tpk.	Calculated from raw EMG data sampled at 1000 samples/sec.
Value of BI EMG prior to Tpk	$BIEMG_{Tpk}$	mV	Obtained from rotation trials. RMS value of BI EMG during 60 ms window. Window=20 ms prior to Tpk to 80 ms prior to Tpk.	Calculated from raw EMG data sampled at 1000 samples/sec.
Value of PT EMG during last 100 ms of "hold phase"	$PTEMG_{L}$	mV	Obtained from rotation trials. RMS value of PT EMG during 100 ms window. Window=100 ms prior to the end of the hold phase to the end of the hold phase.	Calculated from raw EMG data sampled at 1000 samples/sec.
Value of BI EMG during last 100 ms of "hold phase"	$BIEMG_{L}$	mV	Obtained from rotation trials. RMS value of BI EMG during 100 ms window. Window=100 ms prior to the end of the hold phase to the end of the hold phase.	Calculated from raw EMG data sampled at 1000 samples/sec.

Table 4.6. Normalized EMG Values.

Value	Abbreviation	Unit of Measure	Method of Measurement or Calculation	Processing of Data Prior to Obtaining Value
Initial value of EMG normalized to EMG_{max}.	$PTEMG_{inN}$ $BIEMG_{inN}$	% of PTEMGmax % of BIEMGmax	$PTEMG_{in}/PTEMG_{max}$ $BIEMG_{in}/BIEMG_{max}$	Calculated
Net value of EMG for various windows normalized to EMG_{max}	$PTEMG_{RN}$ --- $BIEMG_{RN}$ --- $PTEMG_{TpkN}$ --- $BIEMG_{TpkN}$ --- $PTEMG_{LN}$ --- $BIEMG_{LN}$	% of EMGmax	$(PTEMG_{R}-PTEMG_{in})/PTEMG_{max}$ --- $(BIEMG_{R}-BIEMG_{in})/BIEMG_{max}$ --- $(PTEMG_{Tpk}-PTEMG_{in})/PTEMG_{max}$ --- $(BIEMG_{Tpk}-BIEMG_{in})/BIEMG_{max}$ --- $(PTEMG_{L}-PTEMG_{in})/PTEMG_{max}$ --- $(BIEMG_{L}-BIEMG_{in})/BIEMG_{max}$	Calculated

Definition of BAW

BAW is a rapid and substantial decline in torque and muscle activation that can sometimes occur when muscle that is producing high force undergoes a sudden, unexpected stretch. In these experiments the motion of interest was wrist pronation, and the muscles that were monitored were PT and BI. BAW was defined as a substantial decline in pronation torque and activation of PT and BI that sometimes occurred when subjects were producing a high level of pronation torque and experienced a sudden, unexpected wrist supination that stretched PT.

Results of Phase IV

The data presented here were based on the results of the first and the re-test session of the entire sample of subjects. A total of 32 subjects underwent testing during the first test session, and 30 subjects returned to be re-tested. Two subjects were unable to return to participate in the re-test session.

Classification of Responses as Normal or BAW

A method for classifying responses to large rotation trials as normal or BAW was developed.

A total of 557 responses to large rotation trials were recorded in the first test session, and 505 responses to large rotations were recorded in the re-test session. All 1062 responses were classified as either normal or BAW based on

mechanical and electromyographical criteria that were established through analysis of all the responses from the first test session. Please refer to the definitions in the Methods section for abbreviations.

Mechanical Criteria: A value referred to as BAW Index was calculated from the torque trace of each response. Based on the initial definition of BAW , that it was a rapid decrease in torque and EMG, BAW Index was defined as $T_{min}/T_{in}*100$. If BAW Index exceeded 100, the minimum torque during the trial was always $\geq T_{in}$, and BAW was not present. If BAW Index was <100, torque during the "hold phase" of the rotation dropped below T_{in}, and BAW may have occurred. After visually inspecting the EMG and torque traces of a variety of responses from the first test session and comparing them to BAW Index, it became obvious that there was no clear division between BAW and normal responses; rather, there was a continuum of responses that ranged from BAW Index=-40 to BAW Index=318. The continuum of BAW Indices is represented by the frequency histograms that are shown in Figure 4.2A.

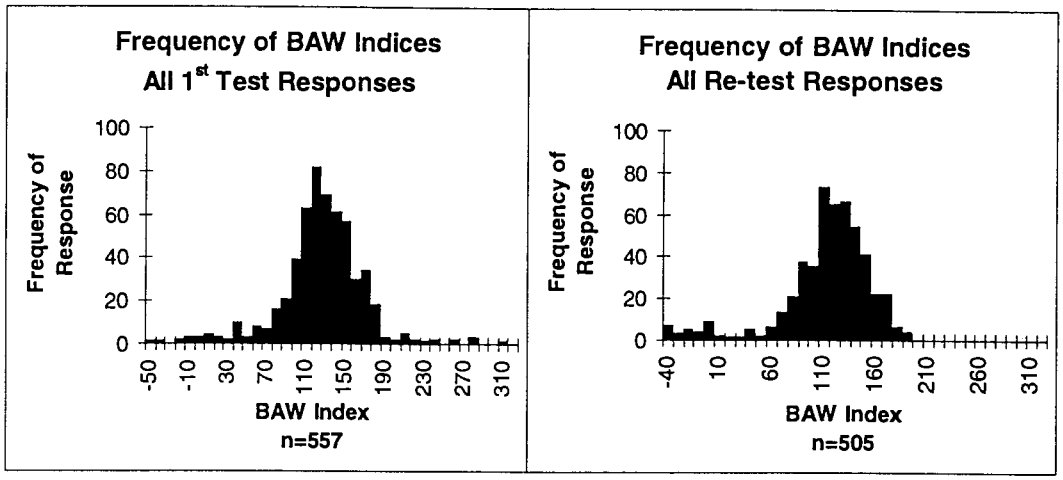

Figure 4.2A. In both the first and re-test sessions a continuum of BAW Indices was observed, as represented by the normal distribution of responses. There was no clear division between normal and BAW. In the re-test session, there were slightly more cases of BAW, and the histogram displays a rise in frequency of responses in the range of -40 to 10. These data may suggest that there was a tendency for the responses in the re-test session to be negatively skewed.

Hence, an arbitrary division between BAW and normal was drawn. It was decided that any response with BAW Index $\geq$ 80 did not display a rapid or substantial enough decline in torque or EMG to be considered BAW. Hence, these responses were classified as normal. Responses with BAW Index <80 were classified as BAW as long as electromyographic criteria were also met. There were 60 responses in the first test session that had BAW Index < 80.

Table 4.7. Mechanical Classification of BAW. (Based on values from torque trace.)

BAW Index = T_{min}/T_{in}*100	
BAW Index ≥80	Normal
BAW Index < 80	BAW (If EMG criteria also met.)

Electromyographical Criteria: PT and BI EMG were measured during the last 100 ms of the "hold phase" of the rotation ($PTEMG_{LN}$ and $BIEMG_{LN}$). Based on the definition of BAW, it was assumed that if BAW had occurred, PT and BI EMG during the last 100 ms of the "hold phase" would be substantially smaller than initial EMG (EMG_{in}). In most cases of BAW this assumption was valid, and responses with BAW Indices < 80 displayed values for PT and BI EMG in the last 100 ms of the "hold phase" that were smaller than EMG_{in}. In some cases, however, BAW Indices < 80 were associated with values for PT and BI EMG in the last 100 ms of the "hold phase" that were larger than EMG_{in}. In other cases, BAW was associated with values of $PTEMG_L$ that were larger than EMG_{in} while values for $BIEMG_L$ were smaller than EMG_{in}. Other examples of BAW displayed values for $PTEMG_L$ that were smaller than EMG_{in}; while values for $BIEMG_L$ were larger than EMG_{in}.

The four combinations of $PTEMG_L$ and $BIEMG_L$ that were associated with BAW Indices < 80 are depicted in Figure 4.3. Values for $PTEMG_L$ and $BIEMG_L$ were normalized according to the formula in Table 4.6 and were expressed as

the change in EMG as a percentage of EMG_{max}. Values that were > 0 indicated that EMG_{LN} was > EMG_{in}. Values < 0 indicated that EMG_{LN} was < EMG_{in}.

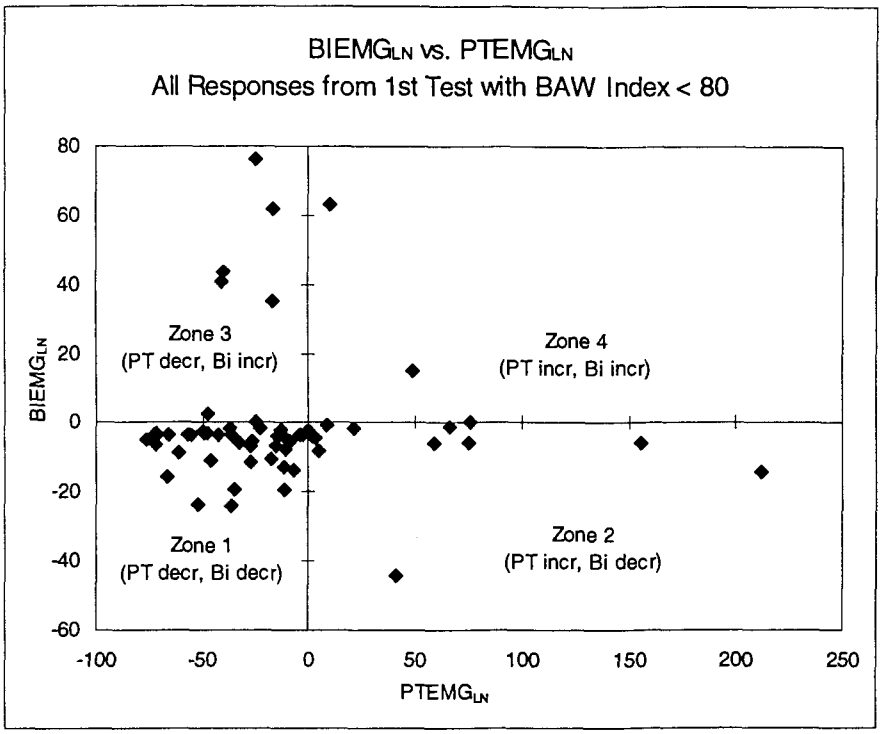

Figure 4.3. $PTEMG_{LN}$ vs. $BIEMG_{LN}$ for all first test responses with BAW Index <80. The majority of responses fell into Zone 3 in which PT and BI EMG_{LN} were < 0.

As predicted, most responses (38/60) were in Zone 1 in which $PTEMG_{LN}$ was < 0 and $BIEMG_{LN}$ was <0. Twelve responses fell into Zone 2 in which $PTEMG_{LN}$ was >0, while $BIEMG_{LN}$ was < 0. Seven responses fell into Zone 3 in which $PTEMG_{LN}$ was <0, while $BIEMG_{LN}$ was > 0. Only three responses fell into Zone 4 in which $PTEMG_{LN}$ and $BIEMG_{LN}$ were both >0.

The responses in Zone 4 were somewhat paradoxical in that both PT and BI EMG at the end of the "hold phase" were greater than EMG_{in}, but the mechanical classification criteria suggested BAW. On closer inspection of these

3 responses, it was observed that the BAW Indices for these trials were 73, 73, and 77, which was very close to the cut-off of 80 that would satisfy the mechanical criteria for normal. Because these responses marginally achieved the mechanical criteria for normal and did not fulfill the electromyographic definition of BAW, they were classified as normal.

Based on these observations from all of the first test responses and the prior definition of BAW, 4 categories of BAW were created: BAW Type 1, BAW Type 2, BAW Type 3, and BAW Type 4.

BAW Type 1: Responses in this category had BAW Index < 80 *and* displayed a decrease in PT and BI EMG below EMG_{in} in the last 100 ms of "hold phase". Responses in this category were considered the most robust cases of BAW.

BAW Type 2: Responses in this category had BAW Index < 80. Responses displayed an increase in PT EMG and a decrease in BI EMG compared to EMG_{in} in the last 100 ms of "hold phase". BAW Type 2 was considered to be a moderately robust case of BAW.

BAW Type 3: Responses in this category had BAW Index < 80. Responses displayed a decrease in PT EMG and an increase in BI EMG compared to EMG_{in} in the last 100 ms of "hold phase". BAW Type 3 was considered to be a mild case of BAW.

BAW Type 4: Responses in this category had BAW Index << 80. Responses displayed an increase in both PT and BI EMG compared to EMG_{in} in the last 100 ms of "hold phase".

Each response to large rotation trials from the first and re-test test session was classified as either normal, BAW Type 1, BAW Type 2, BAW Type 3,or BAW Type 4. Results are displayed in Table 4.0. A limb was classified as positive for BAW if **at least one trial** met the criteria for any of the 4 Types of BAW. Each limb was classified as BAW Type 1-4, according to the most robust case of BAW that was observed in that limb at least once. Subjects were classified as unilateral BAW if only one limb displayed BAW and bilateral BAW if both limbs displayed BAW. In some cases of bilateral BAW, both limbs did not show the same type of BAW; therefore, limbs were individually classified according to BAW type. See Tables 4.2 and 4.3 for classification of responses and subjects.

There were two BAW limbs (C89R and C81R) that displayed BAW in response to the 4° rotation trials that were administered to detect subjects who had a tendency to respond inappropriately in anticipation of a rotation. Because these limbs "gave away" in response to a rotation that should not have caused BAW, they were not considered true cases of BAW and were classified as normal. These 2 limbs were included in the reliability and prevalence data but were omitted from the description and analysis of normal and BAW trials. Both

subjects were from the group 3 (control subjects). One was male, and the other was female.

Table 4.8. All BAW Responses from first Test Session.
Total number of BAW responses per category.

BAW Type 1		BAW Type 2		BAW Type 3		BAW Type 4	
Subject Code	BAW Index	Subject Code	BAW Index	Subject Code	BAW Index	Subject Code	BAW Index
c61l40	62	c61r37	79	c86r49	36	none	
c61r29	45	c64l31	60	c86r50	35		
c61r31	7	c70r35	39	c86r53	53		
c64r35	-11	c72r50	70	c93l20	32		
c68l36	57	c74r46	62	c93l22	-51		
c68l52	74	c77l43	79	c93l24	-6		
c68r46	-3	c81l33	53				
c72r49	73	c83l49	63				
c73l37	72	c83r48	79				
c73r38	65	c93r46	40				
c73r39	50						
c73r40	48						
c80l33	65						
c80l34	45						
c81l34	5						
c81l40	-40						
c82r50	67						
c83r37	55						
c83r40	56						
c83r42	20						
c85l46	53						
c85l50	74						
c88r53	73						
c93l17	29						
c99l42	72						
Total	25	Total	10	Total	6	Total	0

Table 4.9. All BAW Responses from Re-test Session.
Total number of BAW responses per category.

BAW Type 1		BAW Type 1	(con't)	BAW Type 2		BAW Type 3		BAW Type 4	
Subject Code	BAW Index	Subject Code	BAW Index	Subject Code	BAW Index	Subject Code	BAW Index	Subject Code	BAW Index
p61l30	20	p73l29	70	p65r39	69	p69r32	-15	p69l32	65
p61l31	0	p74l25	79	p69r34	-25	p69r36	-13		
p61l34	36	p74l26	77	p69r38	-3	p69r37	2		
p61l35	-9	p74l28	51	p69r41	-32	p70l31	-61		
p61l36	12	p74l33	66	p70l28	-54	p70l33	-57		
p61l38	52	p74l34	31	p70l29	-59	p73r36	48		
p61l41	57	p74l35	57	p70l34	-63	p74r50	75		
p63r36	-26	p74l37	70	p70l37	-47	p83l30	74		
p65l27	69	p74l39	73	p70r42	-36	p93l49	71		
p65l28	60	p77l37	-5			p93r33	78		
p65r40	71	p85r35	30			p93r35	79		
p65r47	68	p85r36	-6			p93r36	74		
p65r50	64	p85r37	62			p93r47	79		
p65r52	64	p85r38	78						
p67l40	71	p85r43	71						
p67r35	77	p92l33	69						
p67r41	66	p93l42	77						
p68r38	67	p93l43	64						
p68r40	43								
p69l31	54								
p70r29	38								
p70r32	-21								
p70r33	-9								
p70r34	32								
p70r36	-33								
p70r38	-28								
p70r39	-44								
p70r41	-21								
Total			46	Total	9	Total	13	Total	1

Table 4.10. All Subjects Classified with BAW in the first Test Session.
*** Indicates marginal case. See text for details.**

Bilateral BAW			Unilateral BAW		
Subject Code	BAW Type	Number of times BAW was observed in this limb.	Subject Code	BAW Type	Number of times BAW was observed in this limb.
c61			c70		
L	1	1	R	3	1
R	1	3	c72		
c64			R	1	2
L	3	1	c74		
R	1	1	R	3	1
c68			c77*		
L	1	2	L	3	1
R	1	1	c80		
c73			L	1	2
			c81		
L	1	1	L	1	3
R	1	3	c82		
c83			R	1	1
L	3	1	c85		
R	1	4	L	1	2
c93			c86		
L	1	4	R	2	3
R	3	1	c88		
			R	1	1
			c99		
			L	1	1
Total # BAW Subjects	6		Total # BAW Subjects	11	
Total # BAW Limbs	12		Total # BAW Limbs	11	
Grand Total Subjects	17				
Grand Total Limbs	23				

Table 4.11. All Subjects Classified with BAW in the Re-test Session. See text for details.

Bilateral BAW			Unilateral BAW		
Subject Code	BAW Type	Number of times BAW was observed in this limb.	Subject Code	BAW Type	Number of times BAW was observed in this limb.
p65			p61		
L	1	2	L	1	7
R	1	5	p63		
p67			R	1	1
L	1	1	p68		
R	1	2	R	1	2
p69			p77		
L	1	2	L	1	1
R	3	6	p83		
p70			L	2	1
L	3	6	p85		
R	1	8	R	1	5
p73			p92		
L	1	1	L	1	1
R	2	1	P73		
p74			R	4	1
L	1	8			
R	2	1			
p93					
L	1	3			
R	2	4			
Total # BAW Subjects	7		Total # BAW Subjects	8	
Total # BAW Limbs	14		Total # BAW Limbs	8	
Grand Total Subjects	15				
Grand Total Limbs	22				

Table 4.12. Summary of BAW responses in first and re-test sessions.

	Total Number of Large Rotation Trials Administered	*Total Number of Limbs Tested*	*Average Number of Large Rotation Trials per Limb*	*Number of Trials with BAW*	*% of Trials with BAW*	*Number of Normal Trials*	*% of Trials that were Normal*
1st test	557	63	8.8	41	7%	516	93%
Re-test	505	60	8.4	69	14%	436	86%
Combined Total	1062	123	8.6	110	10%	952	90%

Table 4.12A. Summary of subjects displaying the largest number of examples of BAW. In the first test session, the largest number of examples of BAW in any single subject was 3. In the re-test session the largest number of examples of BAW in any single subject was 8. These data are consistent with the observation that subjects displaying BAW on the re-test session displayed more cases per limb than subject on the first test session.

	Number of Subjects Displaying BAW >2 times.	*Number of Subjects Displaying BAW >3 times.*
	1st Test	**Re-test**
Group 1 (Enriched)	**4**	**2**
Group 2 (Semi-Enriched)	**2**	**0**
Group 3 (Control)	**3**	**4**

For the first test session, 7% of the large rotation trials resulted in a BAW diagnosis, and in the re-test session, 14% of the large rotation trials resulted in BAW. Overall, 10% of all rotation trials on the first and re-test sessions resulted in BAW classification. The reason that there were twice as many BAW responses on the re-test session compared to the first test was that subjects displaying BAW on the re-test session displayed more cases per limb. There was not a substantial difference in the number of limbs or subjects with BAW on the first and re-test sessions. In the first test session, 17 out of 32 subjects

displayed unilateral or bilateral BAW. In the second test session, 15 out of 30 subjects displayed unilateral or bilateral BAW.

In the first test session, unilateral BAW was somewhat more commonly observed than bilateral BAW. There were 11 subjects with unilateral BAW and only 6 subjects with bilateral BAW. In the re-test session, bilateral and unilateral BAW were somewhat more equally distributed with 7 subjects displaying bilateral BAW and 8 subjects displaying unilateral BAW. The majority of BAW responses were Type 1, and the remaining cases were Types 2, 3, and 4 with the smallest representation from Type 4.

There was one subject (C77) who displayed BAW only one time, had a marginal BAW Index of 79, and had an electromyograghic classification of Type 3 BAW. This subject was considered to be a marginal example of BAW. All other BAW subjects were more convincing in that BAW Index in one or more response was substantially less than 80 and/or electromyographic data resulted in Type 1 classification.

The next section will address the issue of whether or not the testing and classification methods reliably detected BAW and normal responses on 2 consecutive test sessions.

Reliability of test protocol and classification technique for identifying BAW and normal responses over 2 consecutive test sessions.

To aid the reader's understanding of this section, definitions for reliability, sensitivity, specificity, and predictive value have been provided here.

Reliability- The extent to which a test yields the same result on more than one testing occasion. Relates to the *reproducibility* of test results.

Sensitivity- How well a test can detect the condition for which it is testing. The extent to which the test is positive in patients who have the disorder.

Specificity- How well the test accurately provides negative results in patients who do not have the condition.

Predictive Value of a Positive Test- The percentage of patients with a positive result who actually have the condition.

Predictive Value of a Negative Test- The probability that a patient does not have the condition when the test result is negative.

To determine whether or not our test and classification criteria were capable of reliably detecting BAW and normal responses on two consecutive occasions, subjects underwent two separate but identical test sessions. Responses to large rotations were classified as normal or BAW (as described in the previous section) without knowledge of which trials from the first test session belonged to the same subjects on the re-test session. After all the responses, limbs, and subjects from both test sessions had been classified as

normal or BAW, responses were de-coded and matched appropriately. A total of 30 subjects (59 limbs) were tested on two consecutive occasions. There was an odd number of limbs presented here because data from 1 limb of one subject was lost.

Table 4.13. Subjects Who Participated in the Reliability Study by Being Tested on 2 Occasions.

	Group 1	Group 2	Group 3	Total
males	5	6	4	15
females	4	5	6	15
Total	9	11	10	30

Table 4.14. Number of Limbs Used for Reliability Study.

	Group 1	Group 2	Group 3	Total
males	10	12	7	29
females	8	10	12	30
Total	18	22	19	59

For the purpose of the reliability study, limbs were classified as (+) for BAW if the limb displayed BAW at least one time within the test session. Limbs were classified as (-) or normal if BAW was never observed within the test session. BAW Types 1, 2, 3 and 4 were not taken into consideration for this portion of the study, nor was severity or number of times that BAW was observed. A (+, +) match for limbs occurred when a limb displayed at least 1 example of any type of BAW on both the first test and re-test session.

Subjects were classified as (+) for BAW if **either** one **or** both limbs exhibited BAW at least one time within the test session. Subjects were classified

as (-) or normal if neither limb displayed BAW within the test session. Whether or not the subjects displayed bilateral of unilateral BAW was not taken into account for this phase of the study. A (+, +) match for subjects required that a subject display BAW in one or both limbs at least one time on both the first and re-test sessions. In the case of unilateral BAW, it was not necessary that BAW be observed on the same arm on both occasions.

After all limbs and subjects were classified, subjects from the first test session were matched accordingly to re-test subjects, and the extent to which responses agreed was calculated for limbs and for subjects using a Kappa statistic. Kappa values <0.4 are considered poor, from 0.4 to 0.7 are considered fair, and >0.7 are considered good (Richard Mathias, personal communication).

Table 4.15. Summary of first Test and Re-test Responses for all Subjects.

Group	*1st Test Code*	*Dx. 1st Test*	*Re-test Code*	*Dx. Re-test*	*Group*	*1st Test Code*	*Dx. 1st Test*	*Re-test Code*	*Dx. Re-test*	*Group*	*1st Test Code*	*Dx. 1st Test*	*Re-test Code*	*Dx. Re-test*
1	C61		P93		2	C60		P92		3	C62		P90	
	L	BAW	L	BAW		L	Normal	L	BAW		L	Normal	L	Normal
	R	BAW	R	BAW		R	Normal	R	Normal		R	Normal	R	Normal
1	C64		P72		2	C67		P82		3	C63		P74	
	L	BAW	L	No Return		L	Normal	L	Normal		L	Normal	L	BAW
	R	BAW	R	No Return		R	Normal	R	Normal		R	Normal	R	BAW
1	C65		P80		2	C69		P76		3	C72		P83	
	L	Normal	L	Normal		L	Normal	L	Normal		L	Normal	L	BAW
	R	Normal	R	Normal		R	Normal	R	Normal		R	BAW	R	Normal
1	C68		P68		2	C70		P91		3	C74		P87	
	L	BAW	L	Normal		L	Normal	L	Normal		L	Normal	L	Normal
	R	BAW	R	BAW		R	BAW	R	Normal		R	BAW	R	Normal
1	C73		P67		2	C71		P60		3	C76		P62	
	L	BAW	L	BAW		L	Normal	L	Normal		L	Normal	L	Normal
	R	BAW	R	BAW		R	Normal	R	Normal		R	Normal	R	Normal
1	C79		P69		2	C75		P66		3	C78		P65	
	L	Normal	L	BAW		L	Normal	L	Normal		L	Normal	L	BAW
	R	Normal	R	BAW		R	Normal	R	Normal		R	Normal	R	BAW
1	C80		P63		2	C77		P86		3	C81		P64	
	L	BAW	L	Normal		L	BAW	L	Normal		L	BAW	L	Normal
	R	Normal	R	BAW		R	Normal	R	Normal		R	Normal	R	Normal
1	C83		P77		2	C82		P73		3	C87		P84	
	L	BAW	L	BAW		L	Normal	L	Normal		L	Normal	L	Normal
	R	BAW	R	Normal		R	BAW	R	BAW		R	Normal	R	Normal
1	C88		P85		2	C84		P71		3	C89		P61	
	L	Normal	L	Normal		L	Normal	L	Normal		L	No Data	L	BAW
	R	BAW	R	BAW		R	Normal	R	Normal		R	Normal	R	Normal
1	C98		P98		2	C85		P78		3	C93		P70	
	L	Normal	L	Normal		L	BAW	L	No Return		L	BAW	L	BAW
	R	Normal	R	Normal		R	Normal	R	No Return		R	BAW	R	BAW
					2	C86		P79						
						L	Normal	L	Normal					
						R	BAW	R	Normal					
					2	C99		P75						
						L	BAW	L	Normal					
						R	Normal	R	Normal					

Table 4.16. Kappa by Subject. Kappa=0.333

		1st Test		
		BAW (+)	Normal (-)	Total
Re-test	BAW (+)	9	4	13
	Normal (-)	6	11	17
	Total	15	15	30

Table4.17. Kappa by Limb. Kappa=0.273

		1st Test		
		BAW (+)	Normal (-)	Total
Re-test	BAW (+)	10	9	19
	Normal (-)	10	30	40
	Total	20	39	59

The Kappa statistic that was calculated based on agreement in classification by limbs represented the extent to which our methods reliably detected the same response in the same limb on two test occasions. This Kappa value was 0.273 which was considered poor. The Kappa statistic based on agreement in classification by subjects represented the extent to which the same subject could be classified as normal or BAW, but not necessarily in the same limb or always unilaterally or bilaterally. In this sense, classification by subject was a less conservative approach and indicated the extent to which we could repeatedly classify a subject (but not a specific limb) with BAW. The Kappa value for this classification scheme was 0.333 which was still within the range of poor. There was no tendency for responses to shift in one particular direction,

either BAW to normal or normal to BAW, on consecutive test sessions as per the McNear test of symmetry.

One possible reason that test/re-test reliability was poor may have been that the classification criteria was too lax (or not selective), and limbs that were normal were classified as BAW on some occasions because BAW Index slipped slightly below 80. Recall that the cut-off of 80 for BAW was arbitrary because there was no clear division between normal and BAW responses. To investigate this possibility, we re-classified all the limbs according to several more rigorous (selective) criteria, and counted the number of cases in which limbs were classified inconsistently on the first and re-test trials. We also estimated the sensitivity of each criterion by dividing the combined total of limbs that were classified as BAW on the first and re-test session by the total number of limbs tested in the first and re-test session. Percent detection (sensitivity) and number of inconsistent responses (estimate of reliability) on different classification criteria were compared to the same values for the original classification criteria.

Changes in sensitivity of BAW detection included changing the cut-off BAW Index from 80 to 50 in increments of 10 and changing the number of observations of BAW from one to two. In all cases the requirements for a BAW diagnoses were more rigorous. All the combinations of criteria and results are presented in Table 4.18.

Table 4.18. Relationship Between Criteria for BAW and Rate of Detection of BAW. * Original criteria.

BAW Criteria (BAW Index and # of Observations)	*Combined Total (1st Test and Re-test) Number of Limbs Tested*	*Total Number of Limbs in Which Dx. on 1st Test and Re-test Were NOT the Same. Estimate of Reliability*	*Combined Total (1st Test and Re-test) Number of Limbs with BAW*	*% Detection (# of limbs with BAW/Total # of limbs tested) Estimate of Sensitivity*
*1. BAW Index<80, observed 1x	118	19	36	31%
2. BAW Index≤50, observed 1x	118	13	19	16%
3. BAW Index≤60, observed 1x	118	16	22	19%
4. BAW Index≤70, observed 1x	118	19	29	25%
5. BAW Index≤80, observed 2x	118	17	21	18%
6. BAW Index≤70, observed 2x	118	15	16	14%
7. BAW Index≤60, observed 2x	118	9	12	10%
8. BAW Index≤50, observed 2x	118	7	8	7%

The original criteria resulted in the highest sensitivity (31% of limbs diagnoses as positive) and the lowest reliability (19 limbs with inconsistent diagnoses). The more rigorous (or specific) criteria resulted in more reliable classifications in all but one case (criteria number 4). The increase in reliability occurred at the cost of substantially reduced sensitivity. Figure 4.4 shows the relationship between sensitivity and reliability. As reliability increased, sensitivity decreased. The most substantial changes in reliability occurred with criteria numbers 7 and 8, in which the number of inconsistent responses

dropped from 19 to 9 and 19 to 7, respectively. However, enhanced reliability was accompanied by a reduction in sensitivity to 10% and 7%, respectively. Recall that only 10% of all the rotation trials displayed BAW. Further reducing sensitivity would have resulted in a very small number of BAW responses to be evaluated in subsequent phases. The reduction in detection occurred approximately equally in all 3 groups.

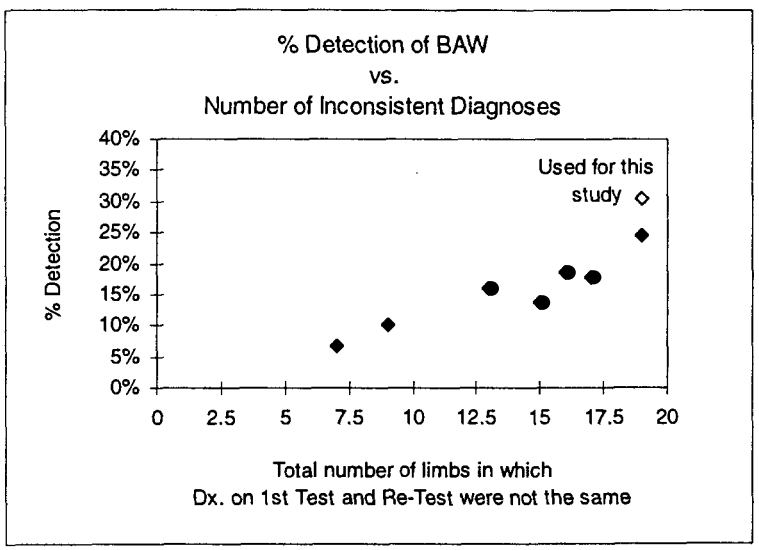

Figure 4.4. % Detection of BAW vs. Number of Inconsistent Diagnoses.

Prevalence of BAW in injured individuals living in the Lower Fraser Valley vs. injured and non-injured individuals living outside the Lower Fraser Valley.

Prevalence of a disease is defined a the number of cases of a disorder that are present at a given point in time (Dawson-Saunders and Trapp, 1994). Incidence is defined as the number of *new* cases of a disease that occur (Dawson-Saunders and Trapp, 1994).

Using the classification criteria for BAW that was initially established (BAW Index < 80, observed at least 1 time) the prevalence of BAW in each of the 3 groups (enriched, semi-enriched, and control) was calculated, and chi-square analysis was used to determine whether or not significant differences in prevalence existed between the three groups. For all analyses in this section significance was determined at $p \leq 0.05$. If a significant difference between groups was observed, 3 additional pair-wise chi-square tests were conducted to determine which of the 3 groups differed from the others. In these cases, the observed p value was multiplied by 3. After adjustment, if p was ≤ 0.05, the difference was considered significant. The adjusted p value is referred to as a Bonferoni adjusted p value and was used to minimize experiment-wise error. Because the test/re-test reliability was poor, prevalence of BAW was calculated for the first and re-test session, separately and together.

Table 4.19. Number of Subjects from Each Group who were Used for Evaluation of Prevalence of BAW.

	1st Test	Re-test
Group 1 (Enriched)	10	9
Group 2 (Semi-enriched)	12	11
Group 3 (Control)	10	10
Total	32	30

In the first test session, the proportion of subjects in each group with either unilateral or bilateral BAW ranged from a minimum of 40% for Group 3 to a maximum of 70% for Group 1. There was no significant difference in the proportion of subjects in each of the 3 groups who displayed either bilateral or unilateral BAW. However, a trend was observed suggesting that subjects in Group 1 may have displayed slightly more BAW than subjects in the other 2 groups and that subjects in Group 2 may have displayed a slightly higher proportion of BAW than subjects in Group 3. (Figure 4.5, left.)

In the re-test session, the proportion of subjects with unilateral or bilateral BAW ranged from 18% in Group 2 to 77% in Group 1. There was a large decline in observations of BAW in subjects from Group 2. In the re-test session, Group 1 displayed a significantly larger proportion of subjects with bilateral or unilateral BAW than in Group 2. There was no significant difference between Groups 1 and 3 or Groups 2 and 3 with respect to prevalence of BAW.

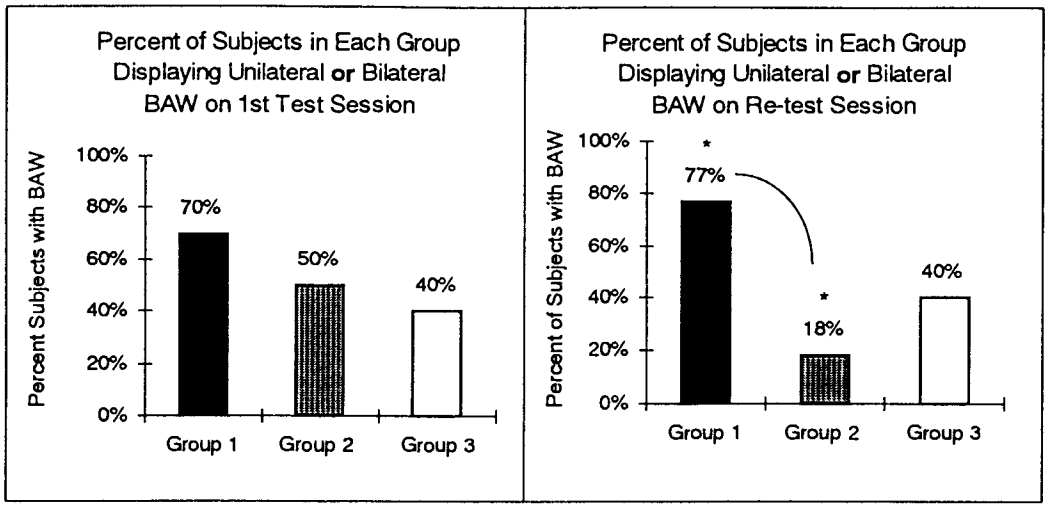

Figure 4.5. Proportion of subjects in each of the 3 groups who displayed either bilateral or unilateral BAW on the first and re-test sessions. There was no significant difference between groups for the first test session. There was a significant difference between groups 1 and 2 on the re-test session. Despite the lack of significance, these data suggested that group 1 may tend to display more BAW than the other 2 groups.

Looking only at subjects with bilateral BAW, the proportion of subjects with bilateral BAW in the first test session ranged from 0% in Group 2 to 50% in Group 1. For the re-test session, the proportion of bilateral BAW ranged from 0% in Group 2 to 33% in Group1. There was a significantly larger proportion of subjects in Group 1 compared to Group 2 who displayed bilateral BAW in the first test session. This difference did not exist in the re-test session, however. For both the first and re-test session, there was no significant difference in the proportion of subjects displaying bilateral BAW between Groups 1 and 3 and Groups 2 and 3. Despite the lack of significance, these data suggested that subjects in Group 1 may have had a tendency to display more bilateral BAW than the other 2 groups.

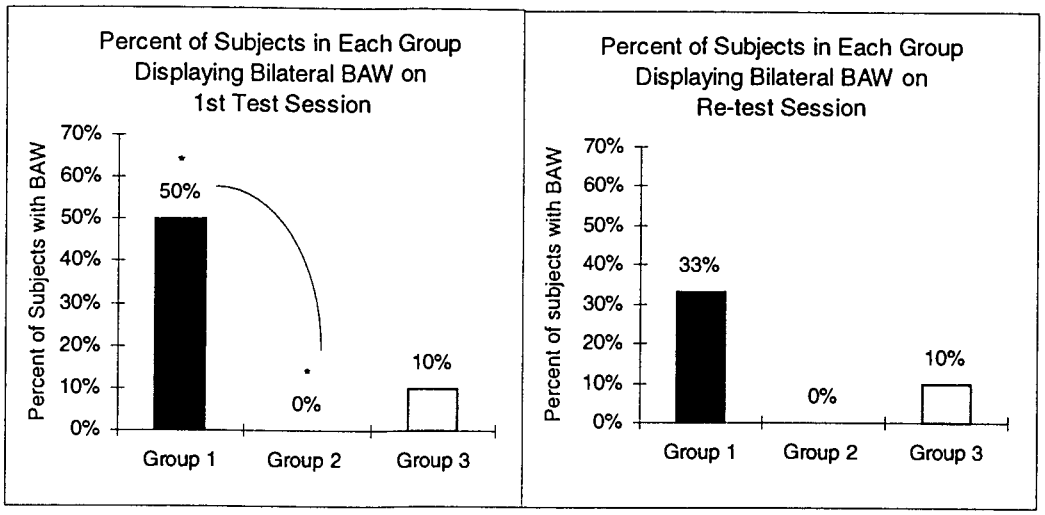

Figure 4.6. Proportion of subjects in each of the three groups who displayed bilateral BAW on the first and re-test sessions. Group 1 had a significantly larger proportion of subjects with bilateral BAW than group 2 in the first test session.

The most interesting observations occurred when the first and re-test sessions were evaluated together. A total of 69% of subjects (22/32) displayed BAW at least one time on either the first or the re-test session. The proportion of subjects who displayed bilateral or unilateral BAW in either the first or re-test session was very high in all 3 groups, ranging from on minimum of 58% in Group 2 to a maximum of 80% in Group 1. There was no significant difference in the proportion of subjects from each group who displayed either bilateral **or** unilateral BAW on either the first **or** the re-test session. These data suggested that if subjects were allowed 2 separate test sessions to display BAW there would be an equal prevalence of BAW in each of the 3 groups and that the prevalence in each group would exceed 50%.

The percentage of subjects in each group who displayed bilateral or unilateral BAW on **both** the first **and** the re-test session was 8% for Group 2,

10% for Group 3, and 60% for Group 1. There was a significant difference between Groups 1 and 2 and Groups 1 and 3 with respect to the percentage of subjects in each group who displayed BAW on both the first and the re-test session. The enriched group had a significantly larger proportion of subjects displaying BAW on both test sessions than the other 2 groups.

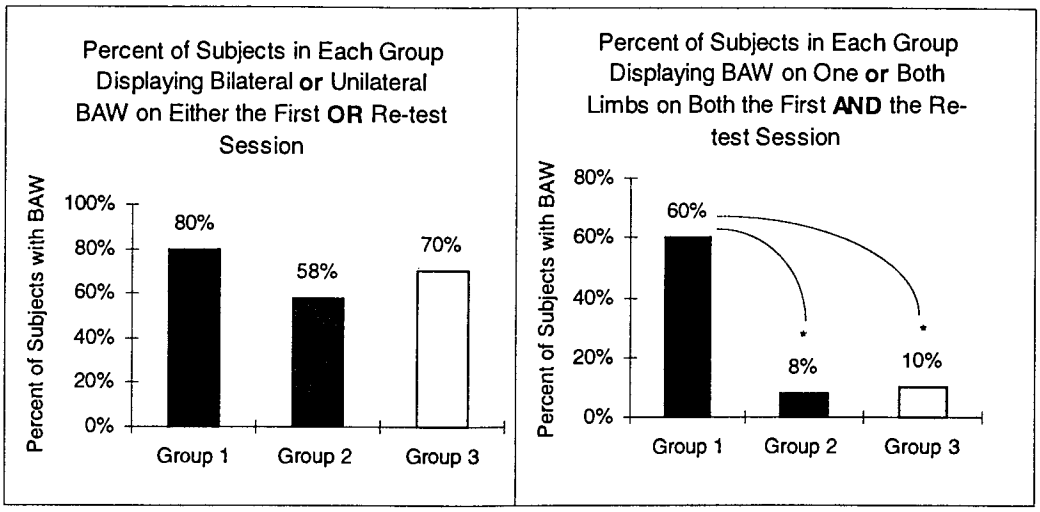

Figure 4.7. Prevalence of BAW in the 3 groups when first test and re-test were evaluated together. There was no difference in the proportion of subjects in each of the three groups who displayed unilateral or bilateral BAW on *either* the first *or* re-test session. Group 1 displayed a significantly larger proportion of subjects with BAW on *both* the first *and* the re-test sessions than the other two groups.

Quantification of torque, timing, position and EMG variables of normal and BAW responses.

The goal of this portion of the project was to describe the characteristics of torque and EMG output during handle rotations and to describe the similarities and differences between normal and BAW responses in order to understand better the possible mechanisms involved in the BAW response. Torque, position, and EMG parameters that were thought to represent important events in each of the responses were measured. Definitions and

descriptions of the parameters are listed in the Methods. The glossary in the Appendix also provides a review of the parameters.

Is there a difference in T_{in} between normal and BAW trials?

A single value for T_{in} was measured from the torque trace of each trial 20 ms prior to the onset of handle rotation. As shown in Figure 4.8, values for T_{in} varied widely from 1 Nm to 14.3 Nm. There was a wide range of values for T_{in} in both the BAW and normal groups. The mean value of T_{in} for all normal trials was 6.1 Nm and for all BAW trials was 4.6 Nm. Average T_{in} for normal trials was significantly larger than for BAW trials, suggesting that subjects with BAW produced less initial torque than normal subjects. It is important to note, however, that BAW did not exclusively affect limbs with small values of T_{in}. BAW was present in limbs with the largest and the smallest values of T_{in}.

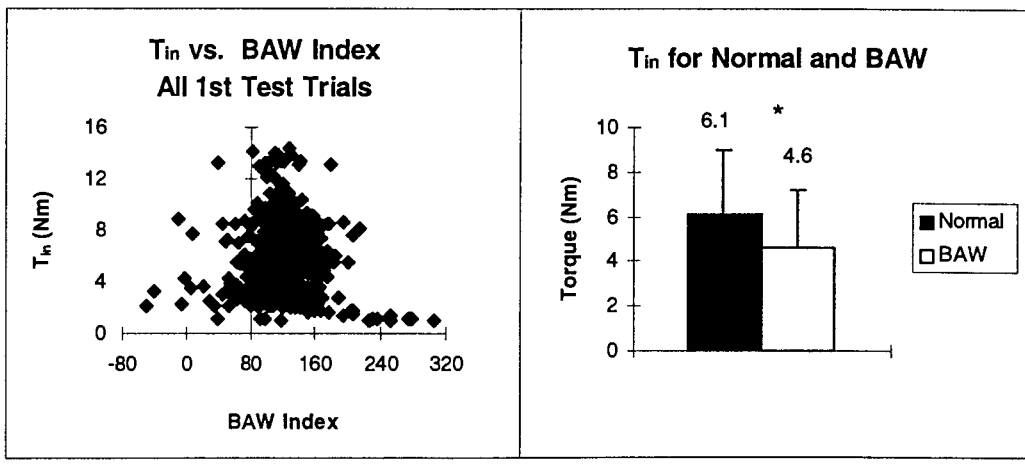

Figure 4.8. Left- T_{in} vs. BAW Index for all First Test Trials. There was a wide range of values for T_{in} in the BAW and normal group. Right- Mean T_{in} for BAW and normal trials. T_{in} for normal trials was significantly larger than for BAW trials. (n=530, $p \leq 0.05$)

One possible explanation for this outcome was that subjects who had BAW may have produced less T_{in} only on BAW trials, although this was unlikely because the test protocol demanded that all subjects be within ±5% of the target range before the handle rotated. To examine this possibility, we calculated mean T_{in} for all normal trials within a given limb and mean T_{in} for all BAW trials within the same limb. Mean T_{in} for normal trials was divided by mean T_{in} for BAW trials to obtain a ratio of T_{in} in normal trials to T_{in} in BAW trials. An overall mean ratio was calculated for all BAW limbs. The mean ratio of T_{in} for normal trials to T_{in} for BAW trials was 1.0. A pairwise comparison of the means showed no significant difference between T_{in} on BAW trials and T_{in} on normal trials ($n=23$, $p \leq 0.05$). These results demonstrated that subjects with BAW produced the same T_{in} on normal trials as on BAW trials and further suggested that the limbs with BAW produced less initial torque than normal limbs.

Is there a difference in %Tincr between normal and BAW subjects?

A value for $\%T_{incr}$ was calculated from the torque trace of each rotation trial. Values ranged widely from 12 to 238 percent of T_{in} and were always greater than zero. These data clearly demonstrated that there was always an increase in torque associated with handle rotation. As shown in Figure 4.9, there was a linear relationship between BAW Index and $\%T_{incr}$ for all normal responses ($r=0.84$). This relationship was not surprising because BAW Index was calculated by dividing the minimum value of torque by T_{in}. The larger the

$\%T_{incr}$, the more likely it was for subjects to have a value for T_{min} that was well above T_{in}. For BAW Indices <80, the relationship between BAW Index and $\%T_{incr}$ became more flat and there was no correlation between BAW Index and $\%T_{incr}$ (r= -0.05), suggesting that trials with BAW had approximately the same $\%T_{incr}$.

The mean value of $\%T_{incr}$ for normal trials was 65 and for BAW trials was 59 percent of T_{in}. There was no significant difference between $\%T_{incr}$ for normal and BAW trials.

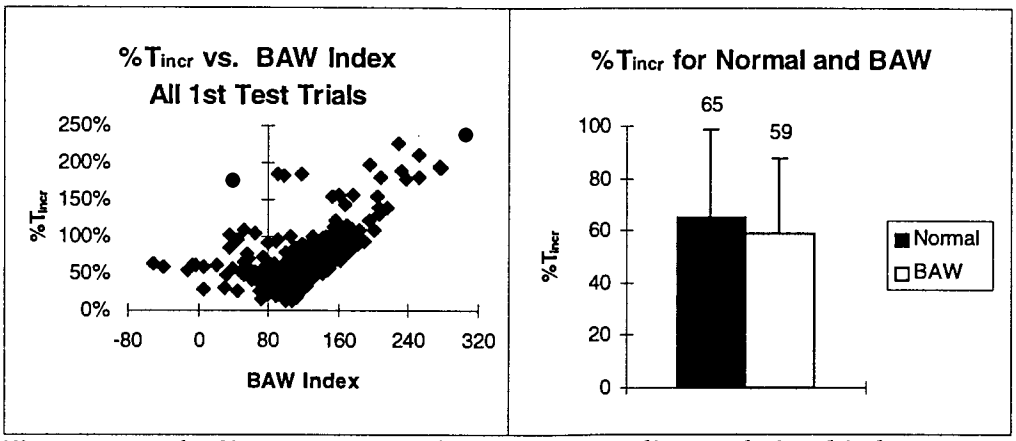

Figure 4.9. Left- $\%T_{incr}$ vs. BAW Index. There was a linear relationship between BAW Index and $\%T_{incr}$ for normal responses. Right- Mean values for $\%T_{incr}$ for normal and BAW trials. There was no difference in $\%T_{incr}$ between normal and BAW responses. (n=530, $p \leq 0.05$)

Is there a difference on t_{Tpk} between normal and BAW subjects?

For each trial, t_{Tpk} was measured with respect to onset of handle rotation. There was a range of responses for t_{Tpk} from a minimum of 100 ms to a maximum of 384 ms. There was a linear relationship between BAW Index and t_{Tpk} for normal responses, as suggested by a correlation coefficient equal to 0.84. This relationship was predictable because t_{Tpk} was related to $\%T_{incr}$, and the larger

the value for $\%T_{incr}$, the more likely its was that BAW Index was also large. There was no relationship between BAW Index and t_{Tpk} for BAW trials (r= -0.09).

The mean value for t_{Tpk} for normal trials was 192 ms and for BAW trials was 161 ms. BAW responses had a significantly shorter t_{Tpk} than normal responses.

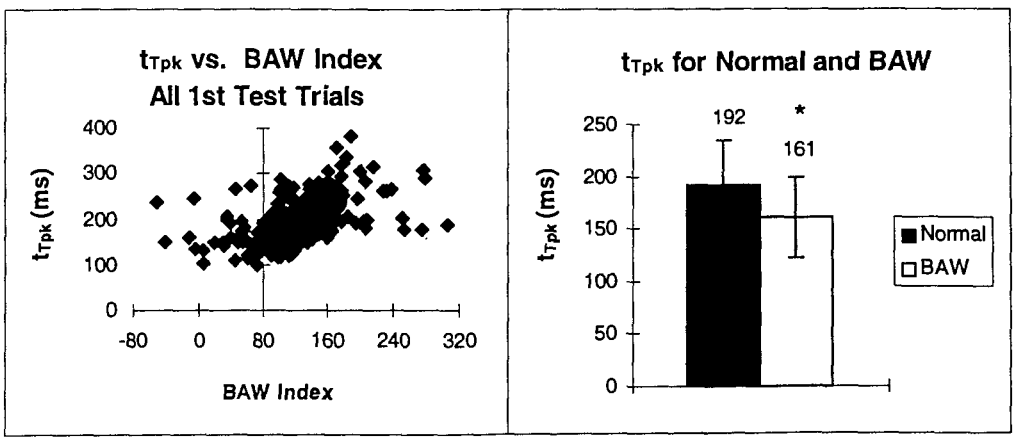

Figure 4.10. Left- t_{Tpk} vs. BAW Index. There was a linear relationship between BAW Index and t_{Tpk} for normal responses. Right- Mean value for t_{Tpk} for BAW and normal responses. There was a significant difference in t_{Tpk} for normal and BAW trials. (n=530, $p \leq 0.05$)

To investigate whether this difference existed between normal and BAW responses within the same limb, we calculated mean t_{Tpk} for all normal trials within a given limb and mean t_{Tpk} for all BAW trials within the same limb. Pairwise comparison of the means by T-test revealed a significant difference between t_{Tpk} on BAW trials and T_{tpk} on normal trials for the same limb (n=23, $p \leq 0.05$) This result suggested that subjects with BAW had a shorter t_{Tpk} on their BAW trials than on their normal trials.

Is there a difference in POS_{Tpk} between normal and BAW subjects?

The handle position at which peak torque occurred was measured for each large rotation trial. Values for POS_{Tpk} ranged from 15° to 39°. The mean value of POS_{Tpk} for normal trials was 30° and for BAW trials was 25°. POS_{Tpk} for normal responses was significantly larger than POS_{Tpk} for BAW responses. This result is consistent with the result obtained for t_{Tpk}.

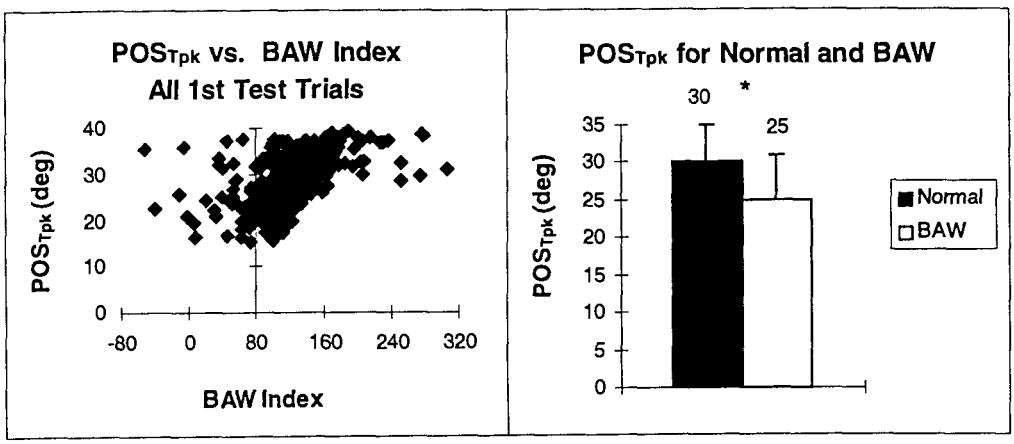

Figure 4.11. Left- POS_{Tpk} vs. BAW Index. Right- Mean values of POS_{Tpk} for BAW and normal responses. POS_{Tpk} was larger for normal responses than for BAW responses. (n=530, $p \leq 0.05$)

Is there a difference in T_{minyN} between normal and BAW subjects?

T_{minyN} was calculated for each trial and presented as a percentage of T_{in}. Values for T_{minyN} ranged from 65 to 315 percent of T_{in}. As displayed in Figure 4.12, T_{minyN} was linearly related to BAW Index for all responses (r=0.84 for BAW Indices >80, r=0.25 for BAW Indices <80). With the exception of a few severe cases of BAW, T_{minyN} was always ≥100%, demonstrating that BAW could not have been detected as early as the "yield phase" of the torque response. The mean value for T_{minyN} for normal responses was 153% and for BAW responses was 127% of T_{in}. The magnitude of the yield in the torque trace of BAW responses was significantly larger than the yield in normal responses.

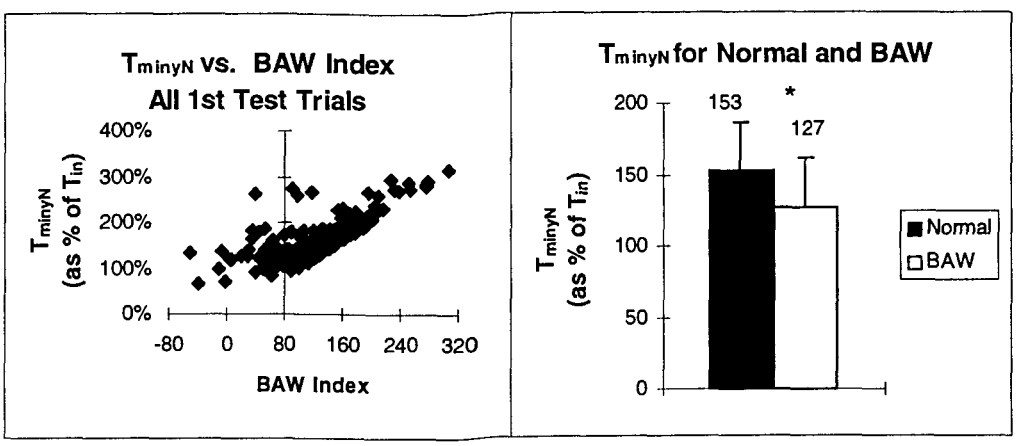

Figure 4.12. Left- T_{minyN} vs. BAW Index. There was a linear relationship between BAW Index and T_{minyN} for the entire range of BAW Indices. Right- Mean values of T_{minyN} for BAW and normal responses. BAW trials display a significantly larger yield than normal responses. (n=530, p≤0.05)

Is there a difference in rate of change of torque during the yield and plateau phases of the torque trace between normal and BAW subjects?

The average and maximum rate of change of torque during the "yield" and "plateau phases" of the torque trace were measured for all large rotation responses. The average rate of change of torque for BAW and normal trials are listed in Figure 4.13. (Note that values for rate of change of torque were expressed as negative numbers because torque was declining during these phases. The largest values for rate of change of torque refer to the values that were most negative; i.e. torque was declining fastest. All values were normalized to T_{in}.)

For all 4 variables that were measured (RtaveryN, RtmaxyN, RtaverpN, and RtmaxpN), BAW responses displayed significantly larger values for rate of change of torque than normal responses ($n=530$, $p \leq 0.05$). In other words, torque declined more rapidly in the "yield" and "plateau phases" of BAW trials than in normal trials. These data are displayed in Figure 4.13.

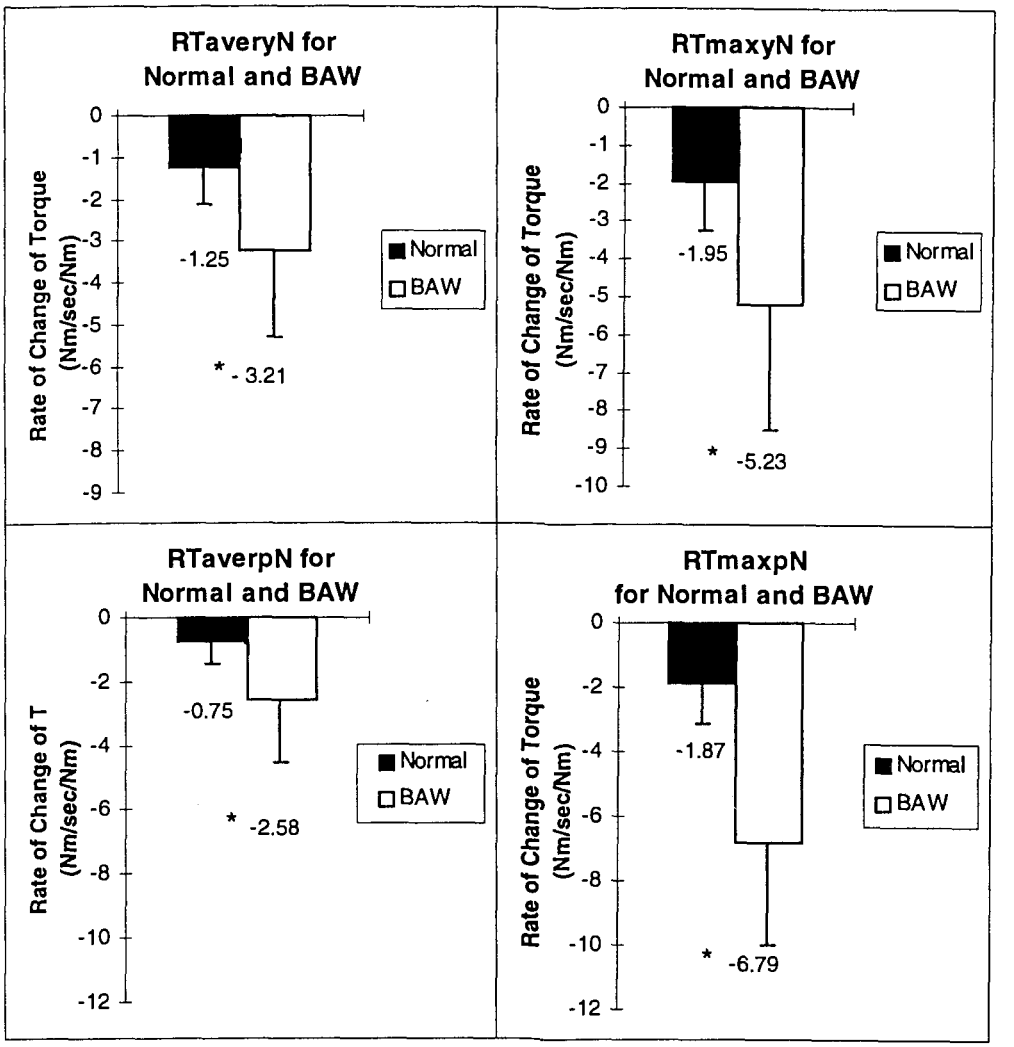

Figure 4.13. Top-Mean values for maximum and average rate of change of torque during the "yield phase" of the torque response. BAW responses displayed a significantly faster decline in torque during the "yield phase" than normal responses. Bottom- Mean values for maximum and average rate of change of torque during the "plateau phase" of the rotation. BAW responses displayed a significantly faster decline in torque during the "plateau phase" than normal responses.

To investigate whether these differences existed between normal and BAW responses within the same limb, we calculated mean RTaveryN, RTmaxyN, RTaverpN, and RTmaxpN for all normal trials within a given limb and mean RTaveryN, RTmaxyN, RTaverpN, and RTmaxpN for all BAW trials within the same limb. Pairwise comparisons between the means revealed that

Rtavery, Rtmaxy, and Rtmaxp were significantly different, but Rtaverp was not ($n=23$, $p \leq 0.05$). This result suggested that subjects with BAW had higher rates of change of torque on their BAW trials than on their normal trials according to all but one measure.

Is there a difference in initial PT and BI EMG output between normal and BAW subjects?

Values of initial PT and BI activity were obtained from each trace and were presented as a percent of maximum EMG. We observed a wide range of initial values of PT and BI EMG in both BAW and normal responses, as depicted in Figure 4.14. The mean value for $PTEMG_{inN}$ in trials with BAW was 42% of maximum and for normal trials was 44% of maximum. The mean value for $BIEMG_{inN}$ in BAW trials was 15% and in normal trials was 13%. There was no significant difference between initial EMG output as a percent of maximum EMG between normal and BAW trials for either PT or BI. BAW and normal subjects used substantially more PT EMG (approximately 40% of maximum) than BI EMG (approximately 15% of maximum) to produce 85% of maximum pronation torque output.

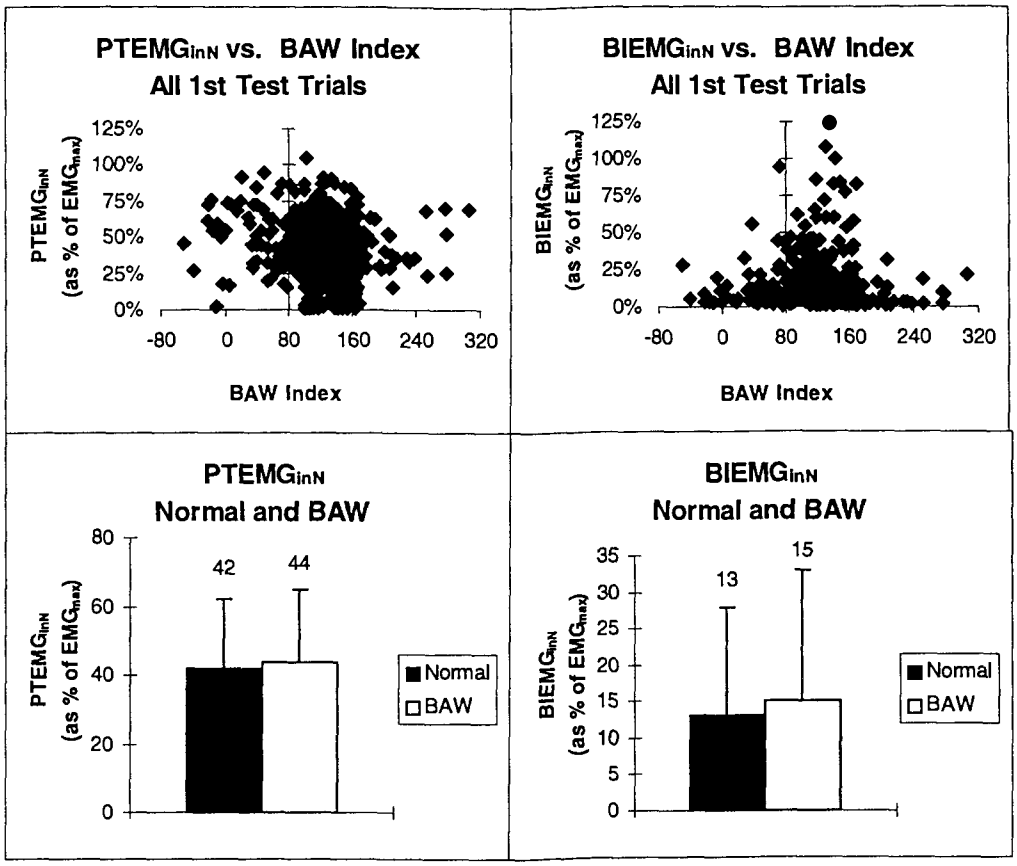

Figure 4.14. Top- $PTEMG_{inN}$ and $BIEMG_{inN}$ vs. BAW Index. Bottom- Mean values for $PTEMG_{inN}$ and $BIEMG_{inN}$. Subjects used a larger percent of maximum PT EMG than BI EMG to produce T_{in}. There was no difference in EMG_{in} for PT or BI between normal and BAW responses.

Is there a difference in short latency PT and BI EMG between normal and BAW subjects?

EMG activity in the PT and BI was measured 20 ms after onset of handle rotation for a period of 60 ms to assess EMG activity in the short latency reflex time frame. Data are expressed as the net change from EMG_{in} as a percentage of EMG_{max}. For both BAW and normal responses, mean PT EMG activity during the short latency time frame was greater than initial EMG and mean BI activity during the same time frame was less than initial EMG. The mean value of $PTEMG_{RN}$ for BAW responses was 19% and for normal responses was 27% of

EMG_{max}. The mean value of $BIEMG_{RN}$ for BAW responses was -10% and for normal responses was -6%. There was no significant difference between mean $PTEMG_{RN}$ or mean $BIEMG_{RN}$ between the 2 groups. See Figure 4.15.

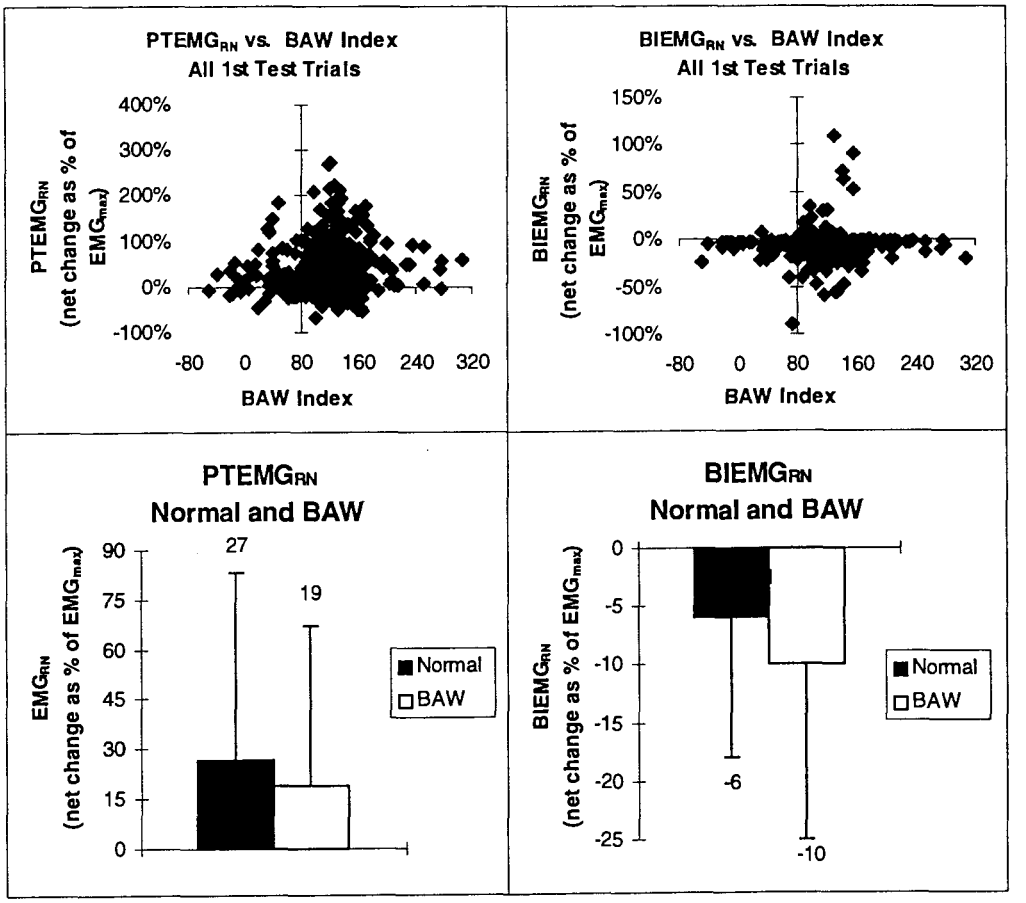

Figure 4.15 Top- $PTEMG_{RN}$ and $BIEMG_{RN}$ vs. BAW Index. There was evidence of stretch reflex activity in PT and reciprocal inhibition of BI. In the top left figure, there is an outlier at 591% of maximum that was not shown in the figure. Bottom- Mean values of $PTEMG_{RN}$ and $BIEMG_{RN}$ for normal and BAW trials. There was no significant difference between the normal and BAW trials with respect to EMG activity in either muscle group.

These data suggest that stretch reflex activity in PT was present in both groups and that there was no difference in the average magnitude of the stretch reflex. These data also provide evidence of reciprocal inhibition of BI in both

groups and suggest that the magnitude of reciprocal inhibition was the same in both groups.

Is there a difference in PT and BI EMG prior to peak torque between normal and BAW subjects?

The magnitude of PT and BI EMG prior to peak torque was measured in each rotation trial over a 60 ms window, and data were presented as net change in activity from EMG_{in} as a percent of EMG_{max}. In both the normal and BAW group, there was a net increase in mean PT EMG prior to T_{pk}. The mean value for $PTEMG_{TpkN}$ for normal responses was 15% and for BAW responses was 11%. There was no significant difference between the two groups with respect to $PTEMG_{TpkN}$. Mean $BIEMG_{TpkN}$ for normal and BAW trials were both significantly greater than EMG_{in} with values of 16% and 5%, respectively. See Figure 4.16.

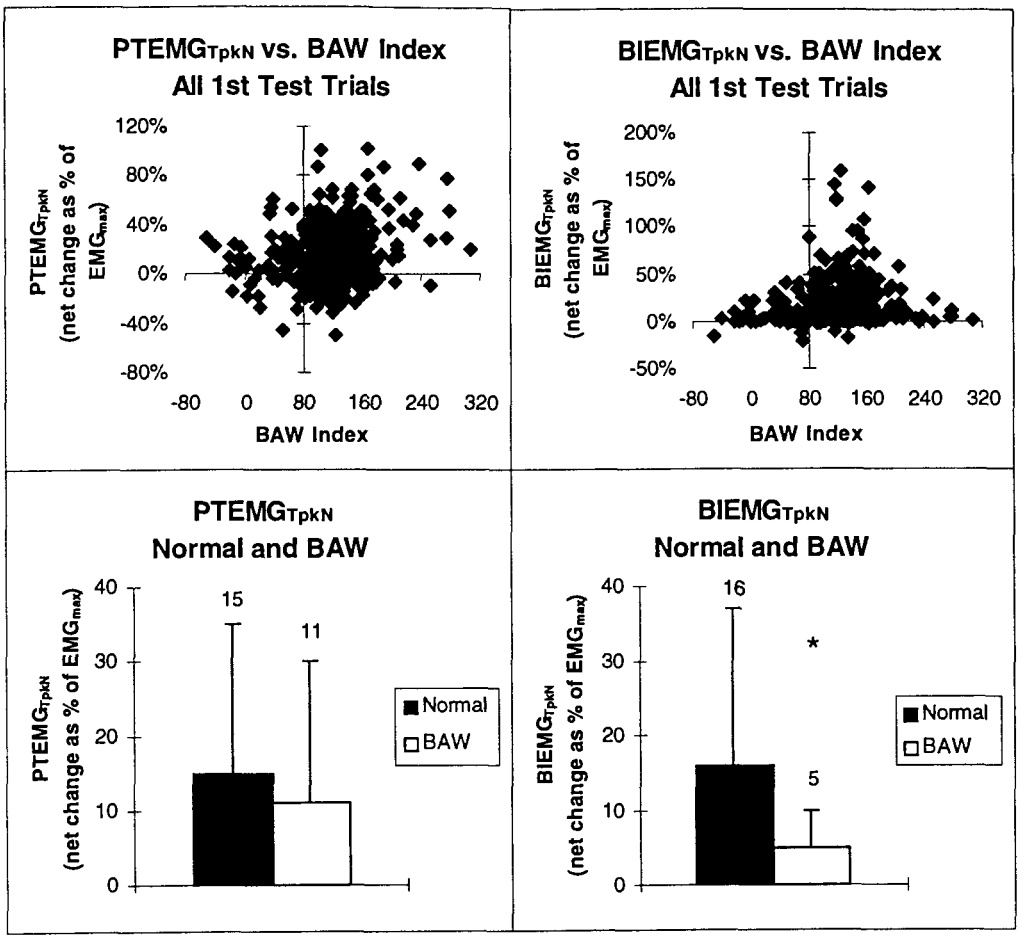

Figure 4.16. Top- $PTEMG_{TpkN}$ and $BIEMG_{TpkN}$ vs. BAW Index. Bottom- Mean values for BAW and normal responses for $PTEMG_{TpkN}$ and $BIEMG_{TpkN}$. There is significantly less EMG activity in BI prior to T_{pk} in BAW trials than in normal trials.

Is there a difference in PT and BI EMGin the last 100 ms of the "hold phase" between normal and BAW subjects?

PT and BI EMG were measured during the last 100 ms of the "hold phase" of the rotation to determine whether or not there was a decrease in activation of both muscle groups associated with BAW. Values were presented as net change from EMG_{in} as a percentage of EMG_{max}. For normal responses, average values of $PTEMG_{LN}$ and $BIEMG_{LN}$ were greater than EMG_{in}, demonstrating that both muscle groups remained active though the end of the

"hold phase" of the rotation. In contrast, mean values for $PTEMG_{LN}$ and $BIEMG_{LN}$ in the BAW group are below EMG_{in}, indicating that activity in PT and BI dropped substantially by the end of the "hold phase". Values for $PTEMG_{LN}$ and $BIEMG_{LN}$ were significantly larger in normal trials than in BAW trials. The scatter plots in Figure 4.17 display the drop in PT and BI activity associated with BAW.

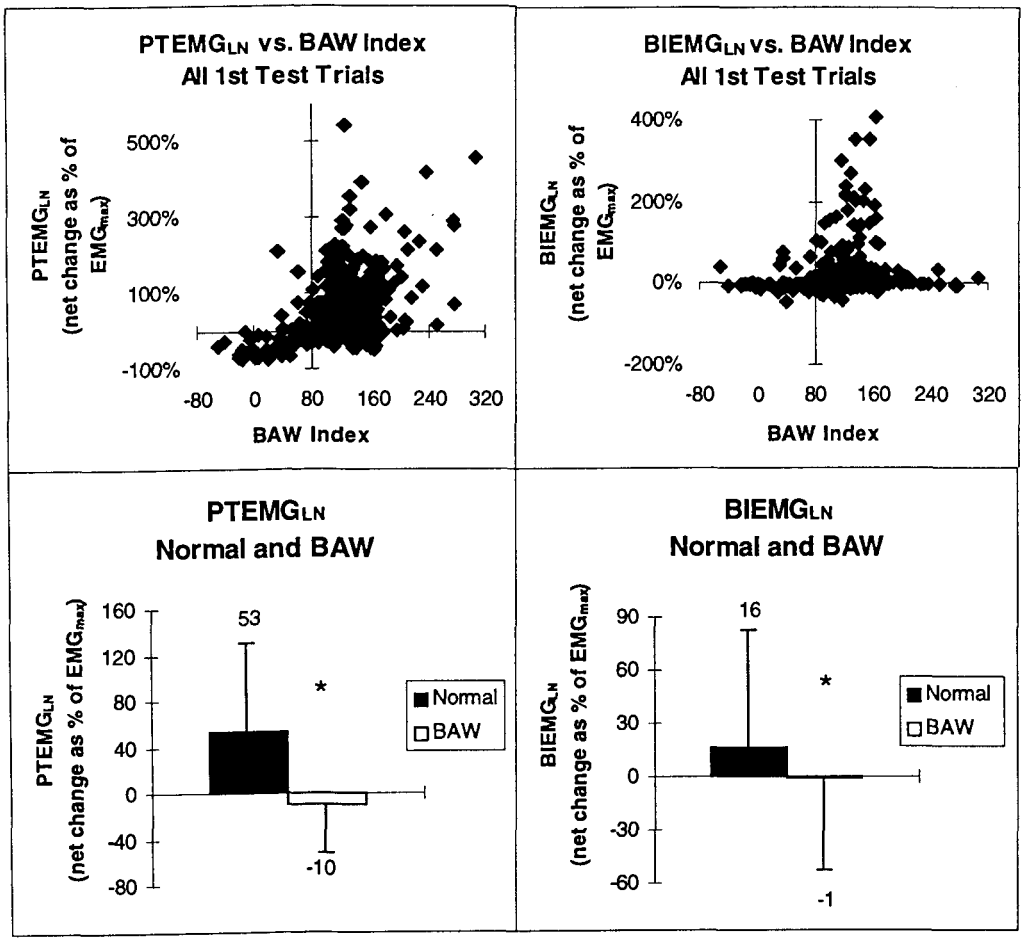

Figure 4.17. Top- $PTEMG_{LN}$ and $BIEMG_{LN}$ vs. BAW Index. There was a substantial decrease in PT and BI activity associated with BAW. Bottom- Mean values of $PTEMG_{LN}$ and $BIEMG_{LN}$ for normal and BAW trials. EMG in the last 100 ms of the "hold phase" was significantly smaller for BAW than for normal trials.

Is BAW associated with muscle fatigue?

The median frequency of PT and BI EMG prior to onset of handle rotation was calculated for the first and sixth rotation trial to determine whether or not there was a shift in the median frequency of the EMG signal from the beginning to the end of the test session. A shift in median EMG frequency from a high to a low value would be suggestive of muscle fatigue. Analysis of variance with repeated measures was performed to determine whether or not there was a significant difference in median frequencies between groups and whether or not there was a significant shift in median frequency from the first trial to the 6th trail. Differences were considered significant at $p \leq 0.05$.

The mean value of PT EMG median frequency before the first rotation was 99 Hz for normal limbs and was 102 Hz for BAW limbs. There was no significant difference in median frequency of PT EMG at the onset of testing between BAW and normal limbs. Median frequency of BI EMG before the sixth rotation was 97 Hz for normal limbs and 101 Hz for BAW limbs. There was no significant shift in median frequency in either of the 2 groups from the first to the sixth trial. The same trend was observed for BI EMG. The mean value of the median frequency of BI EMG at the onset of testing was 58 Hz for BAW limbs and 68 Hz for normal limbs, which was not significantly different. Before the sixth rotation trial, median frequency of BI EMG was 61 Hz for BAW limbs and 72 Hz for normal limbs. There was no significant shift in the median frequency

component of BI EMG from the first to the sixth trail. These data strongly suggest that muscle fatigue did not occur in the BI or PT from the first to the sixth trial. See figure 4.18.

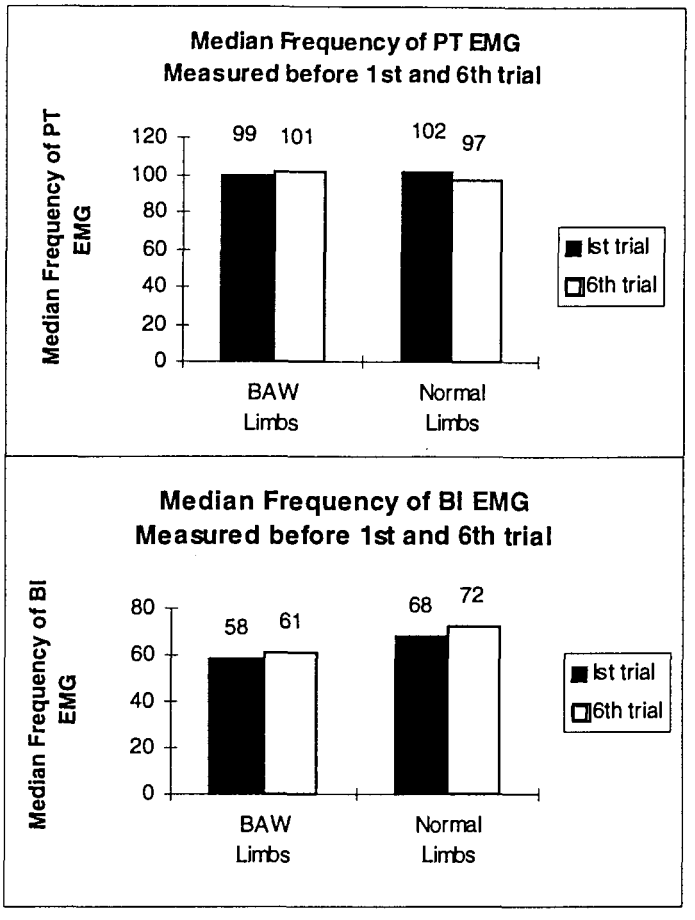

Figure 4.18. Median frequency of PT and BI before first and 6th rotation. Units of median frequency are Hz. There was no significant difference between the two groups with respect to median frequency of PT or BI before the first trial. There was no significant shift in median frequency of PT or BI from the first to the 6th trial in either of the 2 groups. (n=62, $p \leq 0.05$)

Is detection of BAW associated with trial order?

To determine whether or not BAW was associated with the number of trials that a subject had performed, we calculated the number of times that BAW was observed for the first time in each block of trials. The results are displayed

in Figure 4.0. An equal number of limbs (7) displayed BAW for the first time in either the first or the 2nd block of trials. Six limbs displayed BAW for the first time in the 3rd block of trials, and 3 limbs displayed BAW for the first time in the 4th block of trials. These data demonstrated that there was no tendency for subjects to begin to display BAW either early or late in the test session. One reason that a very small number of limbs showed BAW for the first time in the 4th block is that very few subjects performed 4 blocks of trials.

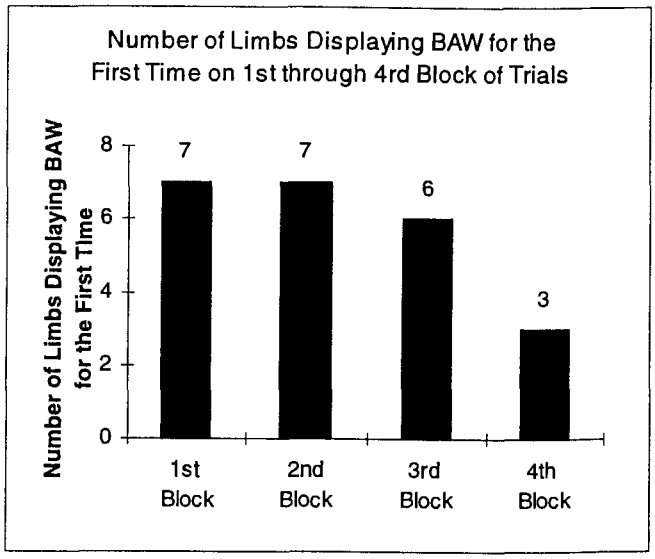

Figure 4.19. Number of limbs displaying BAW for the first time in the first, 2nd, 3rd, or 4th block of trials. Subjects were equally likely to display BAW for the first time in the early, late, or middle blocks of trials.

There was no pattern with respect to when BAW was observed. In some cases, subjects showed BAW early in the session and then did not show it again. In other cases, subjects showed it only late in the session. Sometimes subjects showed BAW throughout the test session, either intermittently or repeatedly.

Latency of BAW and Relationship to Clasp-knife Reflex

We evaluated individual BAW responses to estimated the time at which BAW occurred and to determine whether or not inhibitory clasp-knife mechanisms might be involved. A total of 17 cases of BAW from 9 different limbs were studied. Twelve cases had BAW Index <50 and were thought to be the cases in which latency would be easiest to estimate. It was thought that if latency detection was not possible in the most severe cases, then it would be impossible in less robust cases. Therefore, only 5 cases with BAW Index >50 were evaluated.

For this portion of the analysis, BI and PT EMG traces were quantified using a 20 ms moving RMS window and then low pass filtered at 20 Hz. Torque traces were low pass filtered at 20 Hz. All the normal responses from a particular limb were averaged, and each BAW trial was compared to the average of all the normal trials observed in the same limb. Hence, the average of a limb's normal trials served as a control for BAW trials. BAW trials were compared to the average of normal trials by visual inspection to detect the time at which torque and EMG began to deviate from normal. When there was a slight difference in initial torque between normal and BAW trials, the difference was subtracted from one trace so that the traces were matched from the start.

Latency of BAW was identified in the torque, PT EMG, and BI EMG traces whenever possible. Latency was first identified in the torque trace, and

then in PT and BI EMG. If the EMG and torque traces of the BAW trial closely resembled the normal trials prior to BAW, the point at which the 2 traces began to deviate from each other was considered the latency of onset of BAW in the respective traces. If the traces did not mimic each other prior to BAW, the point at which torque began to decrease substantially was considered the latency of BAW in the torque trace. The latency of BAW in PT EMG was a point prior to the onset of the decrease in the torque trace at which a substantial decrease in EMG occurred. The latency of BAW in BI EMG was any point in the BI EMG trace at which activity began to decrease substantially. The final latency of BAW was defined as the time at which PT EMG began to decline.

Figures 4.20 and 4.21 contain examples of traces in which latency was detected. Table 4.20 summarizes the results of all 17 cases that were examined. Latency values were presented with respect to onset of handle rotation. In most cases (14 out of 17), the rate of decline of PT EMG was gradual, and a distinct onset of BAW was very difficult to detect as in C83R37. (See Figure 4.21.) In 3 cases, EMG decline was so gradual that latency could not be estimated. In 4 other cases, the decline in PT EMG occurred rapidly, and a fairly reliable estimate of latency was obtained. (See C68R46 in Figure 4.20.)

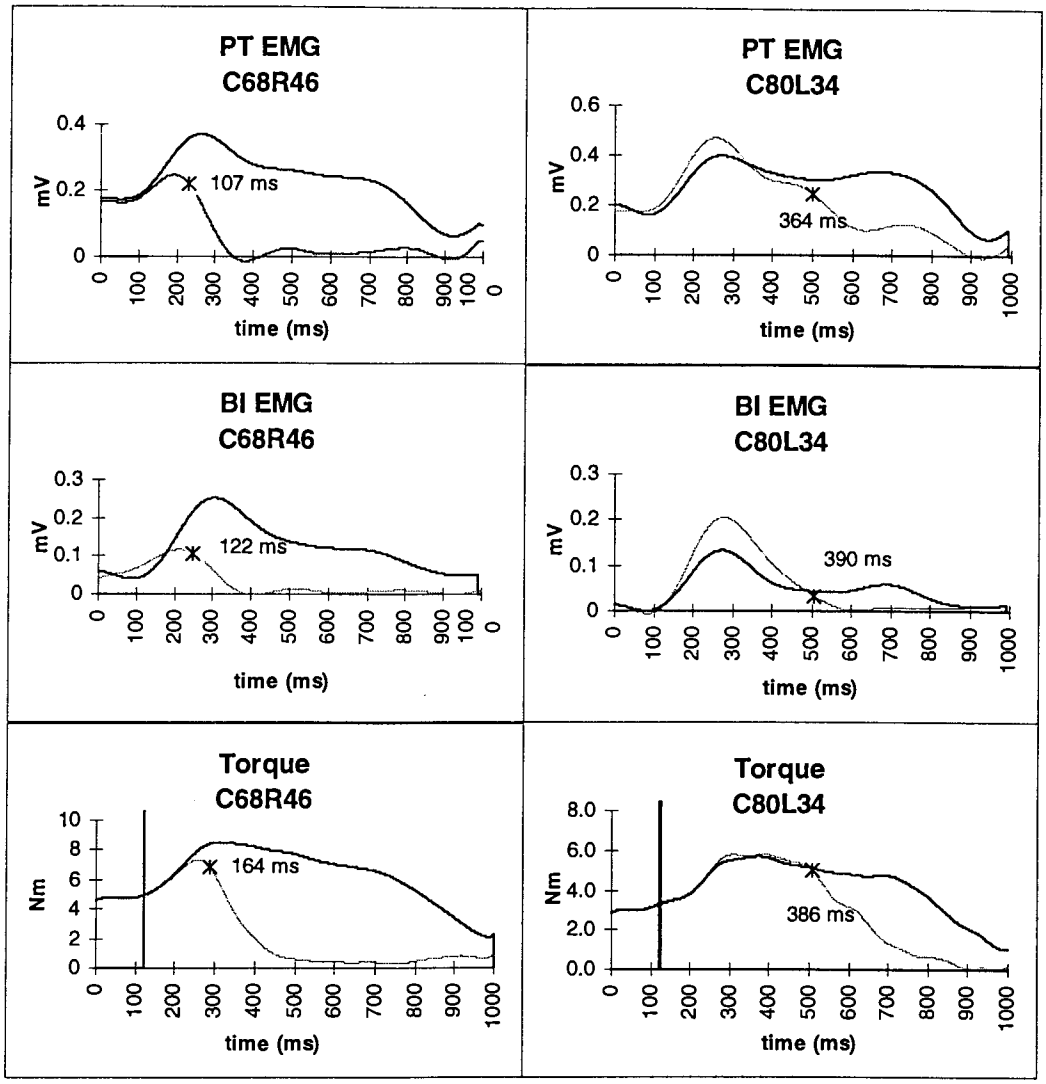

Figure 4.20. Two Examples of the Time Course of BAW. Heavy lines are normal traces, and light lines are breakaway traces. Vertical line in torque trace represents onset of handle rotation. Left- C68R46 was one of the most dramatic cases of BAW that was observed. Decreases in EMG and torque occurred rapidly and were easy to detect. BAW torque began to deviate from normal in the "ramp phase" of the rotation. Initial dynamic EMG responses were smaller in the case of BAW. Right- In C80L34, BAW occurred much later and more gradually. There was no difference in torque until the "plateau phase", when BAW began. Initial dynamic EMG responses were robust in the BAW case. In both cases, torque traces were shifted vertically to match initial values.

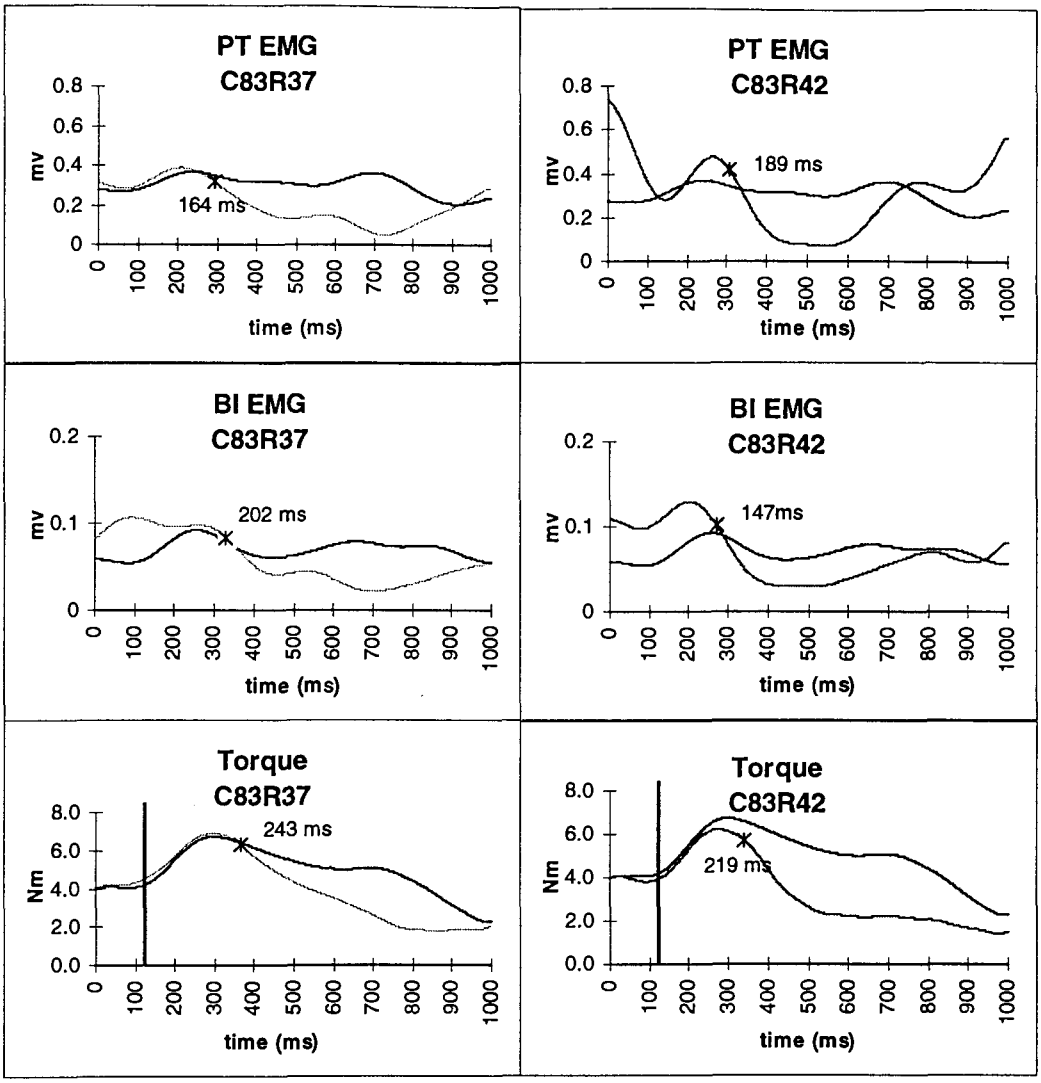

Figure 4.21. C83R37 Displayed More than 1 Example of BAW. The time course and pattern were different in each case. Left- Initial dynamic torque and EMG responses were the same in normal and BAW cases. Right- There was a larger dynamic EMG response, but a smaller dynamic torque response in BAW than in normal. In both cases, torque traces were shifted vertically to match initially.

According to our estimates, there was a great deal of variability among subjects with respect to latency of onset of BAW. Latencies ranged from 107 to 364 ms with a mean of 210 ms (std=71). Even when one limb displayed BAW more than one time, the time course and pattern of BAW was often different. Of the four BAW responses in which PT EMG decreased rapidly and a reliable estimate of latency was obtained, latencies ranged from 107 to 184 ms. These latencies are with in the time frame of both clasp-knife reflex and voluntary reaction time. Because of the long and variable latency of BAW, it was difficult to determine whether or not long latency clasp-knife reflexes could be involved.

Table 4.20 Latency of Onset of BAW.

Subject Code	Group	BAW Index	BAW Type	Latency of Decline in Torque (ms)	Latency of decline in PT EMG (ms)	Latency of decline in BI EMG (ms)	Rapid or Gradual Decline in PT EMG
C61R29	1	45	1	292	Unable to detect	409	gradual
C61R31		7	1	211	184	230	gradual
C64R35	1	-11	1	194	145	210	rapid
C68R46	1	-3	1	164	107	122	rapid
C80L33	1	65	1	303	Unable to detect	257	gradual
C80L34		45	1	386	364	390	gradual
C81L34	3	5	1	184	182	171	rapid
C81L40		-40	1	193	141	197	rapid
C93L17	3	29	1	303	262	Unable to detect	gradual
C93L20		32	2	313	278	BI increasing at end	gradual
C93L22		-51	2	311	221	BI increasing at end	gradual
C93L24		-6	2	336	284	BI increasing at end	gradual
C82R50	2	67	1	Unable to detect	Unable to detect	Unable to detect	gradual
C83R37	1	55	1	243	164	202	gradual
C83R40		56	1	233	207	255	gradual
C83R42		20	1	219	189	147	gradual
C99L42	2	72	1	Unable to detect	Unable to detect	Unable to detect	gradual
min--imum					107 ms		
max-imum					364 ms		
mean					210 ms		
std					71		

Almost all cases of BAW were associated with a decline in PT and BI activity. In most cases, PT declined before BI. Decreasing PT activity coupled with maintained BI activity may have resulted in a net increase in supination torque which could have partially accounted for BAW. Later, the reduction in

BI activity may have resulted in reduced co-contraction about the joint, contributing further to BAW by reducing the overall joint stiffness. There were 3 cases in which BAW was associated with an early decline in PT activity and a later increase in BI activity. In these cases, BAW could have started because of a decrease in PT activity and was later augmented by increased BI activity.

Discussion of Phase IV Results

Classification of Responses

In Phase IV, we were successful in developing a highly reproducible test protocol for detecting BAW and a quantitative method for classifying responses as normal or BAW. We were also able to identify four different types of BAW. However, the division between normal and BAW was arbitrary because we obtained a continuum of responses for BAW Index. There was no clear division between normal and BAW. These data strongly suggest that there was no obvious distinction between BAW and normal populations.

Reliability

Our results showed that the test/re-test reliability for detecting BAW was poor, despite the fact that the test protocol was highly reproducible and relatively impervious to influences from the examiner. These observations suggest that the test was not flawed. Rather, BAW occurred only sporadically and infrequently and was, therefore, difficult to detect repeatedly.

We evaluated eight different criteria for detecting BAW and chose to classify responses according to the criterion that had the highest sensitivity and the highest rate of detection. An inverse relationship exists between sensitivity and reliability: as sensitivity increases, reliability decreases. The reliably of our test procedure would have improved if we had chosen to reduce the sensitivity of the test. Furthermore, because of the high sensitivity, it is likely that we detected a number of false positives (i.e. false BAW detections). However, in the absence of a gold standard, it is impossible to estimate the number of false positives that may have been obtained. The observation that BAW occurs sporadically may suggest that reliability would improve if more rotations were delivered, and subjects had more chances to displaying it. This approach is somewhat unrealistic, however, because subjects tire quickly and would be unlikely to be able to perform substantially more trials. Furthermore, from a diagnostic testing standpoint, it is impractical administer a test that is highly time consuming. Because of its inherently poor test/re-test reliability, we cannot recommend that this test be used for diagnostic purposes. Further investigation into BAW should not been done unless a different approach that can clearly distinguish between groups is developed.

Prevalence

Despite these reliability results, we chose to evaluate the prevalence of BAW in the enriched, semi-enriched, and control subjects. We observed a very

high proportion of BAW in each of the 3 groups. Overall, 69% of all subjects that were tested displayed BAW at least once in either the first or re-test session. Almost all control subjects (70%) displayed BAW on either the first or re-test session, which was not different from the prevalence in either of the other two groups. These data suggest that BAW, as defined here, is common, not limited to a particular group of individuals, and probably not abnormal.

Another important finding was that there was no difference in the prevalence of BAW between the control and semi-enriched groups. Recall, that both groups lived outside the LFV but differed with respect to injury status. The semi-enriched group had lower extremity injuries, and the control group was injury-free. We found no difference in prevalence of BAW between the semi-enriched and control groups which strongly suggests that BAW in the PT was not related to injuries of the lower extremities or back. This finding was counter to Sweeting's clinical observation that BAW was related to musculoskeletal injuries throughout the body.

Subjects from the enriched group displayed BAW with a higher degree of consistency than subjects from other groups. Although the difference was not significant, enriched subjects had a tendency to display bilateral BAW more often than subjects from the other 2 groups. Furthermore, enriched subjects were significantly more likely to display BAW on *both* the first and re-test sessions than either of the other 2 groups. It is possible that this difference was

due to residence in the LFV, but it is more likely that these results occurred because enriched subjects were not blind (they knew why they had been selected for the study) and may have been trained inadvertently by Sweeting to respond with BAW. It is also possible that enriched subjects felt that there may have been an advantage to testing positive for BAW. Some subjects may have been injured at work or may have felt frustration from the lack of a clinical diagnosis to account for their symptoms.

Quantitative Measures

Similarities and Differences in Initial Conditions

Subjects with BAW had significantly smaller values for T_{in} than normal subjects. This observation suggests that subjects with BAW had less isometric muscle strength than normal subjects. More female subjects displayed BAW than male subjects, which may have accounted for this observation. Weakness may have accounted for their inability to maintain the specified level of torque under the additional demand of handle rotation. Contradicting this interpretation was the observation that BAW affected both the strongest and the weakest subjects. This observation suggests that BAW did not exclusively affect weak subjects. There was no difference in initial EMG of PT and BI between BAW and normal responses. Both groups displayed initial values for PT EMG that were approximately 40% of maximum and initial values for BI EMG that were approximately 15% of maximum. Both groups displayed approximately

the same degree of co-contraction about the elbow. Hence, a deficit in initial EMG output was unlikely to have accounted for BAW. Impaired initial muscle activation was unlikely to have accounted for isometric muscle weakness.

Similarities and Differences in Initial Dynamic Responses to Handle Rotation

The dynamic torque and EMG responses during handle rotation were similar for normal and BAW responses. In response to handle rotation, both the normal and BAW groups displayed increases in torque that were the same percentage of T_{in}. However, t_{Tpk} and POS_{Tpk} were both significantly smaller in BAW responses which suggests that BAW responses achieved the same percent of T_{in} in less time or after less handle rotation. Both groups displayed convincing evidence of PT stretch reflex activity and reciprocal inhibition of BI that were equal in magnitude. These data confirmed our previous observation that stretch reflex in PT was normal in subjects with BAW and further suggests that the initial dynamic torque response and reciprocal inhibition in BI were also normal in subjects with BAW. These data strongly suggest that neither abnormal PT stretch reflex nor abnormal reciprocal inhibition of BI were responsible for BAW.

Similarities and Differences in Later Responses to Handle Rotation

BAW responses had a larger and faster yield than normal responses. T_{minyN} was significantly smaller and rate of change of torque was significantly larger in BAW responses than in normal responses. These data suggest that

BAW responses deviated from normal responses as early as the "yield phase" and that BAW may have begun as early as the "yield phase" of the torque response, but could not yet be detected. PT EMG prior to T_{pk} was slightly larger than initial EMG and was the same for normal and BAW responses. However, BI EMG prior to T_{pk} was significantly smaller in BAW responses and was near initial values. These data suggest that reduced activation of BI prior to T_{pk}, resulting in reduced co-contraction and less stiffness about the joint may have contributed to BAW.

It was very clear that, on average and in almost all cases of BAW, PT and BI EMG in the last 100 ms of the "hold phase" were significantly reduced. These data strongly suggest that reduced activation of PT and BI EMG were responsible for BAW. The mechanism(s) underlying reduced muscle activation remain unclear, however. Possible mechanisms include activation of clasp-knife reflex pathways, reduction in voluntary effort, and muscle fatigue.

Muscle Fatigue

It is unlikely that muscle fatigue contributed to BAW because there was no evidence of muscle fatigue by the 6th rotation trial, as measured by frequency spectrum analysis. There was no tendency for BAW to occur in later trials compared to earlier trials. The test protocol was designed to minimize fatigue in that subjects were encouraged to rest as long as necessary between trials. We were certain that there was no decrease in torque output with

repeated trials because the handle would not rotate unless the subject was producing the specified level of effort. Nevertheless, there may have been an increase in the perceived level of effort or an increase in the contribution from other muscles to produce torque.

Clasp-knife Reflex and Other Possible Mechanisms

It was difficult to determine whether or not BAW may have been caused by activation of clasp-knife pathways because the decrease in EMG was usually gradual, and the onset was difficult to detect. In 2 cases that we examined, the latency of BAW was estimated to be <150 ms, which is within the range of clasp-knife reflex. However, the latency data that exists for clasp-knife reflex is limited to reduced cat preparations. The average latency of onset of BAW was 210 ms, which is well within the range of voluntary reaction time. From a qualitative standpoint, it is possible that clasp-knife reflex may have been involved in BAW because the stimuli (high muscle force and muscle stretch) were the same as those used to elicit clasp-knife reflex in brain injured humans and reduced animal preparations. Furthermore, these stimuli are capable of exciting the FNE that most likely mediate clasp-knife reflex. Like clasp-knife reflex, BAW was less apparent at lower levels of force. Other characteristics of clasp-knife reflex (such as activation through gentle tendon manipulation and similarity to flexion withdrawal reflex) could not be assessed in this experimental set-up.

We observed a great deal of individual variability with respect to EMG and torque shutdown, as evidenced by the variability in latency and the 4 types of BAW that were observed. Variability in latency and pattern of muscle deactivation may suggest that non-reflex mediated mechanisms were underlying BAW. We are confident that all subjects were trying very hard to perform the test properly, but the test was very challenging. Subjects may have inadvertently shutdown muscle activation simply because it was easier than continuing to maintain torque against the strong rotation.

At this point, we are unable to pinpoint the mechanism(s) responsible for BAW. There is clearly a reduction in activation of PT and BI associated with BAW that may be caused by a combination of mechanisms which include:

1. Activation of clasp-knife reflex pathways.

2. Inadvertent shutdown of voluntary muscle activation.

3. Reduced activation of BI leading to reduced joint stiffness.

4. Isometric muscle weakness.

From a neuromuscular standpoint it is difficult to determine whether or not BAW was an abnormal muscle response to stretch because even if it does represent a case in which clasp-knife reflex is active, it does not necessarily suggest that it is pathological. The role of clasp-knife reflex in non-

neurologically impaired humans is not known. One can imagine how a sudden decrease in EMG and torque about a joint could either hinder performance if it occurred at inappropriate times, or protect muscle from injury if it had been overloaded.

Conclusions of Phase IV

In this phase, we were successful in using an objective and reproducible test to assess for presence or absence of BAW. We developed a quantitative method of classifying responses as normal or BAW. However, there was no clear distinction between these 2 groups. The reliability of the test was poor due to the intermittent nature of BAW. Hence, we cannot recommend that this testing method be used to diagnose BAW.

We found that BAW occurred in all 3 groups that were tested. Approximately 69% of all subjects displayed BAW at least 1 time, suggesting that it is too common to be pathological. There was no evidence that BAW was related to injuries in the lower extremities or back. Subjects living in the LFV displayed BAW more consistently than subjects from outside the LFV. This result could have been due to their residence or association with Sweeting. However, it is most likely that this result occurred because subjects were not blind and had been inadvertently trained to BAW.

The mechanisms underlying BAW remain unclear. It is associated with a substantial reduction in pronation torque, PT EMG, and in most cases BI EMG. BAW is clearly not caused by an abnormality of the PT stretch reflex or reciprocal inhibition of BI. It may be related to isometric muscle weakness, reduced co-contraction about the joint, clasp-knife reflex activation or inadvertent shutdown of voluntary muscle activity.

General Discussion

This thesis was the first in-depth research where the main focus was to develop a novel testing approach and apparatus that was capable of assessing muscle response to stretch at high levels of force. This characteristic of muscle performance had not been evaluated quantitatively in the past. The purpose of this project was to develop an objective, quantitative, and reliable testing method that was capable of distinguishing between normal and BAW responses and to describe these two types of responses in order to understand underlying mechanisms. We also investigated whether or not BAW was related to residence in the LFV and injury of the lower extremities and back.

Test Development

Prior to Sweeting's introduction of a manual test for BAW, muscle strength had been assessed clinically by isometric manual muscle testing, concentric or eccentric isokinetic testing, concentric or eccentric isotonic muscle testing, and functional evaluation (Kendall and McCreary, 1983; Bohannon, 1986; Miller *et al.*, 1988; Wadsworth *et al.*, 1987). With the manual test for BAW, Sweeting introduced the concept of assessing strength by evaluating subjects' ability to maintain muscle force when producing high force and challenged with a rapid increase in load. This manual test had serious limitations because it was neither quantitative nor objective. Furthermore, Archibald and Mathias (1991) found that it was unreliable and not capable of distinguishing between subjects

previously diagnosed with BAW and healthy control subjects from the general community.

The test apparatus that we developed was a considerable improvement over the manual test because it provided the same input to all subjects, was not prone to examiner bias, and provided quantitative measures of EMG, torque, joint position, and timing. The protocol provided a quantitative assessment of severity of responses by way of the BAW Index. This mechanical test would be simple for examiners to learn and to administer properly, although it was not attempted in this study.

Test Optimization

Considerable time and effort was dedicated to developing a mechanical test that could distinguish between the BAW and normal populations. Despite these efforts, the test was unable to clearly distinguish between two populations. The optimization phase of this study (Phase III) was dedicated to developing a test that could detect BAW in those thought to have it, while not detecting BAW in so-called normal subjects. A major difficulty with this task was the absence of a gold standard (independent testing method known to be valid and reliable) to which our results could be compared. Although we were aware of the shortcomings of the manual test, we used it as a gold standard and attempted to

develop a test that elicited BAW in subjects diagnosed by Sweeting, while not eliciting BAW in healthy control subjects.

In Phases I and II, we found that BAW was sometimes present in subjects referred by Sweeting and was never present in control subjects. Later in Phases III and IV, we detected BAW in many control subjects. There are several possible reasons for these conflicting results. In the first two phases, control subjects consisted of Kinesiology graduate students who were very athletic, exceptionally motivated, not blinded, and familiar with the anticipated results. Many of these students viewed the test as a contest to see who was the strongest of the group. The patients referred by Sweeting were less active, probably less motivated, and may have been inadvertently conditioned to respond with BAW because they had been manually tested and told that they had BAW. In the last two phases, control subjects were matched to patients with respect to age and activity level. Control subjects were selected from the general community and were not familiar with the reasons for testing. These results suggest that control subjects from the initial phases were exceptionally good at performing the test. When matched control subjects from outside the Graduate Program in Kinesiology were evaluated (as in Phases III and IV), we observed that BAW was present in control subjects as well as patients.

In the optimization phase of these experiments (Phase III), subjects referred by Sweeting sometimes failed to display BAW, and control subjects

sometimes displayed BAW. These observations were interpreted as shortcomings of the mechanical test, and further test development was carried out to converge on an optimal test. Finally, optimization was terminated when it became increasingly apparent that no particular test was better able to distinguish between the groups. In view of our more recent Phase IV results which showed that test/re-test reliability was poor, a different explanation may be possible.

Table 3.2 (p. 57) summarizes the data from 3 control subjects and 5 patients who were tested with the original rotations in Phase II and three new rotations in Phase III to see if one particular rotation emerged as best for detecting BAW. (One of the rotations in Phase III was the same as the step rotation used in Phase II.) There were 3 control subjects and 1 patient who never showed BAW with any rotation which reflects a high degree of reliability in diagnosis. However, the other four patients displayed changes in diagnosis. Two patients went from NO BAW to BAW, one subject went from bilateral BAW to unilateral BAW, and one subject went from unilateral BAW to bilateral BAW. Some of the changes in classification may be attributed to new tests, but all four subjects displayed at least one conflicting diagnosis when tested in Phases II and III with the same rotation. In other words, subjects responded differently to the same rotation administered on two different occasions. These data can now be interpreted as further evidence of the sporadic occurrence of BAW and the lack

of reliability in detecting it. We feel that the inability to develop a reliable and selective test for BAW was not a shortcoming of our development approach, but was due to the inconsistent occurrence of BAW and the lack of a gold standard.

Continuum of Responses

Sweeting's clinical observation that muscles are sometimes able to resist rapid stretch and sometimes "give out" when rapidly stretched has been confirmed in this study. However, there were not 2 distinct types of responses as he initially suggested. Instead, we have identified a continuum of responses with dramatic "giving away" at one end, robust resistance to "giving away" at the other end, and all gradations in-between. The spectrum of responses is represented by the wide range of values for BAW Index with no clear "break" in the spectrum of responses that would suggest that a natural division between groups existed. Previous methods of strength testing have not assessed this "dynamic" characteristic of muscle performance, so it is useful to know that muscles may behave at different points on this continuum. Depending on the functional situation, this behavior may interfere with function or protect muscle from damage caused by high force and stretch (Rymer *et al.*, 1979).

Reliability of Test

An important finding in this study was that, despite the fact that the mechanical test was objective, quantitative and reproducible, it proved to be no more reliable than the manual test for detecting BAW and no more capable of

distinguishing between populations. In this respect our results support those of Archibald and Mathias (1991). We feel that the poor reliability was due to the sporadic occurrence of BAW, not a shortcoming of test development. We cannot recommend that this mechanical test be used for diagnostic purposes because it is neither reliable nor capable of classifying subjects into two distinct groups.

Prevalence of BAW and Relationship to LFV and Lower Limb Injury

In Phase IV, there was a high observed prevalence of BAW in all 3 groups that were tested (enriched, semi-enriched, and control). In either the first or re-test session, 70% of controls, 58% of semi-enriched subjects and 80% of enriched subjects displayed BAW. These proportions are very high and not significantly different which suggests that all three groups were equally likely to display BAW at least one time. Furthermore, the high prevalence suggests that BAW, as we have defined it, it too common to be considered pathological. Furthermore, the fact that we found no difference in prevalence between injured and non-injured subjects living outside the LFV strongly suggests that BAW in PT is not related to low back or lower limb injuries. These data are counter to Sweeting's clinical observation that subjects with BAW are prone to musculoskeletal injury.

Subjects from the enriched group displayed BAW with more consistency than subjects from the other two groups, as evidenced by the significantly larger proportion of enriched subjects displaying BAW on two consecutive test sessions. High observed consistency of BAW in the enriched group may have

been due to residence in the LFV or association with Sweeting because these are two things that differentiated this group from the other two. It is more likely, however, that enriched subjects were consistent in displaying BAW because they had been inadvertently trained to BAW, were not blind to the purpose of the experiment, knew why they had been selected for the study, and had been previously told that they have BAW.

This study originated from the suspicion that people living in the LFV may be exposed to environmental toxins that cause BAW. It is important to point out that this study did not address the issue of pesticide exposure in people who live in the LFV. Our goal was to develop a test for BAW and to estimate the prevalence in three groups of subjects. The fact that we observed a higher consistency of BAW in subjects who were from the LFV does not suggest that they have been exposed to toxins or that BAW was caused by toxins. We did not perform biochemical testing to determine whether or not the LFV subjects had a higher exposure to toxins than people living outside the LFV.

If future investigators wish to pursue the relationship between BAW and toxin exposure, we recommend that a different and more reliable testing method be developed before any other experimentation begins. However, we feel that at better test does not exist because BAW does not appear to be a phenomenon that can separate subjects into two distinct populations. We also suggest that a more appropriate way of evaluating the relationship between BAW and pesticide

exposure is to perform testing on pesticide applicators and non-applicators. Applicators have a high likelihood of pesticide exposure and have been shown to have levels of toxins above those of the normal population (Stokes *et al*, 1995). We further recommend that biochemical testing for toxins be performed to establish a correlation between BAW and pesticide exposure.

Mechanisms Underlying BAW

As discussed in detail in *Discussion of Phase IV Results*, the mechanisms underlying BAW remain unclear. Our data strongly suggest that BAW was caused by a reduction in EMG in PT and BI that resulted in a reduction in pronation torque, but the cause of reduction in muscle activity was not clear. Our data have shown that BAW was not caused by an abnormal PT stretch reflex or abnormal reciprocal inhibition to BI. Furthermore, initial EMG activation of both the PT and BI was not different in BAW and normal responses and were unlikely to have been responsible for BAW.

It is possible that the reduction in EMG activity was caused by activation of a clasp-knife reflex. This long latency reflex, that resembles flexion withdrawal reflex, has previously been identified in neurologically impaired humans and reduced animal preparations, but has never been detected in neurologically normal humans (Burke *et al.*, 1971a, b; Sherrington, 1909 in Burke *et al*, 1972; Burke *et al.*, 1972; Rymer *et al.*, 1979; Cleland and Rymer, 1990; Cleland *et al.*, 1990). BAW resembles clasp-knife reflex in that BAW is characterized by a

rapid and substantial decline in torque and EMG that occurs when muscle produces high force and undergoes rapid stretch (Rymer *et al*, 1979). Like clasp-knife reflex, BAW becomes less apparent at low levels of background force (Cleland and Rymer, 1990). In some cases of BAW, the latency of EMG reduction was consistent with the latency of clasp-knife reflex which is 106-138 ms in cat (Cleland and Rymer, 1990). However, it is difficult to attribute EMG shutdown to clasp-knife reflex because the latencies of clasp-knife reflex and voluntary reaction time are overlapping. Furthermore, lack of human data on clasp-knife reflex forces us to make comparisons with latency of clasp-knife reflex in animals.

Our results showed that T_{in} in BAW subjects was smaller than T_{in} normal subjects. This observation suggests that subjects with BAW may be weaker than normal subjects and that BAW may be related to isometric muscle weakness. Weak muscles may be less able to resist the additional challenge of the rapidly turning handle. However, a larger number of female subjects displayed BAW than male subjects which may have accounted for this result. Furthermore, the task was very difficult. Subjects may have inadvertently decreased their effort because it was easier than continuing to maintain force against the rapidly turning handle. Our data cannot confirm or refute these hypotheses. Given the difficult nature of the task and the inconsistent latency and time course of BAW,

it is possible that inadvertent shutdown of voluntary EMG and torque was partially responsible for BAW.

In the early phases of this research, it was thought that BAW may be influenced by muscle fatigue or may require a conditioning input to activate a polysynaptic pathway. Initially, BAW was never observed on the first rotation trial. The clinical observation was that BAW was often easier to detect after two or three manual test trials. The results from Phase IV failed to confirm these initial observations. There was no pattern of detection of BAW with respect to trial order. Subjects displayed BAW on first, last, and middle trials. Data from spectral analysis of EMG suggest that subjects did not experience muscle fatigue between the first and sixth trial. Furthermore, the test protocol was designed to minimize fatigue. Subjects were allowed to rest as long as needed between trials, and rotations were not administered unless the specified level of torque was achieved. It is possible that fatigue was present and was not detected in analysis, but there is no evidence to support this. One difference between the Phase IV protocol and the protocols used in other phases was that in Phase IV, subjects were given at least 1 practice trial at 50% MVC. It is possible that this difference may have accounted for the difference in results. At this time, we are unable to resolve the discrepancy in observations that BAW seemed to be related to trial order in early experiments but not in later experiments.

The Role of Clasp-knife Reflex in Normal Movement

Although it is possible that clasp-knife reflex may play a role in normal human movement, it has only been identified in brain injured humans and reduced animal preparations. Nearly all the quantitative data describing clasp-knife reflex has been obtained from the animal model. It is an interesting possibility that clasp-knife reflex may be present in non-neurologically impaired humans in such a form as BAW. It may function to protect muscle from being injured when it is overloaded, or it may cause a reduction in muscle force at times when high force is required. Continued investigation into the role of clasp-knife reflex in normal movement may shed light on its role in motor control. To begin to understand the role that clasp-knife reflex may play in normal movement, studies should focus on understanding, quantifying, and describing the characteristics and behavior of clasp-knife reflex in neurologically impaired humans. Only after we understand human clasp-knife reflex will it be possible to begin to understand its role in normal movement and whether or not it is involved in such a phenomenon as BAW.

BAW: Normal or Abnormal?

Our data do not suggest that BAW is an abnormal or pathological response for a number of reasons.

1. This classification approach was not capable of separating responses or individuals into two distinct groups.

2. BAW was detected in the majority of subjects that we tested including the control subjects who had no demonstrable pathology, and

3. It was not possible to identify a pathological mechanism that is responsible for BAW.

If it was possible to determine that BAW is mediated by clasp-knife reflex, it would still be possible that activation of this inhibitory pathway was normal. One can see that clasp-knife reflex could either be beneficial or detrimental to movement depending the conditions under which it occurred. It is possible that clasp-knife reflex may have a protective role, functioning to protect muscles and tendons from unexpected, rapid increases in force and length by causing limb collapse (Rymer et al., 1979). Alternatively, sudden reduction in muscle activity and force might lead to injury if it occurred when muscle activity was required, as in descending stairs rapidly or landing from a jump. However, these practical examples are different from the BAW test scenerio in that muscles would not have been activated maximally or for a sustained period of time in the practical example.

Because there is no evidence that BAW is an abnormal phenomenon, we propose that breakaway weakness be renamed so that the word "weakness" is

omitted and that the name reflects the continuum of responses that were observed. It seems appropriate to refer to the continuum of possible observations as *resistive* and *non-resistive* responses. Labeling resistive and non-resistive responses according to BAW Index will allow quantification of the extent to which the response was more or less resistive.

Conclusions

In this study we developed a novel computer-controlled motorized apparatus that was capable of detecting and quantifying resistive and non-resistive responses to stretch in wrist muscles. These two responses were formerly referred to as normal and breakaway weakness. A non-resistive response was characterized by a substantial decrease in PT and BI EMG and pronation torque that occurred when muscles were producing high force and underwent a rapid stretch. Our test apparatus and classification procedure identified a wide range of responses and was not capable of distinguishing between impaired and normal populations. This result suggests that presence or absence of a non-resistive response did not separate a population into two distinct groups.

A non-resistive response was detected at least once in 69% of all subjects (impaired and normal) which suggests that it may be too common to be considered a pathology. The test/re-test reliability of our test procedure was poor, largely attributable to the sporadic occurrence of non-resistive responses. For these reasons, we do not recommend that our test for resistive and non-resistive responses to muscle stretch be used for diagnostic purposes. There was no evidence that a non-resistive response in PT was associated with musculoskeletal injuries in the lower limbs or back in subjects living outside the LFV. Injured subjects from the LFV were more likely to display a non-resistive

response on two consecutive test sessions than subjects from outside the LFV. This result may suggest that non-resistive responses were related to residence in the LFV, but did not suggest that they were related to pesticide exposure.

Our results show that the non-resistive response was associated with reduced activation of PT and BI muscles. However, mechanisms remain unclear. Short latency stretch reflex pathways of PT and reciprocal inhibition of BI were normal in subjects with non-resistive responses. Possible mechanisms involved in non-resistive responses include inhibitory clasp-knife reflex pathways, reduced co-contraction about the elbow, isometric muscle weakness, and inadvertent shutdown of voluntary muscle activity.

List of References

Abrams, V.C., Lynn, B.,Richmond, F.J.R. (1984) Organization and sensory properties of small myelinated fibres in the dorsal cervical rami of the cat. *J. of Physiology*, 347, 177-187.

Angel, R. W. (1983) Muscular contractions elicited by passive shortening in *Motor Control Mechanisms in Health and Disease.* J.E. Desmedt, Ed. Raven Press, New York, 1983.

Angel, R. W. (1982) Shortening reaction in normal and parkinsonian subjects. *Neurology* (Ny). 32, 246-251.

Archibald, Chris and Mathias, Richard (1991) An investigation into a cluster of muscle weakness in the Fraser Valley, British Columbia. Internal report for Ministry of Health, B.C.

Baker, B.A. (1986) The muscle trigger: evidence of overload injury. *J. of Neurological and Orthopaedic Medicine and Surgery.* 7(1), 35-44.

Bohannon, R.W. (1986) Manual muscle test scores and dynanometer test scores of knee extension strength. *Arch of Phys Med Rehabil.* 67(6), 390-392.

Burke, D., Knowles, L., Andrews, C., Ashby, P. (1972) Spasticity, decerebrate rigidity and the clasp-knife phenomenon: An experimental study in the cat. *Brain.* 95, 367-372.

Burke, D., Andrews, C., Ashby, P. (1971b) Autogenic effects of static muscle stretch in spastic man. *Arch. Neurol.* 25, 367-373.

Burke, D., Andrews, C., Gilles, J.D. (1971a) The reflex response to sinusoidal stretching in spastic man. *Brain.* 94, 455-470.

Burke, D., Gillies, J.D., Lance, J.W. (1970) The quadraceps stretch reflex in human spasticity. *J. Neurol, Neurosurg and Psych.* 33, 216-223.

Caldwell, G.E. and Van Leemputte, M. (1991) Elbow torques and EMG patterns of flexor muscles during different isometric tasks. *Electromyography and Clinical Neurophysiology.* 31, 433-445.

Cath, C.L. and Crago, P.E. (1982) Low force feedback gain in the stretch reflex of the decerebrate cat following dorsal hemisection of the spinal cord. *Soc. Neuroscience Abstr.* 8:148.11.

Cleland, C., Hayward, L., and Rymer, W.Z. (1990) Neural mechanisms underlying the clasp-knife reflex in the cat. II. Stretch-sensitive muscular-free nerve endings. *J. Neurophysiology.* 64, 1319-1330.

Cleland, C.L. and Rymer, W.Z. (1990) Neural mechanisms underlying the clasp-knife reflex in the cat. I. Characteristics of the reflex. *J. Neurophysiology.* 64(4), 1181-1191.

Cleland, C.L. and Rymer, W.Z. (1993) Functional properties of spinal interneurons activated by muscular free nerve endings and their potential contributions to clasp-knife reflex. *J. Neurophysiology.* 69(4), 1181-1191.

Dawson-Saunders, B and Trapp, R. (1994) *Basic and Clinical Biostatistics.* Appleton and Lange, Norwalk, Connecticut, Second Addition.

Grigg, P., Schaible, H.G., Schmidt, R.F. (1986) Mechanical sensitivity of group III and IV afferents from posterior articular nerve in normal and inflamed cat knee. *J. Neurophysiology.* 55, 635-643.

Hayward, L., Wesselmann,U, and Rymer, W.Z. (1991) Effects of muscle fatigue on mechanically sensitive afferents of slow conduction velocity on the cat triceps surae. *J. Neurophysiology.* 65(2), 360-370.

Hoffer, J.A. and Andreassen, S. (1981) Regulation of soleus muscle stiffness in premammillary cats: intrinsic and reflex componants. *J. Neurophysiology.* 45, 267-285.

Hoffer, J.A., Leonard, T.R., Cleland, C.L., and Sinkjaer, T. (1990) Segmental reflex action in normal and decerebrate cats. *J. Neurophysiology.* 64, 1611-1623.

Hussain, M.A. (1979) Studies in Incipient and delayed neurotoxic effects of some organophosphorous insecticides. Ph.D. Thesis, Simon Fraser University.

Iles, J.F., Jack, J.J., Kullman, D.M., Roberts, R.C. (1989) The effects of lesions on autogenetic inhibition in the decerebrate cat. *J. Physiology.* 419, 611-625.

Karalliedde, Lakshman and Henry, John A, (1993) Effects of Organophosphates on Skeletal Muscle. *Human and Experimental Toxicology.* 12, 289-296.

Kelly, Sean S., Mutch, Elaine, Williams, Faithe M., Blain, Peter G. (1994) Electrophysiological and biochemical effects following single doses of organophospates in the mouse. *Arch. of Toxicology.* 68, 459-466.

Kendall, P.K. and McCreary, E.K. (1983) *Muscles: testing and function.* Williams and Wilkins, Baltimore, 3rd edition.

Matthews, B.H.C. (1933) Nerve endings in mammalian muscle. *J. Physiology.* 78, 1-53.

Matthews, P.B.C. (1972) *Mammalian muscle receptors and their central actions.* London: Arnold.

Mense, S., and Meyer, H. (1985) Different types of slowly conducting afferent units in cat skeletal muscle and tendon. *J. of Physiology.* 363, 403-417.

Miller, L.C., Michael, A.F., Baxter, T.L., Kim, Y. (1988) Quantitative muscle testing in childhood dermatomyositis. *Arch Phys Med Rehabil.* 69(8), 610-3.

Nichols, T.R. and Houk, J.C. (1976) Improvement in linearity and regulation of stiffness that results from actions of stretch reflex. *J. Neurophysiology.* 39: 119-142.

Rymer, W.Z., Houk, J.C. and Crago, P.E. (1979) Mechanisms of the clasp-knife reflex studied in an animal model. *Exp. Brain Research.* 37, 93-113.

Shepherd, C. (1993) Organophosphate pesticides-cause for concern? *The Practitioner.* 237, 212-214.

Stacey, M.J. (1969) Free nerve endings in skeletal muscle in cat. *J. Anatomy.* 105(2), 231-254.

Stokes, L., Stark, A., Marshall, E., Narang, A. (1995) neurotoxicity among pesticide applicators exposed to organophosphates. *Occupational and Environmental Medicine.* 52, 648-653.

Teschke, K., Kelly, S., Wiens, M., Hertzman, C., Ward, H., Ward, J. and Van Oostdam, J. (1992) Organochloride pesticides and polychlorinate biphenyls in the adipose tissue of British Columbia residents. Report to the British Columbia Ministry of Environment.

Toft, E., Sinkjær, T., Andreassen, S. (1989) Mechanical and electromyographical responses to stretch of the human anterior tibial muscle at different levels of contraction. *Exp. Brain Research.* 74, 213-219.

Wadsworth, C.T., Krishnan, R., Sear, M., Nielsen, H. J. (1987) Intrarater reliability of manual muscle testing and hand-held dynametric muscle testing. *Physical Therapy.* 67 (9), 1342-1347.

Weinbaum, Z., Schenker, M., O'Malley, M., Gold, E., Samuels, S. (1995) Determinants of Disability in Illness Related to Agricultural Use of Organophosphates (OPs) in California. *Am J. of Industrial Medicine.* 28, 257-274

Winters, J. and Kleweno, D. (1993) Effect of initial upper limb alignment on muscle contributions to isometric strength curves. *J. Biomechanics.* 26:2, 143-153.

Appendix: Glossary of Terms

$\%T_{incr}$- Percent Torque Increase

BAW- Breakaway Weakness

BI- Biceps

EMG- Electromyograph

EMG_{in}- Initial value of EMG

EMG_{inN}- Initial value of EMG, normalized to EMG_{max}

EMG_{L}- Value of EMG during last 100 ms of "hold phase"

EMG_{LN}- Net value of EMG during last 100 ms of "hold phase", normalized to EMG_{max}

EMG_{max}- Maximum value of EMG

EMG_{R}- Value of EMG during stretch reflex latency

EMG_{RN}- Net value of EMG during stretch reflex latency, normalized to EMG_{max}

EMG_{Tpk}- Value of EMG prior to T_{pk}

EMG_{TpkN}- Net value of EMG prior to T_{pk}, normalized to EMG_{max}

FNE- Free Nerve Ending

GTO- Golgi Tendon Organ

LFV- Lower Fraser Valley

MVC- Maximum Voluntary Contraction

POS_{Tpk}- Handle Position at Peak Torque

PT- Pronator Teres

r- Correlation Coefficient

RMS- Root Mean Square

RTavep- Average Rate of Change of Torque during Plateau

RTavepN- Average Rate of Change of Torque during Plateau, normalized to T_{in}

RTavey- Average Rate of Change of Torque during Yield

RTaveyN- Average Rate of Change of Torque during Yield, normalized to T_{in}

RTmaxp- Maximum Rate of Change of Torque during Plateau

RTmaxpN- Maximum Rate of Change of Torque during Plateau, normalized to T_{in}

RTmaxy- Maximum Rate of Change of Torque during Yield

RTmaxyN- Maximum Rate of Change of Torque during Yield, normalized to T_{in}

T_{in}- Initial Torque

T_{incr}- Torque Increase

T_{min}- Minimum Torque

T_{miny}- Minimum Torque during Yield

T_{minyN}- Minimum Torque during Yield Phase, normalized to T_{in}

T_{pk}- Peak Torque

$t_{pos90\%}$- Time that Handle Reached 90% of Maximum Displacement

t_{Tpk}- Time of Peak Torque