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Clinical applications of surface mechanomyography

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Abstract

Surface mechanomyography (MMG) has been used in two primary clinical applications: (1) as a control signal for an externally-powered prosthesis, and (2) for examining and/or diagnosing neuromuscular disorders. The results have generally shown that the MMG signal could be used for prosthesis control and has several advantages over the surface electromyographic (EMG) signal, including reduced sensitivity to exact sensor placement and robustness to changes in skin impedance. More research needs to be done, how ever,

to develop techniques that can be used to reduce movement artifacts and improve the classification accuracy with MMG signals. In addition, the results from several studies have shown that the MMG amplitude and/or frequency responses are different for normal, healthy subjects versus those that suffer from a neuromuscular disease, such as myotonic dystrophy and spastic cerebral palsy. Thus, MMG may be useful for diagnosing these disorders, as well as understanding how they affect muscle function. A particularly interesting application of MMG is for examining the effectiveness of anaesthesia. The recent studies that have investigated this issue have shown that the MMG signal is a useful alternative to the surface EMG and force signals. However, future studies are needed in this area to identify the feasibility of using MMG for this application in practical situations.

Introduction

One of the first studies to examine the possibility of using mechanomyography (MMG) in clinical applications was Barry et al. (1986), who investigated the use of MMG as a control signal for an externally powered prosthesis. The authors found that MMG provided several advantages over using surface electromyography (EMG) for prosthetic control, including no need for direct skin contact, the fact that the MMG signal was unaffected by skin impedance, and its amplitude was high enough to produce a 50 mV output from a standard microphone. In addition, the MMG signal required less amplification and electrical shielding, and was qualitatively less sensitive to precise placement over the muscle than was EMG. Furthermore, the disadvantages of MMG, such as movement artifact and rubbing of the sensor on the skin were relatively easy to overcome. Thus, the authors concluded that MMG could be a reliable and inexpensive alternative to EMG for the control of an externally powered prosthesis. This hypothesis was supported by the finding that two subjects that had already been accustomed to EMG-controlled prostheses successfully learned to use an MMG-controlled prosthesis after only three minutes of practice (Barry et al. 1986). L'Estrange et al. (1993) investigated the possibility of using MMG to examine masseter muscle function in humans. Specifically, surface EMG and MMG signals were detected simultaneously from both the right and left masseter muscles during 4-second isometric jaw clenching. The results indicated that the within-day reliability for MMG amplitude was good, with a test-retest correlation of $r = 0.70$ for the right masseter muscle and $r = 0.71$ for the left muscle. It was suggested, however, that strict control of the contact pressure of the sensor over the muscle was important for ensuring reliability. In addition, frequency analysis of the MMG signals showed that they all had a mean power frequency

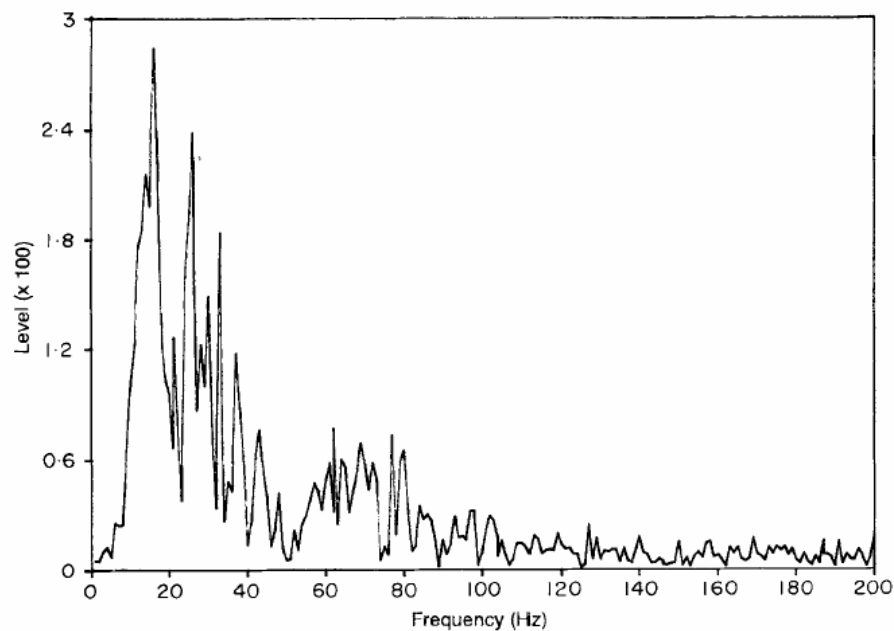


Figure 1. Frequency spectrum of the mechanomyographic (MMG) signal detected from the left masseter muscle of one subject. Notice that most of the power in the MMG signal is below 50 Hz. *Reprinted with permission from L'Estrange et al. (1993).

(MPF) of 9-10 Hz. Visual inspection of the frequency spectrum, however, indicated distinct peaks that were hypothesized to reflect exact harmonics of a fundamental frequency (Figure 1). Furthermore, it was suggested that MMG could potentially be useful for examining cranio-mandibular disorders, since tenderness of the masseter muscles is a common complaint of those that suffer from jaw pain (L'Estrange et al. 1993).

Wright and Stokes (1992) performed a similar study to examine the possibility of using EMG and MMG to investigate the etiology of low back pain. Specifically, the subjects were required to perform a 60-second sustained isometric muscle action of the back extensors in which they supported their body weight, and MMG and EMG signals were detected simultaneously from both the right and left erector spinae muscle groups. The results showed that EMG amplitude increased with time during the fatigue test for both the right and left erector spinae. There were no changes, however, for MMG amplitude during the fatigue test for either the right or left sides. In addition, the MMG amplitude / EMG amplitude ratio decreased during the fatigue test with similar slopes for both the right and left sides, thus indicating reduced efficiency of electrical activity. Finally, the within-day reliability of MMG amplitude was very good, with coefficients of variation ranging from 5.5-8.9%. Thus, it was concluded that for the subjects that participated in the study, the changes in erector spinae EMG and MMG amplitude were symmetrical on both the right

and left sides, thus indicating normal paraspinal muscle function. In addition, since MMG amplitude did not change during the fatigue test, but EMG amplitude increased, MMG may be a better indicator of force production than EMG. Furthermore, when used in combination, the sensitivity of MMG and EMG may be useful in rehabilitative settings where a patient initially demonstrates asymmetry of the right and left paraspinal muscles. As the patient progresses through their rehabilitation program, however, their stages of recovery could be monitored with MMG and EMG to ensure that the program is developing symmetry between the paraspinal muscles of the right and left sides (Wright and Stokes 1992).

Lee et al. (1992) also examined the reliability of MMG amplitude for the erector spinae muscles during quiet standing, prone lying with the back half-extended, fully extended with no resistance, and fully extended against manual resistance. The results showed that the MMG signals recorded when the subject was in the prone position and the back fully extended were more reliable (as reflected by lower coefficients of variation) than those recorded in the other positions. In addition, it is important to point out that some of the subjects that participated in the study were patients that suffered from chronic, post-surgical low back pain. Thus, it was suggested (Lee et al. 1992) that MMG could be useful for monitoring the stages of recovery in patients that suffer from low back pain, since force production by the erector spinae is difficult to measure directly.

Dascalu et al. (1999) performed an interesting study to determine if MMG could be used to monitor the effectiveness of anaesthesia. Specifically, 25 patients that were undergoing either abdominal or orthopaedic surgery were anaesthetized, and three techniques were used to determine the effectiveness of the anaesthesia. The first method involved detecting EMG signals from the adductor pollicis muscle during supramaximal electrical stimulation of the ulnar nerve. Surface MMG signals were also detected simultaneously, as was the force signal from the muscle. The results indicated that the amplitude of the MMG signal was highly correlated with the force signal ($r=0.86$) and the EMG signal ($r = 0.85$). Thus, it was suggested that MMG may be an effective alternative to EMG and force signals for monitoring the effectiveness of neuromuscular block. It was also recommended, however, that future investigations still need to be performed before MMG can be widely accepted as a tool for monitoring anaesthesia (Dascalu et al. 1999). Lee et al. (1996) also examined the possibility of using MMG for determining the effectiveness of anaesthesia. Specifically, seven male pigs were subjected to Mg^{2+} -induced neuromuscular block, and the EMG and MMG responses of the tibialis anterior muscle were investigated during electrical stimulation. The results showed that MMG was generally more sensitive than EMG for testing the effectiveness of

neuromuscular blockade. It was also suggested, however, that more investigations still needed to be performed before MMG could be used to gauge the effectiveness of anaesthesia in humans (Lee et al. 1996).

Orizio et al. (1997) were among the first to examine the possibility of using MMG to diagnose neuromuscular diseases. Specifically, the authors recorded MMG and EMG signals from the biceps brachii and flexor digitorum profundus during isometric muscle actions at 20%, 40%, and 60% of the maximum voluntary contraction (MVC) in ten subjects that suffered from myotonic dystrophy, as well as ten age-matched controls. The results indicated that the isometric strength values for the myotonic dystrophy patients were about 30% less than those of the controls for the forearm flexors, and approximately 74% less for the finger flexors. In addition, at any given force level, the mean MMG and EMG amplitude values for the myotonic dystrophy patients were significantly lower than those for the control subjects. Finally, the ratio of MMG amplitude / EMG amplitude (which was used as a measure of electromechanical efficiency) was similar for the myotonic dystrophy patients and control subjects for the forearm flexors, but much lower for the myotonic dystrophy patients for the finger flexors. Thus, it was suggested that the MMG amplitude / EMG amplitude ratio may be useful for estimating the efficiency of the mechanical contributions of the active motor units. In addition, the combined use of MMG and EMG may be able to differentiate between muscles that are more affected by muscular dystrophy (i.e., the finger flexors) and those that are less affected (Orizio et al. 1997).

The potential for MMG to be used in examining muscle atrophy is also an interesting clinical application. Pisöt et al. (2008) investigated the changes that occur in MMG amplitude and muscle contractile parameters during thirty-five days of bed rest in ten healthy men. Specifically, MMG amplitude for the biceps brachii, vastus medialis, biceps femoris, and gastrocnemius medialis were assessed during electrically stimulated isometric twitches both before and after the thirty-five days of bed rest. Changes in muscle thickness were also assessed by ultrasound imaging. The results showed that bed rest resulted in a significant increase (18%) in contraction time for the gastrocnemius muscles, and decreased muscle belly thickness (15%). In addition, the maximal displacement of the muscle (which is analogous to MMG amplitude) increased after bed rest for the vastus medialis (24%) biceps femoris (26%) and gastrocnemius medialis (30%). Thus, it was suggested (Pisöt et al. 2008) that MMG may be sensitive to changes in the viscoelastic properties of a muscle that is undergoing the process of atrophy. This, in turn, could have application to evaluating the decreases in muscle function that accompany space flight.

McKay et al. (2004) examined the MMG and EMG amplitude and MPF responses for the rectus femoris muscle immediately after aerobic exercise.

Specifically, all subjects were required to cycle on a stationary cycle ergometer at a power output that corresponded to 70% of the power output at VO_2 max for a duration of 30 minutes. Immediately before and after the exercise protocol, MMG and EMG signals were detected from the rectus femoris in the resting state. The results showed that MMG amplitude was elevated immediately after exercise (i.e., in relation to the pre-exercise value), but decayed over time with a time constant that was equivalent to that for VO_2 . In addition, there were no significant differences between the pre-exercise and immediate post-exercise values for EMG amplitude, EMG MPF, and MMG MPF. Thus, it was concluded that the elevated MMG activity after exercise reflected increased mechanical work that also caused greater oxygen consumption. In addition, it was suggested that MMG may be a more sensitive indicator of resting muscle activity than EMG, since EMG amplitude was not elevated after exercise. It was also recommended, however, that additional studies need to be performed to determine if post-exercise muscle sounds are related to exercise intensity, and whether these sounds can be affected by obesity or muscle disease (McKay et al. 2004). McKay et al. (2006) have also conducted some interesting studies that examine the MMG responses from resting muscle immediately after resistance exercise. Specifically, the subjects were required to perform maximal isokinetic muscle actions of the right leg extensors at a velocity of $60^\circ \cdot \text{s}^{-1}$. These muscle actions were performed as separate sets of 1, 5, 10, 20, and 30 repetitions, and the resting MMG activity of the rectus femoris muscles of both the right and left legs were assessed with separate accelerometers. In addition, the resting MMG activity was measured at both full leg extension (i.e., a short muscle length) and full leg flexion (i.e., a long muscle length). The results indicated that MMG amplitude for the right leg was elevated after exercise, but only after the set that required 30 repetitions to be performed. In addition, there was a significant positive linear relationship between the total work performed and resting MMG amplitude after exercise for both the right ($r = 0.61$) and left ($r = 0.67$) legs. Furthermore, when the rectus femoris was at a long muscle length (i.e., when the leg was fully flexed), there was no relationship between MMG amplitude and total work, but the resting MMG amplitude before exercise was greater at a short muscle length than at a long length. Thus, it was concluded that resting MMG activity is likely neural in origin, since it was present in a contralateral muscle that had done no mechanical work. In addition, the greater resting MMG amplitude at the short muscle length than the long length was hypothesized to be due to a more compliant musculotendinous unit that allowed greater muscle fiber oscillations and larger MMG amplitude values.

Thus, these findings (McKay et al. 2006) were consistent with those from the previous study of aerobic exercise (McKay et al. 2004) and indicated that

resting MMG activity is elevated immediately after isokinetic exercise. McKay et al. (2007) also measured resting MMG after conventional resistance exercise. Specifically, the subjects were required to perform one set of 10 repetitions with 50% of their one-repetition maximum (1-RM) as a warm-up, followed by five sets of 8 repetitions with 75% of their 1-RM on both the bilateral leg extension and leg press exercises. This workout took approximately 30 minutes to complete, after which resting MMG and EMG signals were recorded from the rectus femoris for 5.75 hours. The results showed that MMG amplitude increased from $3.0 \pm 0.99 \text{ mm}\cdot\text{s}^{-2}$ pre-exercise to $10.1 \pm 4.5 \text{ mm}\cdot\text{s}^{-2}$ post-exercise, which corresponded to an increase that ranged from 1.8 to 7.7 times for all subjects. In addition, the mean MMG amplitude value after exercise decayed over time with a time constant that was statistically equivalent to that for VO_2 . Finally, EMG amplitude was significantly greater after exercise than before exercise, but only four data points exceeded the lower limit of resolution for the EMG amplifier. Thus, it was concluded that resting MMG amplitude increased about threefold after strenuous resistance exercise and then decayed exponentially over time in a manner that was similar to that for VO_2 . The authors (McKay et al. 2007) also proposed an interesting hypothesis to explain the elevated MMG amplitude after exercise, with no change in EMG amplitude. Specifically, it was suggested that most commercially available EMG systems are simply not sensitive enough to detect resting muscle activity, but since muscle tissue is an excellent conductor of sound, MMG may be a more useful method than EMG for quantifying resting muscle activity. It was also recommended, however, that more research needs to be done to identify the mechanisms underlying resting muscle sound (McKay et al. 2007).

Hu et al. (2007) simultaneously recorded surface MMG and EMG signals from the biceps brachii during submaximal isometric muscle actions of the forearm flexors at 20%, 40%, 60%, and 80% MVC in normal healthy subjects and patients that had suffered a stroke. For the stroke patients, both the affected and unaffected limbs were tested. The results showed that the mean MMG and EMG amplitude and MPF values for the limb that was affected by the stroke were less than those for the unaffected limb, as well as those from the limbs of the healthy controls. Thus, it was suggested that a loss of fast-twitch motor units in the muscles of the affected limb and/or reduced neural drive to those muscles could have caused the MMG and EMG amplitude and MPF responses. In addition, MMG could potentially be used as a complement to EMG for examining the loss of muscle function that occurs following a stroke, as well as the return to normal function during rehabilitation (Hu et al. 2007).

McAndrew et al. (2005) examined the potential for MMG to be used for several applications in musculoskeletal rehabilitation. Specifically, the authors used a laser-based MMG system that measured muscle displacement during voluntary or electrically-stimulated contractions. The use of the laser-based system allowed for calculation of contraction time, normalized contraction time, sustained time, and relaxation time, which are not usually assessed when MMG is detected with an accelerometer, condenser microphone, or piezoelectric device. One of the first tests that the authors conducted examined the effect of changes in pulse duration from the electrical stimulator on the twitch properties of the muscle and MMG responses. The results showed that a pulse duration of 100 μ s was optimal for producing a maximal muscle contraction, as well as for optimizing the lateral displacement of the muscle and the contraction and relaxation speeds. The second part of the study involved a fatiguing stimulation protocol of isolated rat muscle (40-second duration at 2 Hz) that was designed to simulate the fatigue associated with chronic low back pain. Both before and after the fatiguing protocol, the twitch parameters and MMG responses of the muscle were tested. The results indicated that fatigue caused a decrease in peak displacement of the muscle and impairment of contraction time and half-relaxation time. Thus, the authors (McAndrew et al. 2005) concluded that MMG could be a useful method for examining chronic low back pain.

Nolan and de Paor (2004) investigated the possibility of using MMG to help disabled individuals communicate. For example, individuals that have suffered a stroke often find it difficult to communicate due to impairment of the muscles used for speech. Thus, the authors (Nolan and de Paor 2004) developed an accelerometer-based system that detected the MMG signals from the biceps brachii and sternocleidomastoid muscles. These signals were then used to control a software program that scanned through letters on a keyboard. When the software program reached the letter that the patient wanted to select, they contracted the biceps brachii or sternocleidomastoid muscles, and the resulting MMG signals from the muscles were used to control the software and select the letter. Although the authors reported that facial movements sometimes caused unintentional triggering in the software, they also found that their system was fairly reliable and useful for people that needed an alternative method for communicating. They also suggested, however, that additional work needed to be done before the system could be used on a widespread basis (Nolan and de Paor 2004). Barry et al. (1990) also used MMG to examine various neuromuscular diseases. Specifically, the authors recorded surface MMG signals from the biceps brachii in both normal children (age range = 7-16 years) and kids that suffered from some form of neuromuscular disease (e.g., muscular dystrophy, myotonic dystrophy, dermatomyositis, etc.). The

results showed that the electromechanical efficiency (i.e., EMG amplitude / MMG amplitude ratio) of the diseased patients was less than that of the healthy subjects, and it was hypothesized that this was due to atrophied fibers in diseased muscle that generated electrical activity, but provided little mechanical contribution to force production. Thus, it was concluded that MMG allows mechanical data to be obtained from the muscle when force measurements are unavailable, such as when examining the paraspinal muscles, which are often one of the first muscle groups affected by myopathies (Barry et al. 1990). Akataki et al. (1996) examined the EMG and MMG responses from subjects that suffered from spastic cerebral palsy. Specifically, surface EMG and MMG signals were detected simultaneously from the biceps brachii during submaximal isometric muscle actions of the forearm flexors at force levels ranging from 10-50% MVC. The results indicated that for all force levels, the mean MMG amplitude values for the normal subjects were significantly greater than those for the cerebral palsy patients. In addition, both the MMG amplitude / muscle cross sectional area and MMG amplitude / EMG amplitude ratios were greater for the normal subjects than for the cerebral palsy patients at all force levels. The mean isometric forearm flexion strength value for the cerebral palsy patients was also roughly 50% that of the normal subjects. Thus, it was concluded that in addition to the EMG amplitude / force ratio, the MMG amplitude / EMG amplitude ratio may be a useful indicator of electromechanical efficiency. In addition, it is likely that the altered MMG amplitude / EMG amplitude ratio in the cerebral palsy patients was due to the deterioration of muscle contractile properties that accompanies muscle atrophy (Akataki et al. 1996).

Madeleine et al. (2007) investigated the possibility of using MMG to determine the etiology of chronic low back pain. Specifically, 12 separate MMG sensors were placed over the right and left erector spinae muscles (6 sensors on each side of the vertebral column). The subjects were then required to perform submaximal isometric muscle actions of the back extensors with different external loads, ranging from 0-15 kg in 2.5 kg increments. The subjects also performed a 6-minute sustained isometric muscle action of the back extensors with an external load of 7.5 kg. The results showed that MMG amplitude generally increased with the external load that was being supported, while MMG MPF decreased. Similar results were also reported for the MMG amplitude and MPF responses during the fatiguing muscle action, since MMG amplitude increased, and MMG MPF decreased over time. Perhaps the most important finding from the study, however, was that when the MMG amplitude and MPF values were expressed in absolute terms (i.e., $m \cdot s^{-2}$ and Hz, respectively), the patterns of responses were different for each sensor location. The patterns for normalized MMG amplitude and MPF were the same,

however, for all sensor locations. Thus, it was concluded that when measuring the mechanical activities of the paraspinal muscles with MMG, it is important to detect signals from multiple locations to account for differences that can be caused by sensor placement (Madeleine et al. 2007).

Madeleine and Arendt-Nielsen (2005) examined the effects of experimentally-induced muscle pain on MMG signals from the biceps brachii during submaximal isometric muscle actions of the forearm flexors. Specifically, the experimentally-induced pain was administered by injecting a hypertonic saline solution into the right biceps brachii muscle, while injection of an isotonic saline solution was used as a control. Following injection of either the hypertonic saline or isotonic saline, the subjects were required to perform submaximal isometric muscle actions at 0%, 10%, 30%, 50%, and 70% MVC, as well as a 25-second isometric ramp muscle action from 0-50% MVC. The results showed that MMG amplitude increased significantly after the hypertonic saline injection during both the constant force and isometric ramp muscle actions. There were no changes, however, for the EMG variables. Thus, it was concluded that the hypertonic saline injection not only caused increases in muscle pain, but it may also have changed the muscle's contractile properties such that compensatory mechanisms (e.g., decreased firing rate and increased twitch force) were used to meet the force demands of the task. In addition, it was suggested that under well-controlled conditions, MMG may be more sensitive than EMG for detecting changes in the mechanical properties of the muscle due to experimentally-induced pain (Madeleine and Arendt-Nielsen 2005).

Jaskólska et al. (2006) examined the EMG and MMG responses for the triceps brachii, biceps brachii, and brachioradialis during maximal isometric muscle actions of the forearm flexors and extensors at different elbow joint angles (i.e., the optimal joint angle for force production, as well as 30° greater and 30° less than the optimal joint angle) in both young (mean \pm SD age = 20.1 \pm 1.1 years) and old (mean \pm SD age = 64.9 \pm 5.1 years) women. The results showed that the ratio between MMG amplitude and force was lower in the old women compared to the young women, but only for the triceps brachii. In addition, the MMG amplitude versus force ratio increased for both the biceps brachii and triceps brachii as the muscle was shortened for both the young and old women. Thus, it was concluded that the EMG and MMG responses to changes in muscle length were generally similar in the old and young women, but the optimal angle with respect to force production was greater in the older women.

Ng et al. (2006) conducted an interesting study to examine differences in force-related post-activation potentiation and MMG-related post-activation potentiation between normal, healthy subjects and those that suffered from

some type of myopathy. Force-related post-activation potentiation refers to the increase in electrically-stimulated force production that occurs after an isometric MVC, whereas MMG-related post-activation potentiation reflects the increase in MMG amplitude immediately after an isometric MVC. All subjects were required to undergo five single supramaximal electrically-stimulated twitches of the biceps brachii both before and immediately after a 10-second isometric MVC. The results showed that the MMG-related post-activation potentiation was positively correlated with the force-related post-activation potentiation for the normal subjects. In addition, the MMG-related post-activation potentiation was significantly lower in the subjects that suffered from a myopathy than it was in the normal or diseased control subjects (Figure 2). Furthermore, there was no relationship between the MMG-related post-activation potentiation and Type 2 muscle fiber atrophy.

Thus, it was concluded that although MMG may have some potential for differentiating between normal and non-dystrophic myopathies, more work still needed to be done before it could be used for diagnostic purposes (Ng et al. 2006). Orizio et al. (1997) investigated the MMG responses for the tibialis anterior in patients that suffered from myotonic dystrophy during electrically-stimulated twitches, as well as repetitive stimulation at 5, 10, 15, or 20 Hz. The results showed that during the single twitches, MMG amplitude was 67% lower, MMG MPF was 44% lower, the twitch duration was 37% longer, and the electromechanical delay was 64% longer in the myotonic dystrophy patients than in the controls. In addition, the peak-to-peak amplitude of the MMG signal was less at each stimulation frequency for the myotonic dystrophy patients than for the controls. Thus, it was concluded that in addition to differences in sarcolemmal excitability, myotonic dystrophy is characterized by altered electromechanical coupling and a failure in the contractile machinery (Orizio et al. 1997).

Alonso et al. (2007) examined the MMG and EMG activities of the genioglossus, sternocleidomastoid, and diaphragm muscles during a high respiratory effort contraction in patients with obstructive sleep apnea syndrome as well as normal, healthy subjects. The purpose of the investigation was to determine if the combined use of MMG and EMG could be used to examine the characteristics of obstructive sleep apnea syndrome. The results showed that at progressively higher levels of respiratory effort, there was an increase in the nonlinear coupling of the respiratory muscles for both the patients and control subjects. That is, the genioglossus, sternocleidomastoid, and diaphragm muscles were more coordinated with their contractions. Thus, it was concluded that the combined use of MMG and EMG may be useful for examining the activities of respiratory muscles during labored breathing, as well as for investigating obstructive sleep apnea syndrome (Alonso et al. 2007). Madeleine

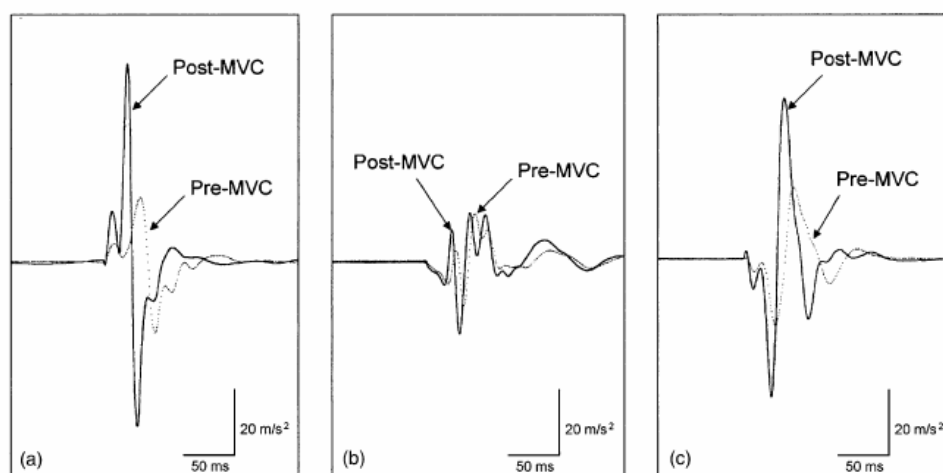


Figure 2. Mechanomyographic (MMG) signals detected from the biceps brachii during a maximal electrically-stimulated isometric twitch immediately before (Pre-MVC) and after (Post-MVC) a 10-second isometric maximum voluntary contraction (MVC). (A) shows the data from a healthy subject, (B) demonstrates the signals from a subject with polymyositis, and (C) shows the data from a subject that suffers from Kennedy-Alter-Sung disease. Notice that in the healthy subject, the amplitude of the MMG signal for the Post-MVC twitch was potentiated, but this was not as apparent for the neuromuscular disease patients. *Reprinted with permission from Ng et al. (2006)

and Farina (2008) also performed an interesting study that examined the time and frequency domain characteristics of surface MMG signals detected from the upper trapezius muscle. Specifically, 12 accelerometers were placed over the dominant upper trapezius muscle, and the subjects were required to perform 10-second isometric shoulder elevation muscle actions at 10%, 20%, 40%, 60%, 80%, and 100% MVC. In addition, the subjects performed a sustained isometric muscle action at 20% MVC until the target force level could no longer be maintained. The results showed that the absolute MMG amplitude and MPF values were dependent on sensor location, but when the values were normalized relative to a standard value, there was no effect of sensor location. In addition, MMG amplitude at the end of the sustained muscle action at 20% MVC was expressed relative to the corresponding value at 100% MVC to calculate the activation ratio. The subjects that showed a greater increase in MMG amplitude over time, higher values for the activation ratio, and lower entropy values were associated with a longer time to task failure. The finding of lower entropy values is reflective of more homogeneous motor unit recruitment. Thus, it was concluded that there was a relationship between the time to task failure, the activation ratio, and MMG amplitude, which suggested that the spatial changes in MMG amplitude during a fatiguing muscle action reflected functional mechanisms that allowed for maintenance of

force production during fatiguing isometric muscle actions (Madeleine and Farina 2008). Huang et al. (2006) have also used MMG to investigate the mechanical and neural aspects of spastic hypertonia. Specifically, the subjects included patients that suffered from either a spinal cord lesion or a stroke, as well as normal subjects. Both the H-reflex and the maximum amplitude of the M-wave, in addition to the MMG amplitude and median frequency responses during the M-wave stimulation were assessed. The results showed that the patients that suffered from spastic hypertonia exhibited greater MMG amplitude values during the M-wave stimulation than the healthy control subjects. In addition, the amplitude of the MMG signal during the M-wave stimulation was correlated with the level of functional impairment displayed by the subjects. Thus, it was concluded that MMG may be a useful tool for assessing the impairment in the mechanical properties of the muscle that occurs with spastic hypertonia (Huang et al. 2006).

Overall, the results from the studies discussed in this chapter indicated that there are many potential clinical applications for MMG, particularly in the areas of neuromuscular disease assessment and control of externally-powered prostheses. There is still a great deal that needs to be done, however, before MMG can be used on a widespread basis for diagnostic purposes. In addition, research that examines MMG activity of resting muscle after exercise and to investigate the effectiveness of anaesthesia are particularly promising clinical applications. Future research should also continue to examine the MMG responses of the lower back muscles to determine if MMG can be used to investigate the etiology of chronic low back pain and asymmetry of the paraspinal muscles.

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