

SECTION TWO

Chapter One

The Contribution of Dynamic Electromyography to Gait Analysis

by Jacquelin Perry, M.D.

Dr. Perry is Director of the Pathokinesiology Lab at the Rancho Los Amigos Medical Center in Downey, California.

INTRODUCTION

The purpose of dynamic electromyography is to accurately define the muscle action that controls joint motion. While gross function of muscle groups can be inferred from motion and moment calculations, specificity of muscle function requires a more discriminating technique.

The Functional Challenge

Walking relies on selective timing and intensity of appropriate muscles at each joint to provide weight-bearing stability, shock absorption, and progression over the supporting foot during stance and to advance the limb in swing. Energy is conserved by activating only the muscles optimally aligned for each task and by substituting momentum and passive tissue tension for direct muscle activity wherever possible.

Throughout this sequence of functions, the muscles perform in groups, as shown in **Figure 1** (1). While the dominant motions of the lower limb occur in the sagittal plane (i.e., flexion and extension for the demands of progression), there also are significant actions in the other two planes (coronal and transverse) to enhance single limb balance and body rotations. Each muscle has a unique three-dimensional (3D) effect determined by its alignment across the joint or joints it crosses. In addition, most muscles are members of two or more

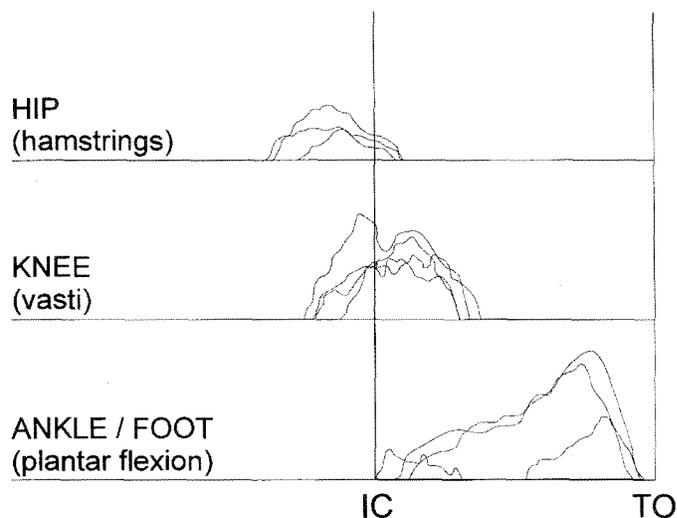


Figure 1.

Normal sequence of synergistic activity of the major extensor muscle groups during stance. Linear display of the EMG amplitudes (vertical scale) of the individual muscles identify their relative intensity and timing. Hamstrings (biceps femoris, semimembranosus, semitendinosus); Vasti (intermedius, lateralis, medialis longus, medialis oblique); Plantar flexors (soleus, gastrocnemius, tibialis posterior [biphasic]). IC (initial contact) indicates onset of stance. Note extensor muscles begin in late swing.

functional groups. This redundancy assures 3D balance and serves to simplify the integration of adjacent joint action. Relative intensity of action of a particular

muscle is determined by which of its functions is momentarily dominant. Hence, just understanding normal function requires a detailed study of individual muscle action. Such information also can identify the effects of orthoses, muscle training regimens, etc. Dynamic electromyography offers the means of precisely relating muscle action to the specific function.

The Influence of Pathology

The normal, complex walking pattern can be disrupted in many ways. Muscles may be weakened by disuse, pain, or direct injury. Fibrous tissue contracture may limit passive mobility. Orthoses incidentally restrict adjacent motion while purposefully protecting the area of concern. Brain and spinal cord injury may disrupt the primary motor control and feedback pathways. Persons with spastic paralysis, stroke, or head trauma, present the greatest diagnostic challenge as muscle function is disrupted at many levels and the overlay of spasticity often causes the clinical tests to differ significantly from the muscle pattern used during walking. Even lower motor neuron lesions can present unpredictable situations. Individuals preserve their ability to walk by substituting, to the extent their selective control allows. Alternate motions and muscle actions are used to overcome the limitations imposed by pathology. Such substitution capability varies markedly among individuals. Consequently, the person's walking pattern is a mixture of primary functional loss and substitutive actions. The results are mixtures of inadequate, excessive, inappropriately timed, or out-of-phase muscle action. To best design retraining protocols, optimize orthotic assistance, or to plan an appropriate reconstructive surgical procedure, it is essential to know muscle function as it is occurring rather than assumed. This requires dynamic electromyography.

METHODS

Myoelectric Signal Anatomy

Electromyography (EMG) is a system that records the electrical signals activating the muscle fibers. From such information, one can determine the timing and relative intensity during both normal and abnormal function. Under specific circumstances, muscle force also can be calculated.

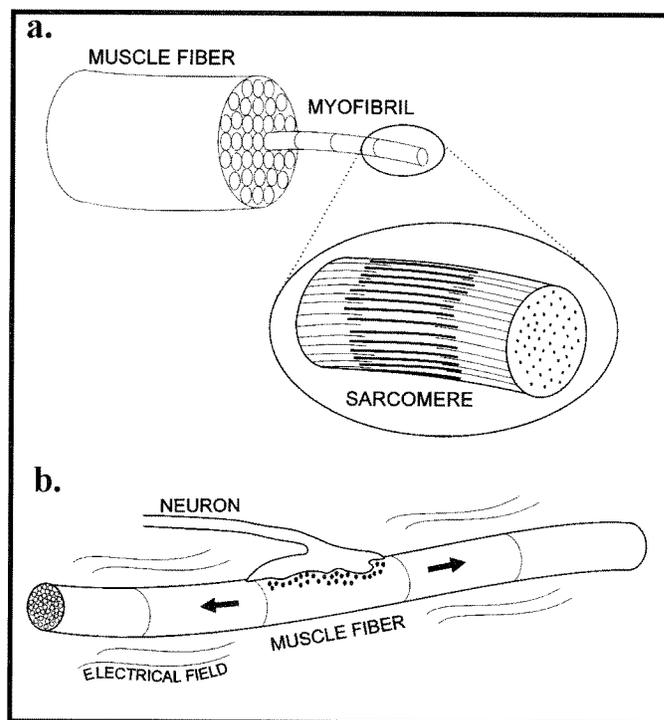


Figure 2.

a) Muscle Fiber Structure: Each muscle fiber is a bundle of myofibrils (chains of contractile units called sarcomeres). Interplay of the thin and thick filament within the sarcomere creates the muscle force. b) Muscle Fiber Myoelectric Signal: An electrical field is created by stimulation from the neuron activating the muscle fiber's chemical receptors (shaded circles), which in turn, send an electrical charge up and down each myofibril to activate the chain of sarcomeres. Adapted from reference (2). Used with permission.

Each muscle fiber consists of multiple long chains (myofibrils) of contractile units (2) called sarcomeres, which create the force of muscle action (**Figure 2a**). As the local neuron chemically activates the muscle fiber at its myoneural junction, an electrical charge is sent up and down each myofibril (**Figure 2b**), stimulating the sarcomeres to contract (3). This event creates an electromagnetic field, which can be used to track muscle activity (4). By volume conduction, the local signal spreads through the tissues making it technically possible to record the signal at the skin surface as well as internally.

Neural control is simplified by having large groups of muscle fibers controlled by a single motor cell body located in the anterior horn of the spinal cord. This

composite of cell body, connecting neuron, and the muscle fiber cluster is called a motor unit. The gastrocnemius, for example, is composed of approximately one million muscle fibers clustered in 600 motor units (5). Animal experimentation has shown that the muscle fibers of each motor unit are widely dispersed throughout the muscle. Only a few units are needed to create a weak effort throughout the whole muscle. In the multipennate soleus, for example, one motor unit is spread across 60 percent of the muscle's volume, as shown in **Figure 3** (6). Theoretically, just two motor units would be sufficient to traverse the whole muscle. In contrast, a motor unit in the unipennate tibialis anterior covers only 16 percent of the volume (7). Now 6 motor units would be needed. The practical interpretation of this anatomical fact is that during walking and other physiological functions, muscle action can be recorded regardless of the location of the electrode over or within the muscle.

Interspersion of tendonous tissues, however, reduces the concentration of muscle fibers; thus, the middle of the muscle belly is the site where the largest signals are obtained. To be even more precise, maximum signal occurs at the muscle's motor point (8). Using the gastrocnemius as an example, 6 motor units would represent only 0.1 percent muscular effort, while a clinical strength grade of 2 (poor), which represents a muscle too weak to accept even the resistance of gravity, averages 5 percent. Theoretically, this represents 30 motor units, a minimum contraction situation. As more motor units are activated, the intensity of the muscular response increases and the EMG signal becomes larger. Clinically, this is reflected as a greater functional force.

Myoelectric Signal Qualities

The signal recorded during functional EMG is described as random because it does not have a consistent waveform. Instead, the individual spikes vary in amplitude and duration without an identifiable sequence. This inconsistency reflects the fact that every muscular effort is a composite of multiple motor units, each activating multiple muscle fibers. In addition, each fiber's response to stimulation is a brief twitch and, thus, repeated stimulation is required to generate a useful force. Hence, the EMG signal of muscle action is

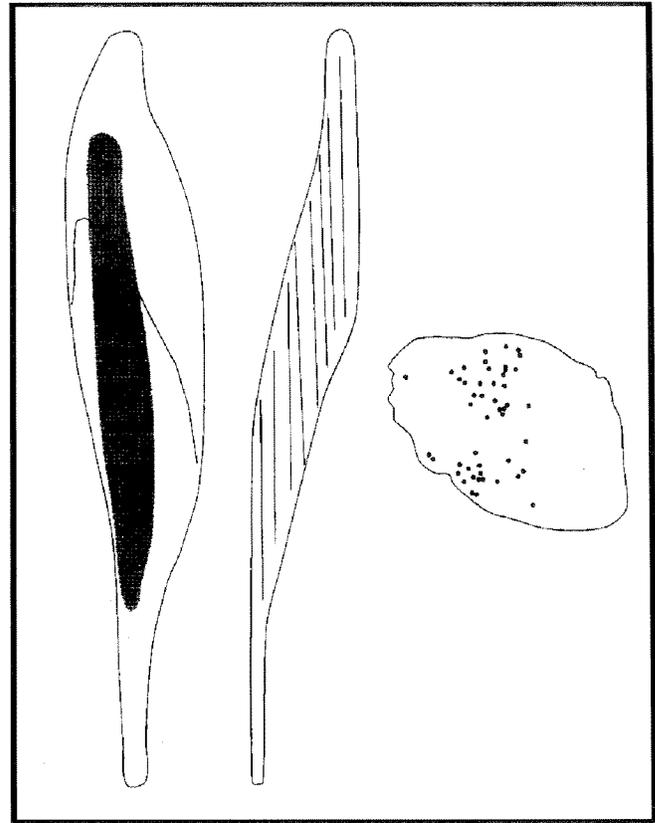


Figure 3.

Motor Unit Territory: The vertical shaded areas in the anterior and lateral projections show the distribution of the muscle fibers of one motor unit within the rat soleus. The cross section identifies the individual muscle fiber distribution (dots) for that motor unit. Adapted from reference (6). Used with permission.

a train of randomly shaped action potentials. In addition, the raw recorded electronic signal is contaminated by noise (i.e., unwanted signals arising from tissue motion and the environment, such as lights, neighboring motors, and so forth). The unwanted electronic noise is excluded by filtering and the use of differential amplifiers, which reject common mode signals.

Waveforms are classified by their content of different sine wave frequencies—Fourier analysis (4). In simplistic terms, sharply peaked waves have a high frequency while broad waves have a low frequency. The complex nature of myoelectric signals includes a very

broad spectrum of frequencies, with the range from 10 Hz to 1,000 Hz being considered significant to identify muscle function related to joint motion (Figures 4a and 4b). Tissue displacement accompanying a muscle contraction can generate 10-Hz signals and floor impact during walking gives rise to signals of 25–30 Hz. Hence, 40 Hz has become a customary lower value for gait EMG. In addition, a notch filter is used to exclude the common 60-Hz signals from electrical equipment. Signals above 1,000 Hz do exist but they represent less than 1 percent of the signal power and add nothing to our knowledge of muscle function, so instrumentation with this capability is unnecessary. Hence, a bandwidth of 40–1,000 is appropriate.

Muscle Specificity: Surface versus Wire Electrodes

For functional EMG, the sensing electrode may be either surface contacts (Figure 5a) or penetrating wires (Figure 5b). The criteria for selecting an appropriate electrode include the purpose of the EMG recording,

muscle anatomy, signal dispersion through the tissues, and tolerance of skin penetration with a fine needle.

Surface Electrodes

These EMG sensors have the advantage of convenience and comfort. An active electrode system merely needs to be taped over the center of the target muscle. Passive disc electrodes require a gel and skin cleansing to improve signal transmission. Of the 28 major muscles controlling each lower limb that can be delineated by EMG, the majority are superficial. The dominant period of activity of these subcutaneous muscles can be readily identified by surface electrodes.

The major disadvantages to surface electrodes are cross talk and low signal reception. Their adverse effects complicate the definition of muscle timing and the relative intensity of the activity.

Cross Talk

During periods of low muscle activity, there is the possibility that the EMG record may include signals from musculature other than the muscle of interest. Surface electrodes sense all the signals that reach its reception area. Volume conduction allows wide dispersion of the myoelectric signals through the tissues (10). The thin films of fascia between adjacent muscles present no significant barrier to the myoelectric signals from nearby muscles. Also, muscles function in groups rather than in isolation. As a result, the recording from a surface electrode, by picking up the signals of a synergist may indicate activity in the designated muscle when actually it is quiet.

Several investigators have documented the presence of cross talk by comparing the output of wire and surface electrodes. Perry et al. (11) confirmed group muscle action by demonstrating simultaneous activity of the soleus, gastrocnemius, and tibialis posterior during traditional manual strength tests purported to isolate the targeted muscle. Peak muscular effort, however, corresponded to the designated muscle. The finding that the surface electrodes included EMG from the adjacent muscles implied greater activity than was confirmed by the wire data. De Luca and Merletti (12) studied the signal spread that accompanied electrical stimulation of the tibialis anterior. They found signals in the peroneus brevis and soleus that approximated 17 percent of the maximum tibialis anterior EMG. Koh and Grabner (13), using both stimulation and voluntary quadriceps activa-

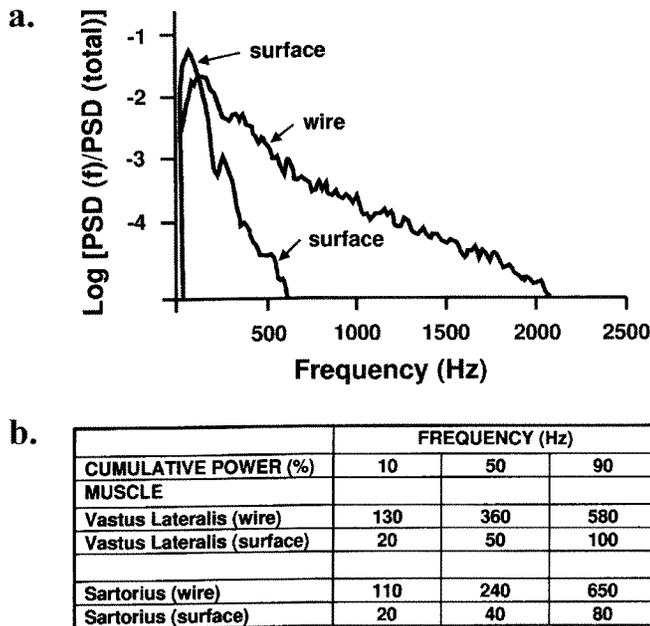
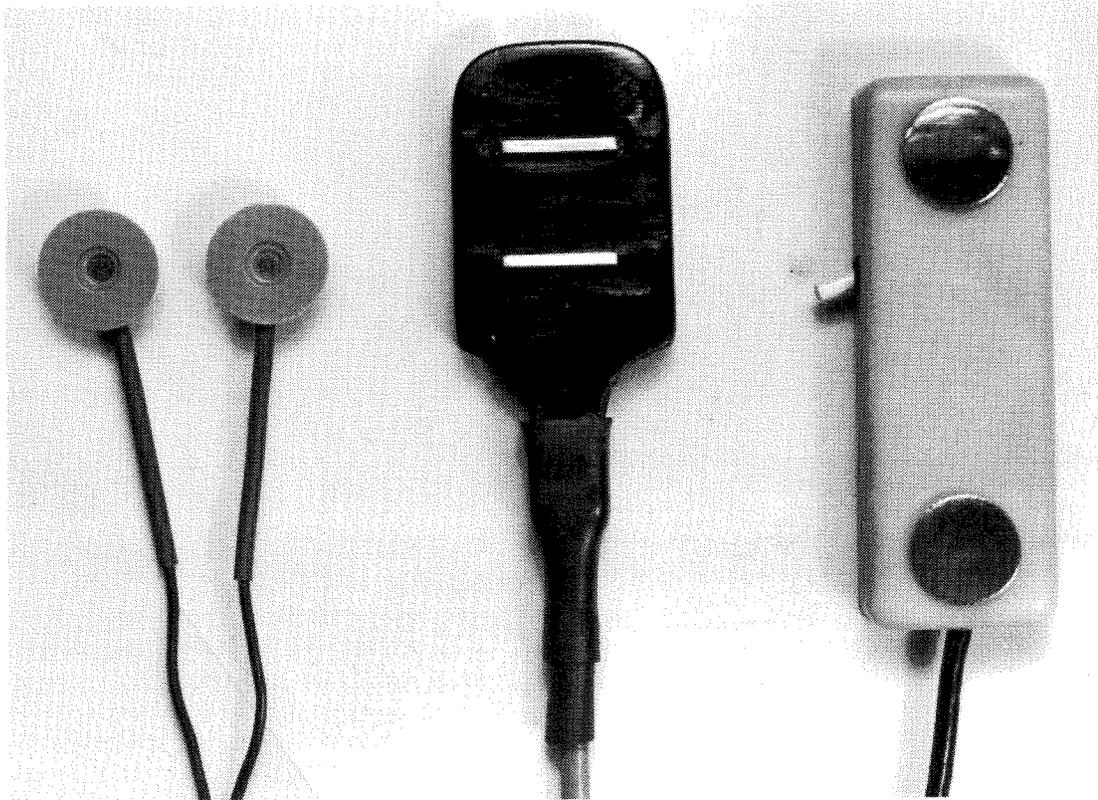
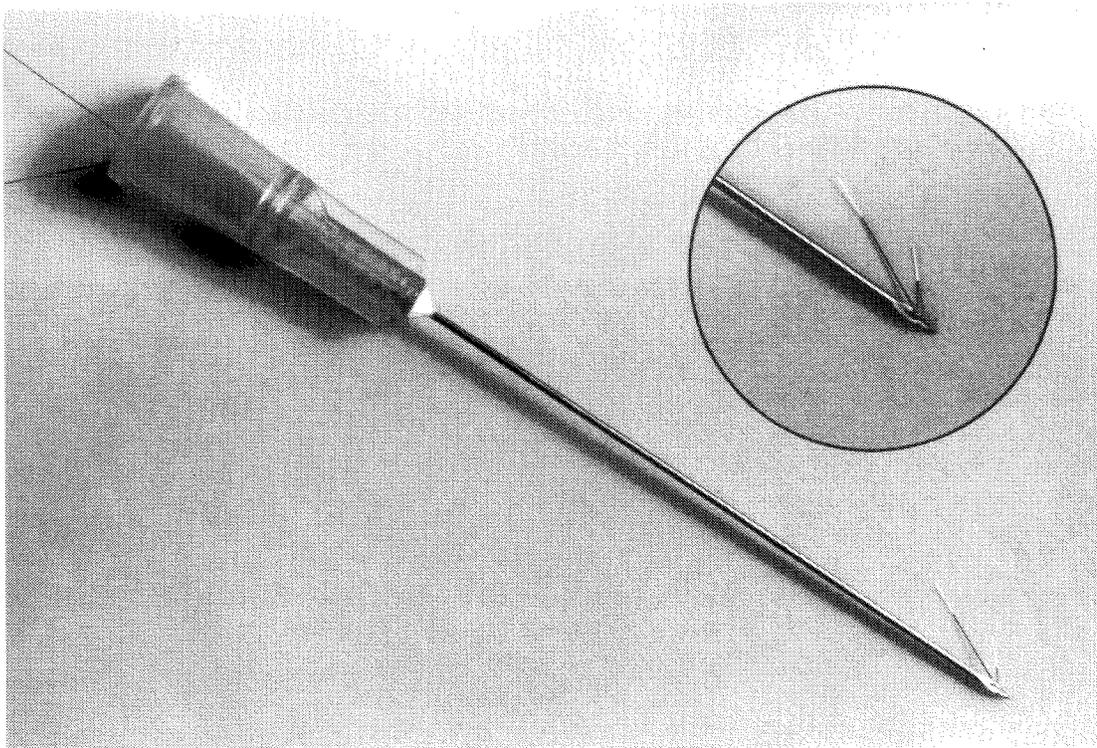


Figure 4. The typical normalized power spectral density (PSD) for wire and surface electrodes: a) Percent total signal power per frequency interval. Determined by direct Fourier transform of data digitized at 5000 samples/sec, total spectrum 5-2500 Hz with a 5 Hz resolution; b) Thresholds of power spectrum distribution. Frequency below which 10, 50, 90% of the power spectrum occurs. From reference (9). Used with permission.

**Figure 5a.**

Electrodes for Dynamic EMG. Surface: (left) A passive electrode pair containing 2-mm diameter silver silver-chloride disc centers. (Center and right) Examples of active electrodes with signal preamplification circuitry imbedded in the electrode housing. The elements of the center electrodes are 1-cm by 0.1-cm bars spaced 1 cm apart. The right electrode elements are 1-cm discs with an interelectrode spacing of 3.5 cm.

**Figure 5b.**

Intramuscular wire electrodes are a pair of 50-micron, nylon insulated nickel-chromium alloy wires¹ with the distal 2-mm bare tips, placed in a 3.81 cm 25- or 30-gauge needle for intramuscular insertion. **Inset:** Note, to allow single needle insertion, the external barbs must differ in length to avoid contact between the bared tips.

¹California Fine-Wire Company, Grover Beach, CA 93433.

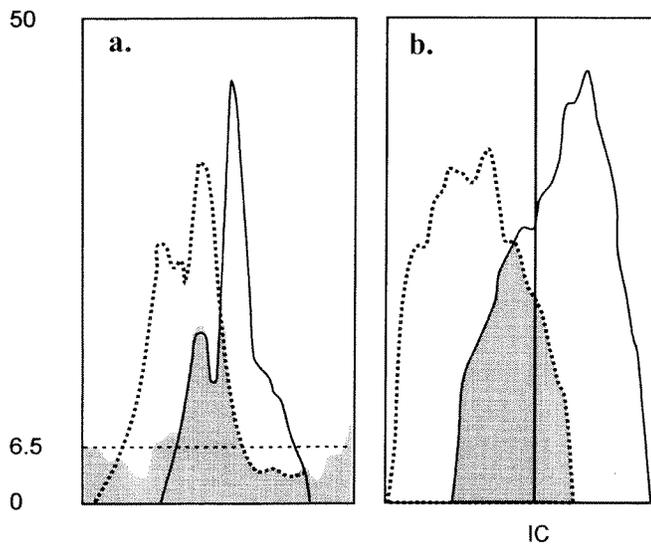


Figure 6.

Cross talk: a) Surface electrode recording of antagonistic flexor (.....) and extensor (—) muscles during walking to identify cocontraction. Shaded area identifies occurrence of simultaneous EMG by both electrodes. Adapted from reference (14). b) Wire electrode recording of similar hamstrings (.....) and quadriceps (—) action. Note the continuous baseline of EMG in the surface recording that is not present in the wire electrode data. This is a display of signal cross talk from adjacent muscles. The taller shaded areas in both recordings represent true cocontraction of antagonist muscles. Adapted from reference (1). Used with permission.

tion to study cross talk, found EMG signals in the medial and lateral hamstrings averaging 11 percent and 17 percent of a maximum effort, respectively. They attributed the difference in these means to the greater distance of the medial electrode from the quadriceps muscle mass. Hence, a significant level of cross talk from adjacent and even moderately remote muscles has been confirmed for both the thigh and lower leg. This complicates the determination of onset and cessation times of muscles' action; thereby confusing the precise identification of muscle phasing, which is a common clinical objective.

At present, there is no established method for circumventing these data complications. Research studies have demonstrated that double differentiation can reduce the cross talk to half or less (12,14). The necessary instrumentation, however, is just becoming available for use in the multiple muscle studies conducted clinically. Faced with this limitation, a possible

pragmatic approach might be to eliminate the low intensity signals representing 17 percent of maximum or from 7 to 10 percent of a typical submaximal peak intensity. This could clarify some of the phasing interpretations.

Cocontraction

The interpretation of simultaneous EMG in an agonist and antagonist may be confounded by the presence of cross talk. As Koh and Grabiner concluded, low-to-moderate signals recorded with surface electrodes may be a cross talk artifact rather than cocontraction (13,14). This was demonstrated in a recent study of cocontraction of antagonists in children (15). The authors showed continuous EMG throughout the gait cycle. Superimposed on an average 6.5 percent maximum intensity baseline were regularly interspersed peaks of 20 percent maximum (**Figure 6**). Wire electrode recording from the literature shows that the hamstrings and quadriceps normally overlap in their functions only during limb loading (1); hence, true cocontraction was phasic not continuous.

Wire Electrodes

Intramuscular placement of the EMG sensors circumvents the specificity limitations of surface electrodes. By having the electrode located within the target muscle, a much stronger signal is obtained and its frequency content is higher (**Figure 7**). Both qualities serve to virtually eliminate the problem of cross talk. While myoelectric signals from neighboring muscles may still spread through the tissues, their intensity is insignificant due to their distance from the electrodes.

A second advantage of wire electrodes is the opportunity to use the same signal gain for all muscles. A gain of 1,000 with wire electrodes provides a strong signal for all muscles. This allows the clinician to visually estimate the relative intensity of one muscle's action compared with the others. In contrast, the low reception of surface electrodes (**Figure 7**) commonly requires increasing the gain many fold to obtain a readable signal and the cross talk signals would be similarly magnified. Variability in soft tissue resistance also often necessitates adjusting the gain for individual muscles in order to obtain a readable signal. Thus, wire electrodes allow precise differentiation in the activity of adjacent muscles, making this technique preferable for surgical decisions.

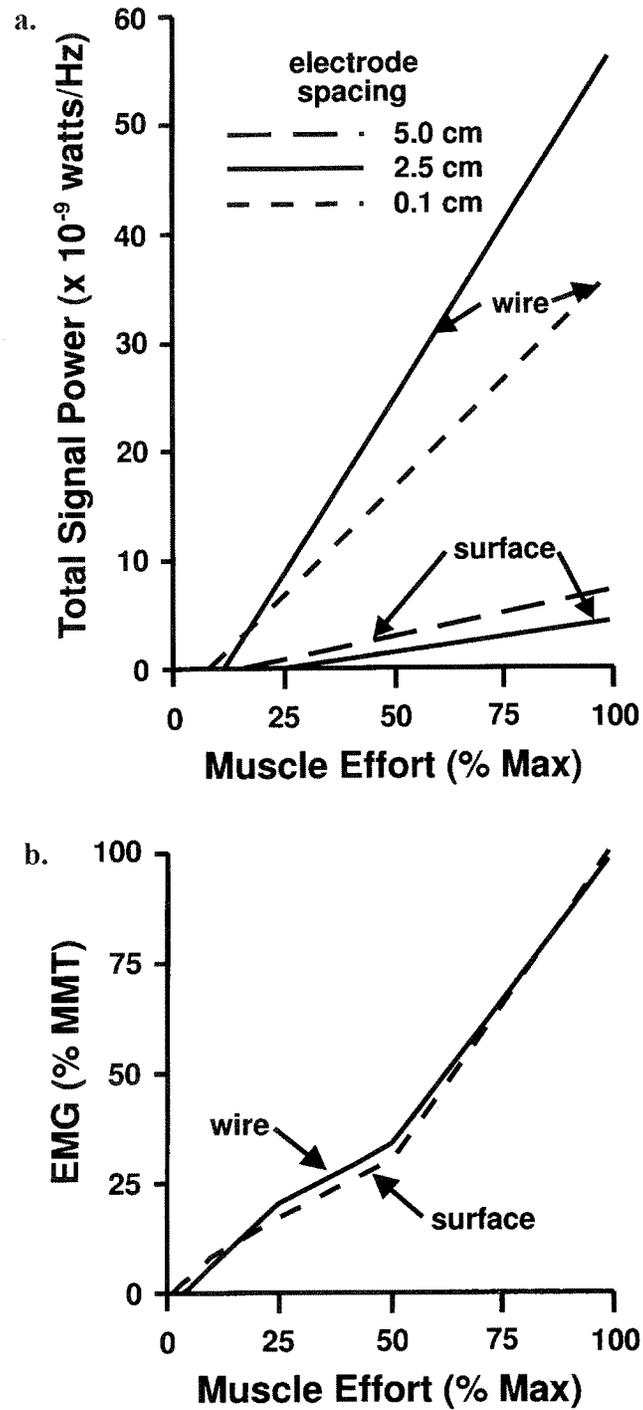


Figure 7.

a) Total signal power of wire and surface with different spacing between the paired electrodes. Wire 2.5-cm spacing inserted with separate needles. Wire 0.1-cm spacing represents single needle insertion. Surface 2.5- and 5.0-cm spacing indicates distance between the centers of two 1-cm diameter discs. b) The Effect of Normalizing: For each electrode (wire and surface), the EMG recorded at each effort level (%Max) was expressed as a percent of the EMG obtained during the isometric maximum muscle test (MMT). From reference (16). Used with permission.

The disadvantage of wire electrodes is the need for skin penetration as the wire electrodes are inserted into the muscle with a fine needle (gauge 25-30). Unless the subject has a bleeding tendency (which would contraindicate wires), the only penalty is momentary discomfort. This is minimized by tensing the skin, knowing the desired location and making a rapid insertion. Children as young as 4 years of age can be successfully tested with wire electrodes. Basmajian and Stecko's technique of inserting both wires with a single needle has simplified electrode location (17). A critical factor, however, is electrode fabrication. The end of the barbs must be of different lengths so that their bared tips will not contact each other and short-out the signal (**Figure 5b**, inset).

For both electrode systems, it is essential that the location relative to the target muscle be accurately determined. Following electrode application, activity of the target muscle is determined by palpable contraction and/or tension of its tendon during a low effort muscle test. Wire electrodes also allow precise localization by light electrical stimulation through the electrodes. Electrodes must be moved until the desired muscle action coincides with the EMG.

EMG Signal Timing

As each muscle provides a specific function, the basic information to be gained by dynamic electromyography is phasing within the gait cycle. The fundamental question is the time of onset and cessation of each muscle's activity relative to the limb motion. A second common concern is the time of peak effort. To make these determinations, some type of event marker must be included with the electromyographic recording to permit phasing. A similarly timed record of limb function is also needed. By itself, the EMG trace is a meaningless sequence of action potentials.

Event Marker

There are basically two methods of identifying the onset of the gait cycle, the use of a footswitch system or a synchronizing indicator on the visible video, motion, or force recordings. Either approach allows one to designate timing as percentage points within the gait cycle. It is customary to begin with initial floor contact as 0 percent and end with next initial contact as 100 percent. The functional significance is made clearer when the gait cycle is further divided into the functional subphases.

Footswitches offer the most versatile approach. While some normal gait studies use just a heel switch, this is seldom adequate as there is no indicator separating stance and swing. For pathological gait, a minimum of four switches on each foot is needed to accommodate the various modes of floor contact (18). The critical sites are heel, medial and lateral forefoot, and great toe. With this system, the basic phases of gait can be determined. The initial double support period identifies initial contact and the loading response phase. Lifting the other foot (contralateral toe-off) identifies single stance. Mid and terminal stance are distinguished as each being half of single stance. One can also relate the EMG pattern to the duration each foot segment is in contact with the floor. Pathology can alter the heel contact pattern in many ways with heel contact being absent, curtailed, or prolonged. While toe-off is the absolute endpoint of stance, a pathological toe drag may obscure the onset of swing. This not uncommon situation, contradicts using "toe-off" as the start of a gait cycle, which some investigators propose (19).

Timing Interpretation

The accuracy of defining the period of significant muscle function by electromyography varies with the technique used. A gross estimate can be made from the raw EMG tracing. This immediately introduces the question of the minimum significant signal (i.e., how small a signal has functional meaning). Most muscle action begins with small spikes representing preparatory activation of a few fibers prior to an EMG record, which progressively shows greater density and amplitude as the effort builds up to the dominant intensity. At the end of the action, there is a corresponding decrement. The slower the action, the more prominent are these small onset and termination packets. They are absent with ballistic movements. In addition, there may be scattered small spikes between the dominant EMG patterns. The inconsistency of these small spikes and amplitudes too small to represent more than trace function imply that they are inconsequential.

With experience, one can learn to subjectively filter out these small spikes by eye. Kaufman found "good agreement" among experienced therapists if they averaged 10 cycles². Di Fabio(20) found that computer designation with established criteria produced consistent reproducibility of onset times, whereas visual

² Personal communication, 1994.

analysis by three experienced therapists showed a 51 percent intra-examiner variability and only a 23 percent consistency among examiners. The Rancho computer criteria exclude spikes, which represent less than 5 percent of the muscle's manual muscle test value, and signal packets, which last less than 5 percent of the gait cycle (21). The purpose is to define meaningful muscle function. A second variable is natural inconsistency in timing between strides. The onset and cessation times from three gait cycles has proved to be representative of average function.

Abnormal Timing

Functionally significant deviations from normal timing may occur independently at either the onset or cessation of the EMG record, or both end points may be abnormal. These deviations have been classed as premature, delayed, curtailed, prolonged, continuous, and out-of-phase activity.

Delayed and curtailed EMG indicate inadequate muscle action. For example, curtailed tibialis anterior EMG shows function is limited to just the primitive flexor pattern during initial swing, while the lack of activity in the loading phase of stance identifies that it cannot accompany limb extension (**Figure 8**). Delayed onset of a muscle's EMG is an indication that activation is stimulated by a stretch stimulus rather than central gait control. For example, delay of gastrocnemius action until late terminal stance implies that the dorsiflexion torque was initially controlled by passive stretch of a contracture (**Figure 9**).

Premature, prolonged, or continuous timing are signs of excessive muscle activity. The usual effect is to oppose or partially inhibit normal motion. Premature onset of soleus EMG in swing is a common finding in persons who are spastic (**Figure 8**). Soleus activation accompanies the onset of the primitive extensor pattern by terminal swing knee extension. The unloaded foot is pulled into equinus, leading to premature floor contact by the forefoot. The functional consequence varies with the vigor of the action. A strong, prematurely active soleus can prevent heel contact with the floor, leading to just forefoot support throughout stance; thereby impairing weight-bearing stability.

Prolonged activity most often is found in the hamstrings and must be differentiated from other causes of persistent knee flexion in stance (**Figure 10**). Also, either or both the semimembranosus and the long head of the biceps femoris may act independently. Differences in their timing need to be clarified.

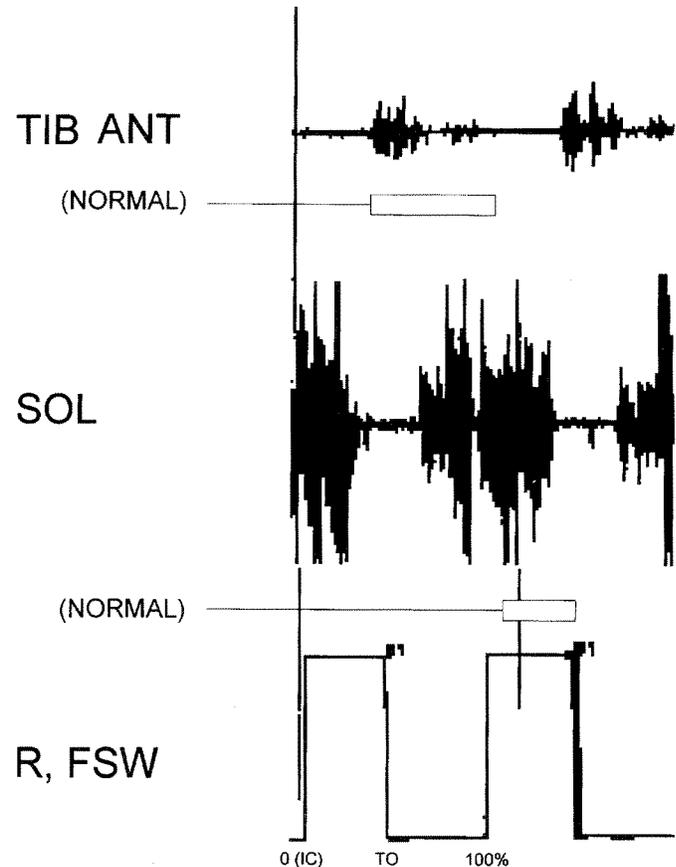


Figure 8.

Curtailed action (Tibialis Anterior): Tibialis anterior onset is appropriate but EMG ceases in mid swing, instead of continuing into the loading response phase of stance. This indicates proper flexor pattern action but inability to contract when the extensor pattern is active. The result is loss of foot support for stance. Premature activity by the soleus: Soleus activity begins in terminal swing rather than after the onset of stance. This implies the presence of a primitive extensor pattern. Footswitches (FSW) pattern identifies forefoot contact only. Diagnosis: cerebral palsy.

Out-of-phase EMG recordings are another form of excessive action. The tibialis posterior may become a swing phase muscle, thereby being the source of excessive foot varus rather than the tibialis anterior. Swing phase quadriceps activity is seen in all types of spastic gait. The effect is obstruction of knee flexion. A major difference among the diagnoses is the source of the obstructive force. Frequently, one or more of the vasti muscles are involved in stroke, head trauma, and spinal cord injury (**Figure 11**), whereas the rectus femoris is the dominant inhibitor of knee flexion in cerebral palsy. This latter situation has led to a

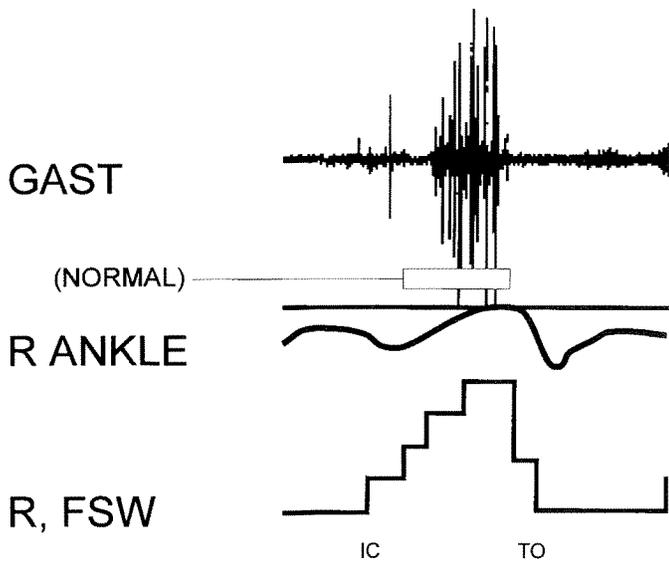


Figure 9. Delayed onset of gastrocnemius: Ankle goniometer (R Ankle) shows equinus (motion below baseline) at initial contact, which decreases under the stretching force of body weight progression. Gastrocnemius (GAST) EMG onset is delayed until 20% of the gait cycle (normal onset is 5%). This implies contracture tension is the early plantar flexor force prior to stretch, stimulating muscle action. Footswitch (R,FSW) "staircase" identifies stance, baseline is swing. IC= initial contact; TO=toe-off. Diagnosis: post polio.

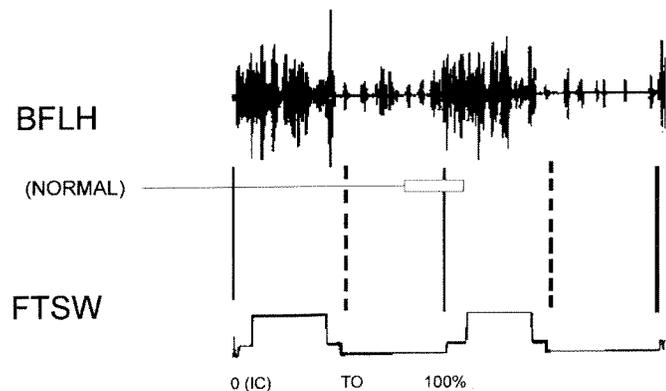


Figure 10. Prolonged activity of the biceps femoris, long head (BFLH) until late mid stance: The effect was persistent knee flexion in stance beyond the loading response phase that followed initial floor contact (IC). FTSW=footswitch. TO=toe-off. 0 to 100% identifies one gait cycle. Diagnosis: Stroke hemiplegia.

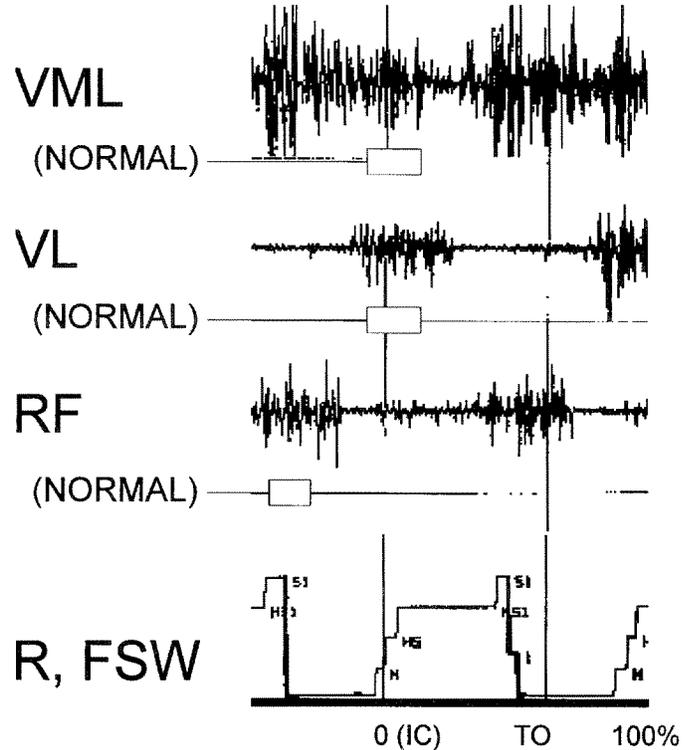


Figure 11. Out-of-Phase activity of the vastus medialis longus (VML): The continuous EMG identifies swing phase action as well as prolonged activity in stance. Vastus lateralis (VL) displays a nearly normal EMG, identifying spastic muscles have individual sensitivities to stretch and primitive control. Rectus femoris (RF) action is prolonged. Both the VML and RF activity could impede swing phase knee flexion but the more dense EMG indicates that the VML is the dominant inhibitor of knee flexion. R,FSW (right footswitch) designates stance (staircase) and swing gait phases. Subscripts (H,5,1) indicate foot area contacting the ground. Nearly continuous H (heel) contact implies calf muscle weakness. IC=initial contact; TO=toe-off. Diagnosis: Adult traumatic brain injury.

technique of using surface electrodes to identify when the rectus femoris is the cause of limited swing knee flexion (22). For all other diagnoses, intramuscular wires are needed to differentiate rectus femoris action from out-of-phase vastus activity (23).

There are no criteria for the duration of a timing error needed before motion is altered but usually the abnormal timing is quite gross. Superimposed on the timing error is the effect of muscle intensity.

EMG Intensity

Muscles increase their force by the activation of additional muscle fibers or by increasing their firing

rate. Both responses create a more intense electromyogram. Signal amplitude is increased as the simultaneous action potentials add together, while asynchronous potentials form new spikes. Visual inspection reveals an electromyogram with both amplitude and density increased. The level of EMG recorded during gait may or may not be similar to that occurring during the baseline muscle test. Normally, peak gait intensity is approximately a third of the maximum test level. A gait record that exceeds the muscle test is an indication of poor voluntary control. In interpreting the raw clinical record, there are four significant levels of function: absent, inadequate (weak), appropriate (strong), and excessive. Absent gait EMG in a muscle with a notable muscle test value implies that either it is shielded from stretch or being avoided as a detrimental force. Inadequate intensity implies muscle presence but inability to meet the functional demand. Excessive intensity, in the presence of a good muscle test record, is a sign of either obstructive force or muscle overuse and potential fatigue (**Figures 12a** and **12b**). Visual comparisons of relative intensity among muscles are very convenient with wire electrode records, since the same amplification is used for all muscles. With surface electrodes, however, obtaining a readable record generally requires the tester to individually adjust the amplification of each muscle record to overcome the difference in the impedance of the overlying skin and soft tissues. Hence, similar record amplitudes can represent very different muscular effort.

Muscle intensity also can be quantified by either a descriptive scale or computer measurement. A customary descriptive scale uses four intensity levels, with grade four indicating maximum. Small changes, however, are difficult to identify. Today, it is more common to quantify the EMG by computer. This allows fine grading of the muscular effort and accurate discrimination of small differences.

Computer Signal Quantification

Three steps are involved in providing a meaningful numerical value for the muscles' EMG. The raw EMG is rectified, digitized, and normalized (**Figure 13**). Normalization permits the comparison of effort changes among two or more muscles despite the inability to either determine or control the number of muscle fibers that an electrode samples.

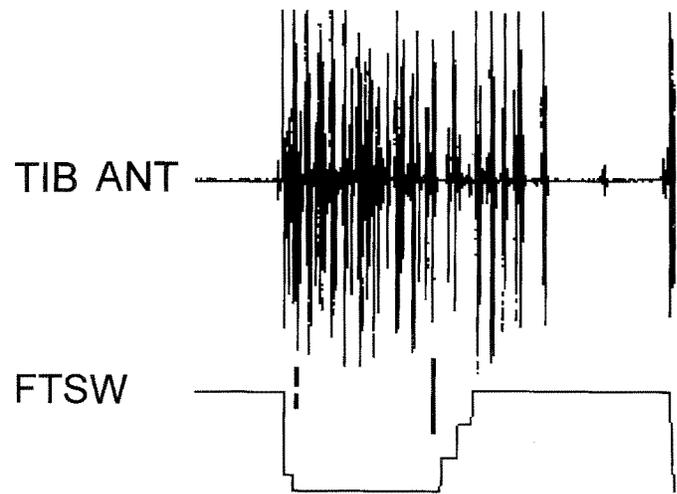


Figure 12a.

The EMG of this tibialis anterior represents a sparse number of enlarged motor units functioning at a higher than normal intensity (quantified as approximately 70% of its muscle test). Excessive action also is evident by the persistence of the same intensity throughout its function phase (swing and early stance). Diagnosis: Post poliomyelitis muscle overuse.

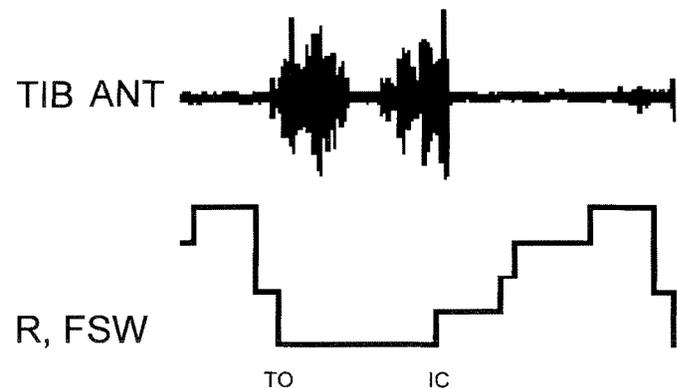


Figure 12b.

Normal tibial anterior is EMG of a young adult: The quick, dense EMG packet provides vigorous dorsiflexion to lift the foot from its plantar flexed position at the onset of swing (TO). Tissue tone is sufficient to support the foot in mid swing (EMG absent). Second burst reactivates dynamic dorsiflexion to support the foot in terminal swing and early stance.

Normalization

To accommodate the need to use uncontrolled EMG samples, all of the EMG values obtained for a given muscle are compared to a normalizing base. Most commonly, this base is the EMG accompanying a

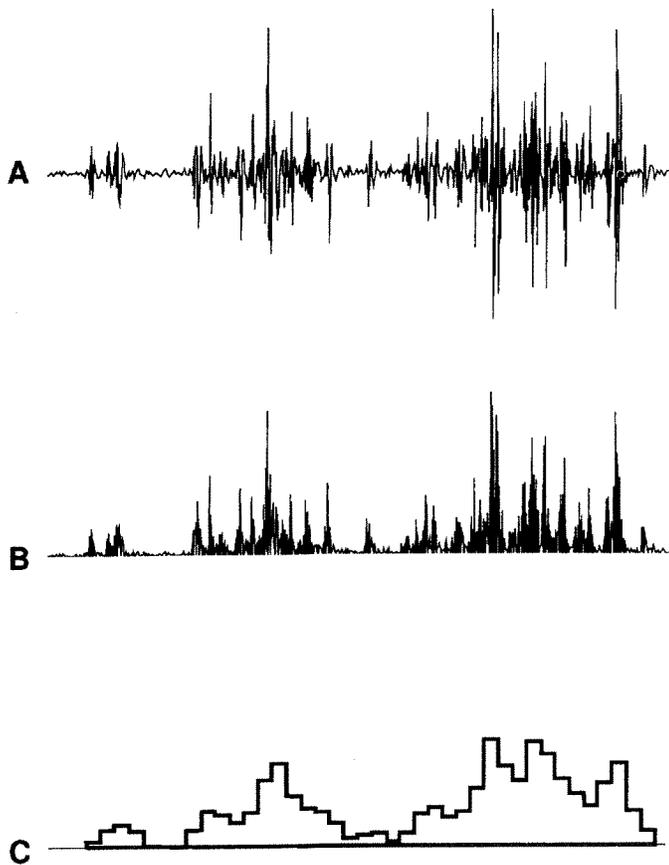


Figure 13.

Computer quantification of EMG record: a) the raw analog data are collected digitally by sampling the signal at 2500 Hz; b) the signals then are rectified by transposing the negative values to positive; and c) the data are normalized and summed over designated intervals (usually 1% of the gait cycle) to generate a linear envelope that expresses the data as percents of the maximum EMG reference.

maximum effort by that muscle. Hence, the individual test values are expressed as a percentage of the base value (i.e., %MVC).

To meet the time constraints of simultaneously testing six or eight muscles in a clinical setting, the manual muscle test maximum is the customary normalizing base (%MMT). The procedure consists of recording the EMG during the maximum effort test, calculating the mean for the one second with the highest EMG, and then relating each functional EMG to that value using a common time interval, generally 0.01 second or 1 percent of the gait cycle.

An alternate approach uses each muscle's peak EMG in the gait cycle as the normalizing base and all

other phase values are related to it. This is convenient but it does not allow one to compare relative intensity among muscles, since the peak effort for each is 100 percent. This most often is used in situations where poor patient cooperation makes muscle testing difficult.

Electromechanical Delay

The time between the onset of the myoelectric signal and the initiation of muscle tension is called the electromechanical delay (EMD). This interval is assumed to represent the propagation of the action potential along the muscle, the excitation-contraction coupling process, and stretching of the muscle's series elastic component by the contracting component (8). This delay is significant only if one wants to precisely relate EMG and motion in selected research studies. In general clinical practice, however, the difference in timing is inconsequential. As the following summary identifies, it also involves a very short time period (5,8,24–26).

The differences have been found to relate to three variables: method of muscle activation, mode of recording the signals, and the method of identifying muscle tension. Voluntary effort created the longest delays, and knee extension, which requires moving a larger mass than elbow flexion, was slower. Significantly faster stimulation was attained with a reflex hammer or an electrical current (**Figure 14**). Among the methods of identifying the onset of muscle tension, the slowest was a gross exercise unit, such as a Kin-Com or goniometer (26). A load cell force transducer in intimate contact with the leg registered a quick response (8,27), but the most sensitive motion instrument was an accelerometer. Involved in these differences are both the inertia of the limb and the lag within the mechanical testing system (8). Different effort levels and comparisons of isometric and isotonic action showed only minor differences in the electromechanical delay between onset times, but increasingly higher target forces required proportionally greater total time. The combination of tendon tap stimulation of knee extension measured with a force transducer registered an EMD of 25 ms and electrical stimulation shortened the delay to 20 ms. The shortest EMD (16 ms) was recorded by testing voluntary biceps activation of elbow flexion using an accelerometer for motion sensing and gross magnification of the record for easier reading of the data. It was calculated that the transport time involved only 10 ms (24). Returning to

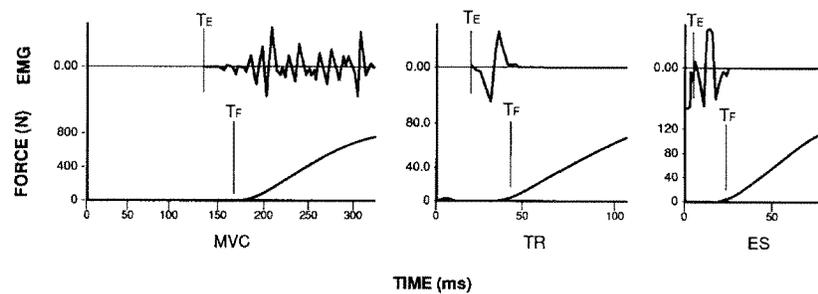


Figure 14.

Electromechanical delay (EMD): Onset timing of EMG and force during three modes of quadriceps (vastus lateralis) activation. Left. Voluntary knee extension (EMD=40 ms); middle. Tendon reflex (EMD=25 ms); right. Electrical stimulation (EMD=19 ms). TE (EMG onset threshold, 0.015 mV), TF (force onset threshold, 3.6N). Note EMD reduced by promptness of muscle activation. Adapted from reference (8). Used with permission.

the question of gait electromyography, the tendon tap response could be likened to eccentric activation during walking. A logical conclusion to draw from these multiple studies is that the average electromechanical delay during gait is no more than 40 ms and perhaps as short as 25 ms or even 10 ms.

EMG Force

Activation of an increasing number of muscle fibers results in a correspondingly greater force. The EMG also increases. The result is a quasi-linear relationship between force and EMG when the muscular effort is isometric but the precise relationship varies with the mode of motor unit recruitment (28). To interpret muscle force from an EMG of different effort levels, however, the data have to be normalized as the ratio (linear slope) between these two factors varies with the muscle studied, electrode placement, and mode of signal recording, and because the number of the motor units sampled and their muscle fiber composition can neither be defined nor controlled (29,30).

Motion markedly distorts the isometric (I) relationship of EMG and force by changing the effectiveness of the muscle fibers, while the EMG continues to identify the relative number of fibers included in the sample. Muscle force (F) is modified by joint position (P), mode of contraction (C), and speed of action (V). The conceptual model may be represented as $F=I(V+P+C)$.

Joint position alters two muscle factors: sarcomere effectiveness and moment arm length. Each muscle

fiber is a chain of force units called sarcomeres; within which force production capability is determined by the number of bonds between its myosin and actin filaments. Maximum bonding occurs in the midrange of the sarcomere with force being reduced by either lengthening or shortening of the sarcomere. The length of the sarcomere chain (i.e., muscle fiber) is determined by joint position. Recent *in vivo* studies of wrist extensor sarcomeres have shown that even synergistic muscles (extensor carpi radialis brevis and longus) have optimum sarcomere bonding at different joint positions. Effectiveness of the resulting muscle force in creating motion (moment) is further modified by its functional leverage (moment arm), which also varies with joint position. Optimum sarcomere bonding and moment arm lengths commonly occur at different joint positions, a situation that seems to extend the functional effectiveness of the muscle. For example, quadriceps muscle force is maximum at 60° of flexion (31), but the longest moment arm for the patellar tendon is found at 15° flexion (31).

Muscles have three modes of contraction: isometric (no motion, the dynamic force equaling the passive resistance), eccentric (active resistance to passive lengthening), and concentric (active shortening). The latter two modes are forms of motion. In some muscles, such as the biceps brachii, the eccentric force can exceed isometric capability by 10 to 20 percent. For the quadriceps, isometric and eccentric appear to be similar (32). Eccentric holding by the actin-myosin bonds is

enhanced by titin, a third protein (33). Concentric contraction requires serial re-bonding of the actin and myosin protein filaments as the muscle actively shortens. This is less efficient, resulting in a force approximately 20 percent less than isometric. Hence, for the same EMG signal, the resulting force depends on whether the effort is isometric, eccentric, or concentric; while the EMG representative of muscle fiber involvement remains unchanged (**Figures 15a and 15b**).

The velocity of motion influences the muscle force of concentric effort but not eccentric activity. As actin – myosin bonding is rate dependent, sarcomere stability is reduced with fast shortening contractions, and muscle force correspondingly decreases. During walking, sarcomere sensitivity to speed relates only to swing phase events. In stance, muscle action is primarily isometric and eccentric; thus, there is a reliable relationship between the normalized EMG and the muscle forces being employed.

EMG Relationship to Moments

During walking, the amount of effort a muscle must exert at any instant in time is determined by the destabilizing influence that falling body weight has on the joint controlled by that muscle. Engineers define this destabilizing rotational force as a moment. The significant factors are the magnitude of the falling body weight force (measured as a ground reaction force) and the perpendicular distance between that force line (vector) and the joint center (moment arm). Stability is preserved by an equal and opposite moment from muscular action. This approach is an accurate representation of normal muscle group function. Antagonistic cocontraction is minimal and there are no other significant destabilizing forces. At the knee, for example, the moment calculation is a good representation of quadriceps effort during weight acceptance as the period of hamstring activity at the onset of stance is brief and of low intensity. At the same time, the mechanics at the foot are contributing to the demand moment. Hence, there are no hidden forces to impose significant deviations in the moment calculations.

A commonly unrecognized problem, however, is the assignment of muscle action to passive events. Contrary to pure mechanics, the human body has an acute feedback system (proprioception), which allows

intelligent use of passive mobility. Examples are mid stance hip extension and late stance hip abduction induced by the fall of body weight following the swinging limb (34). In these instances, passive momentum has been used instead of muscle agonists (35). Moment calculations have the added limitation of identifying only group muscle action. Delineation of individual muscle activity necessitates dynamic EMG.

Pathology can impose serious compromises to the prediction of muscle action with moments. In spastic diseases, such as spastic paralysis or stroke, intense cocontraction may exist. Prolonged cocontraction by the hamstring muscles may require greater quadriceps intensity than is indicated by moment calculations. Faulty foot support by either prolonged heel only or forefoot floor contact also can impose unrecognized instability at the knee and hip, leading to additional muscle action not evidenced by the calculated moment. For example, persons with spastic paralysis who have a crouch gait as the result of prolonged hamstring muscle action, preserve balance over their flexed knee by leaning forward. Associated limitations in ankle dorsiflexion impose a toe stance. The resulting posture is accompanied by EMG recordings showing strong quadriceps and antagonistic hamstring activity. Simulation of this posture in nonimpaired subjects confirmed intense cocontraction of agonists and antagonists at both the ankle and knee resulting from limb posture rather than spasticity (36).

SUMMARY

Dynamic electromyography enables the clinician or research investigator to define the timing and intensity of individual muscle function during gait and other functional activities. Moment calculations identify the action of controlling muscle groups during normal function, but may become inaccurate when pathology alters the balance of passive and active forces. Wire electrodes, by their placement within the designated muscle, provide a more precise definition of both timing and intensity of muscle action than do surface electrodes, but require needle penetration of the skin. Surface electrodes have the advantage of convenience.

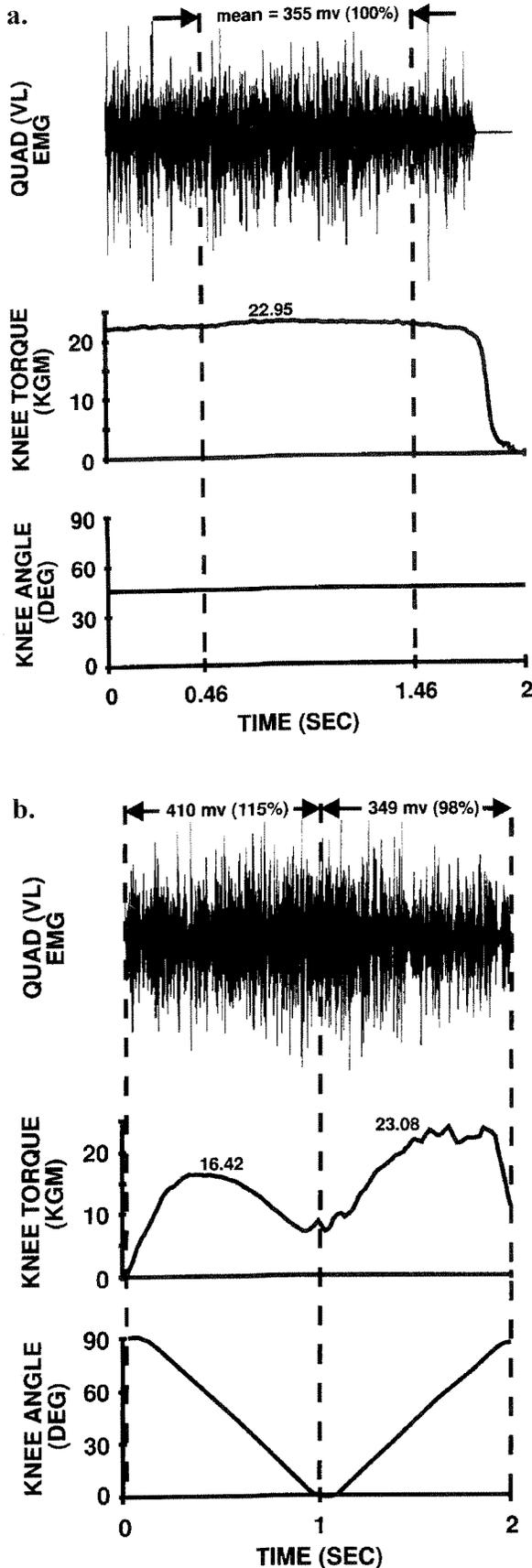


Figure 15.

EMG – Force Relationship per Type of Muscle Contraction: All tests were maximum knee extension and the data were calculated over a 1-second time period. Quadriceps EMG is represented by vastus lateralis (VL). a) Isometric maximum effort at 45° of knee flexion: VL, raw EMG signal and mean intensity (millivolts), also 100%. Torque, analog recording, and peak intensity (KGM, kilogram meters). b) Concentric (left) and Eccentric effort (right). Direction of motion indicated by knee angle pattern. Test arc was between 90° and 0° flexion. Rate was 90° per second. Mean EMG and % isometric were quantified for the 1-second effort in each direction. Torque was calculated as the peak value for 0.1 second. Expression of data as % isometric values showed motion modified concentric force production (EMG 115%, Torque 72%) but not the eccentric effort (EMG 98%, torque 101%).

REFERENCES

1. Perry J. Gait analysis, normal and pathological function. Thorofare, NJ: Charles B. Slack; 1992.
2. Lieber RL. Skeletal muscle structure & function. Baltimore: Williams & Wilkins; 1992. p. 22.
3. Goodgold J, Eberstein A. Electrodiagnosis of neuromuscular disease. Baltimore: Williams & Wilkins; 1972. p. 21–28.
4. Basmajian JV, DeLuca CJ. Muscles alive: their functions revealed by electromyography. 5th Ed., Baltimore: Williams & Wilkins; 1985. p. 19–64.
5. Inman VT, Ralston HJ, Saunders JBdM, Feinstein B, Wright EW, Jr. Relation of human electromyogram to muscular tension. *Electromyogr Clin Neurophysiol* 1952;4:187–94.
6. Burke RE, Levine DN, Salzman M, Tsairis P. Motor units in cat soleus muscle: physiological, histochemical and morphological characteristics. *J Physiol(Lond)* 1974;238:503–14.
7. Bodine-Fowler S, Garfinkel A, Roy RR. Spatial distribution of muscle fibers within the territory of a motor unit. *Muscle Nerve* 1990;13:1133–45.
8. Zhou S, Lawson GA, Morrison WE. Electromechanical delay in isometric muscle contractions evoked by voluntary, reflex and electrical stimulation. *Eur J Appl Physiol* 1995;70:138–45.
9. Freeborn CW, Antonelli D, Perry J. Spectral analysis of EMG signals. Annual Reports of Progress. Downey, CA: Rancho Los Amigos Rehabilitation Engineering Center; 1979. p.35–7.
10. De Luca CJ. The use of surface electromyography in biomechanics. *J Appl Biomech* 1997;13:135–63.
11. Perry J, Easterday CS, Antonelli DJ. Surface versus intramuscular electrodes for electromyography of superficial and deep muscles. *Phys Ther* 1981;61:7–15.
12. De Luca CJ, Merletti R. Surface myoelectric signal cross-talk among muscles of the leg. *Electroencephalogr Clin Neurophysiol* 1988;69:568–75.
13. Koh TJ, Grabiner MD. Cross talk in surface electromyograms of human hamstring muscles. *J Orthop Res* 1992;10:701–9.
14. Koh TJ, Grabiner MD. Evaluation of methods to minimize cross talk in surface electromyography. *J Biomech* 1993;26 Suppl 1:151–7.

15. Frost G, Dowling J, Dyson K, Bar-or O. Cocontraction in three age groups of children during treadmill locomotion. *J Electromyogr Kinesiol* 1997;7:179–86.
16. Perry J, Antonelli D. The dynamics of EMG, force, movement relationships for knee extension. Annual Reports of Progress. Downey, CA: Rancho Los Amigos Rehabilitation Engineering Center; 1980. p. 45–8.
17. Basmajian JV, Stecko G. The role of muscles in arch support of the foot. *J Bone Joint Surg* 1963;45A:1184–90.
18. Bontrager E. Footswitch stride analyzer. *Bull Prosthet Res* 1981;18(1):284–8.
19. DiVita P. The selection of a standard convention for analyzing gait data based on the analysis of relevant biomechanical factors. *J Biomech* 1994;27:501–8.
20. DiFabio RP. Reliability of computerized surface electromyography for determining the onset of muscle activity. *Phys Ther* 1987;67(1):43–8.
21. Perry J, Bontrager EL, Bogey RA, Gronley JK, Barnes LA. The Rancho EMG Analyzer: a computerized system for gait analysis. *J Biomed Eng* 1993;15:487–96.
22. Ounpuu S, DeLuca PA, Bell KJ, Davis RB. Using surface electrodes for the evaluation of the rectus femoris, vastus medialis and vastus lateralis in children with cerebral palsy. *Gait Posture* 1997;5:211–6.
23. Agarwal GC, Gottlieb GL. Mathematical modeling and simulation of the postural control loop, Part II. *Crit Rev Biomed Eng* 1984;11:113–54.
24. Corcos DM, Gottlieb GL, Latash ML, Almeida GL, Agarwal GC. Electromechanical delay: an experimental artifact. *J Electromyogr Kinesiol* 1992;2:59–68.
25. Ralston HJ, Todd FN, Inman VT. Comparison of electrical activity and duration of tension in the human rectus femoris muscle. *Electromyogr Clin Neurophysiol* 1976;16:277–86.
26. Vos EJ, Harlaar J, Schenau GJVI. Electromechanical delay during knee extensor contractions. *Med Sci Sports* 1998;23:1187–93.
27. Viitasalo JT, Komi PV. Interrelationships between electromyographic, mechanical, muscle structure and reflex time measurements in man. *Acta Physiol Scand* 1981;111:97–103.
28. Solomonow M, Baratta R, Shoji H, D'Ambrosia R. The EMG-force relationships of skeletal muscle; dependence on contraction rate, and motor units control strategy. *Electromyogr Clin Neurophysiol* 1990;30:141–52.
29. Lawrence JH, De Luca CJ. Myoelectric signal versus force relationship in different human muscles. *J Appl Physiol* 1983;54(6):1653–9.
30. Perry J, Bekey GA. EMG-force relationships in skeletal muscle. *Crit Rev Biomed Eng* 1981;7(1):1–22.
31. Perry J, Antonelli D, Ford W. Analysis of knee-joint forces during flexed-knee stance. *J Bone Joint Surg* 1975; 57A(7):961–7.
32. Smidt GL. Biomechanical analysis of knee flexion and extension. *J Biomech* 1973;6:79–92.
33. Horowitz R, Podolsky RJ. The positional stability of thick filaments in activated skeletal muscle depends on sarcomere length: evidence for the role of titin filaments. *J Cell Biol* 1987;105:2217–23.
34. Hardt DE. Determining muscle forces in the leg during normal human walking: an application and evaluation of optimization methods. *J Biomed Eng* 1978;100:72–8.
35. Lyons K, Perry J, Gronley JK, Barnes L, Antonelli D. Timing and relative intensity of hip extensor and abductor muscle action during level and stair ambulation: an EMG study. *Phys Ther* 1983;63:1597–605.
36. Thomas SS, Moore C, Kelp-Lenane C, Norris C. Simulated gait patterns: the resulting effects on gait parameters, dynamic electromyography, joint moments and physiological cost indices. *Gait Posture* 1996;4:100–7.

JACQUELIN PERRY, M.D., D.Sc. (HON), is the world's leading authority on the biomechanics of human locomotion. She has been a Board Certified Orthopaedic Surgeon since 1958. She is a long-time member of the American Academy of Orthopaedic Surgeons, as well as of many medical societies. She served in the United States Army as a physical therapist, from 1941 to 1945, after being certified in physical therapy at Walter Reed Army General Hospital and is an active member of the American Physical Therapy Association. She has been a member of the Department of Veterans Affairs, Rehabilitation Research and Development Service, Scientific Review and Evaluation Board, since 1982, reviewing research grant proposals submitted to the VA.

Dr. Perry joined the Rancho Los Amigos Hospital staff as the Chief of Orthopaedic Surgery of the Adult Poliomyelitis Service in 1955 and has been the Chief of Pathokinesiology Service, Director, Quality Assurance Program since that time. Paralytic hand dysfunction led her to initiate a dynamic electromyography program in 1961 to study hand muscle phasing with intramuscular (wire) electrodes. The same year, vaccine eradication of acute polio allowed Dr. Perry to start a CVA Service. Inconsistencies between observed gait deficits and clinical findings led to the initiation of a gait laboratory to define muscle function by EMG. A foot switch system was designed for timing. Motion analysis by video observation was later augmented by kinematic and kinetic systems. Technical refinements to allow clear delineation of adjacent muscle action and automated EMG interpretation have been her major objectives.

Dr. Perry has over 300 publications, the most prominent being her book *Gait Analysis: Normal and Pathological Function*. She has received 27 formal honors for her work in orthopaedic surgery, gait analysis, and rehabilitation.

In 1998, Dr. Perry and her staff moved into the Jacquelin Perry Neuro-Trauma Institute and Rehabilitation Center (JPI), a new three-story, state-of-the-art hospital. She was awarded the honorary degree of Doctor of Science by the University of California and the Helen J. Hislop award for Outstanding Contributions to Professional Literature at the 1998 APTA Annual National Conference Honors and Awards ceremony. Her current status is emeritus professor of orthopaedic surgery, emeritus professor of biokinesiology and physical therapy, medical consultant for the Rancho Los Amigos Pathokinesiology Service (which she established in 1968 for the primary purpose of studying normal and pathological gait) and Centinela Hospital biomechanics laboratory, Chief of the Post-Polio Service, and gait consultant to the Traumatic Brain Injury Service.