PRACTICAL PROBLEMS IN MYOELECTRIC CONTROL OF PROSTHESSES

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Since the day that Luigi Galvani watched his frogs' legs jumping in response to electric stimulation, muscle contraction and its related phenomena have piqued the interest of physiologists and engineers alike (1). Among other things, his work led to postulates concerning the electric characteristics of neural impulse transmissions and muscle contraction, theories elaborated by two Nobelists early in the 20th century (2). Since then the myoelectric activity of muscles has been used for diagnostic purposes and more recently, for controlling prosthetic devices. At the present time there are at least six upper-extremity prosthetic components designed for control by the electrical activity of residual muscles.

One of the earliest examples of control of a prosthetic device by myoelectric signals was a hand developed in the Soviet Union and demonstrated at the Belgium 1958 World's Fair. A wave of interest in this field was generated in this country and abroad leading to the design of other hands and elbows controlled by myoelectric signals.

If it were possible to sense and interpret the signals from the brain, one would assume that a perfectly controlled device could be built. This logic assumes that the brain functions like a switch turning on a motor; actually, the brain depends upon feedback to control motion and needs no linear relation between signal and action. In fact, early myoelectric devices do not require linear relationships. The Russian and Canadian hands (the Canadian is the same as the Russian hand but has an improved integrated circuit for control) detect signals from two muscles; when the signal from the wrist flexors exceeds a threshold the hand closes; when the signal from the wrist extensors exceeds a threshold the hand opens. Several of the presently available electric hands still operate in this manner. Both the Hendon hand (3) and the Boston Arm relate force and position to EMG signal through the use of velocity and force feedback to the amplifier which powers the
device. As in other areas, there is a tendency in a developing field to over-refine and over-elaborate the methods for exploiting a potentially useful phenomenon such as myoelectricity. In this connection a long sought goal has been the design of a prosthetic component or system which would respond in direct proportion to the level of electric activity of the muscle.

Precise proportional control of prosthetic devices depends on the relationship between tension exerted in a traumatized muscle and the myoelectric signal produced. Many attempts to describe these relationships in normal muscles are reported in the literature (4, 5, 6). The work to date presents different and sometimes contradictory results. Several studies report only a rough relationship between muscle tension and myoelectric output while others seem to describe a precise linear relationship. These studies are difficult to interpret since in some cases only isometric tension in the muscle and one myoelectric parameter, such as R.M.S. voltage, were considered. The full spectrum of tension with respect to loads, type of contraction, and duration of contraction has not been systematically studied. Variation is also evident in the treatment of the myoelectric data and the equipment capabilities used in recording the phenomenon. No systematic treatment of myoelectric amplitude, frequency, pulse count, R.M.S. voltage, integrated voltage, and power spectrum analysis has been completed. Despite the inconclusive nature of the data on muscle tension versus myoelectric output, many design engineers continue to work as though linear relationships were clearly established, and continue to expend increasing effort on refining amplifiers and proportional feedback systems to take advantage of a neurophysiological relationship which they assume to be present in the human body but which has not been demonstrated.

The common concept underlying the production of a myoelectric signal is that each muscle fiber produces an electric discharge pattern that is similar from fiber to fiber and muscle to muscle, at least as regards skeletal muscle. If the contraction of each microscopic fiber yields an increment of contractile force and electric signal, then an area over a muscle, sensed by a surface electrode, should show a definite relationship between the total tension in the muscle and the total electric output of that muscle.

A simple experiment conducted in the VAPC laboratory demonstrated that at best, only a rough relationship exists between muscle tension and myoelectric output under conditions appropriate for prosthetic control, i.e., isometric contraction against sub-maximal loads.

One normal adult male was the subject of this experiment. He stood leaning against a tilt table set at an angle of 85 deg. to the horizontal with his arm flexed and parallel to the ground (to stabilize the upper
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The equipment consisted of a Honeywell 1612 Visicorder with galvanometers having a natural frequency of 10,000 cycles per second and a preamplifier and power amplifier. Each element of the amplifier combination had a maximum gain of 250 with a frequency response from 0.1 Hz to 20,000 Hz, an input impedance of 10 megarohms and a built-in 5 Hz microvolt square wave calibrating device. Biopotential skin electrodes were fabricated of a sintered silver-chloride pellet. A liquid junction (electrode paste) to the skin assured the finest signal.
FIGURE 2.—Samples of the raw recorded EMG signals and the average conditional values. Each horizontal line corresponds to 1 millivolt; each vertical line corresponds to 0.1 second. The signal samples report only a small fraction of the information used in arriving at the average figures for voltage or count.

<table>
<thead>
<tr>
<th>CONDITION</th>
<th>BICEPS SIGNALS</th>
<th>RMS VOLTAGE (in volts)</th>
<th>PULSE COUNT (per second)</th>
<th>TRICEPS SIGNALS</th>
<th>RMS VOLTAGE (in volts)</th>
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<tbody>
<tr>
<td>HORIZONTAL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 POUNDS</td>
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<td>1.5</td>
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<td>2 1/2 POUNDS</td>
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<td>1.6</td>
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<tr>
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<td>3.2</td>
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<tr>
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<td></td>
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<td>1.2</td>
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<tr>
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<td>20.0</td>
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Bulletin of Prosthetics Research—Spring 1970

EMG SIGNAL vs LOAD
system available. Both biceps and triceps were monitored as a weight was applied to the palm of the patient's hand with his elbow held at 85 deg. of flexion. The maximum voluntary outputs of the biceps and triceps were of approximately the same magnitude and totally independent of each other. The test consisted of 12 series of loadings from 0 to 20 lb. applied to the hand as follows: 0, 1, 2.5, 5.0, 7.5, 10, 15, 20 lb. A duration test of 15 min. holding a 5-lb. load was also conducted to determine changes with time (Fig. 1).

As shown in Figure 2 the relationship between external load and myoelectric signal in terms of pulses or R.M.S. is neither linear nor exponential. Pulse count using the Bergstrom (6) method varied over a small range from 143 to 222 pulses per second, with no apparent relationship to the applied load. The R.M.S. of the biceps myoelectric output increased with increasing load but certainly not in a linear fashion.

Further reference to Figure 2 indicates that the myoelectric activity of the triceps when only the biceps was loaded, was not significantly affected by the tensions produced in the biceps at loads up to 10 lb. Between 10 and 20 lb., however, the electric activity of the triceps began to increase. We believe that the increase in triceps activity is nothing more than an electric transmission of the increased activity of the biceps. The signal generated by the biceps is transmitted through the skin to the electrodes directly above it with a minimal voltage division. But the signal is also transmitted through the arm in the other direction. The bone, skin, and muscle act as a resistive network. The same signal, sensed by the electrodes on the posterior surface of the arm, has been reduced in intensity but the recordings show that exactly the same characteristics are present in both traces, indicating that they came from the same source. This is in sharp disagreement with the established idea that muscles oppose each other for control. The implication for design and development of myoelectrically controlled devices is that while electronic control circuitry must sense the activity of both biceps and triceps (for example), only the higher signal should be used for a given control mode.

Another widely held conception relates to the onset of fatigue in a muscle under sub-maximal load and its influence on myoelectric output. As shown in Figure 3 when the patient supported a load of 5 lb. for periods of 15 min., he reached excruciatingly painful levels of fatigue, but the myoelectric output was unchanged.

In the face of these results, it is difficult to see why designers continue to search for improved methods of conditioning myoelectric signals in an effort to improve "proportionality," i.e., the relationship between myoelectric signal and mechanical output when the results cannot be better than the raw, fundamental data. They would be
better advised to avoid over-designing hardware beyond the limits of the basic phenomenon. Refining hardware in electronic circuitry to provide better proportional control than is available in the patient is like designing a Hi-Fi system which faithfully reproduces a million cycles per second. This is high fidelity indeed, but no one can hear it. Recognizing that only a rough proportionality exists between muscle tension and myoelectric output will lead to more economical and more useful components. If the afferent nerve system can be reactivated closing the control loop for the man to operate a device then more sophisticated devices will be needed. This is presently being attempted in Osaka, Japan, by Ziro Kawamura, M.D., and Osamu Sueda, M.S., who have successfully used vibrating feedback to the stump to indicate prehension in a conventional Dorrance hook.
REFERENCES

1. Luigi Galvani: De Viribus Electricitatis in Motu Musculari Commentarius, 1792.