

BME/EECS 458 - Biomedical Instrumentation & Design

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6.0 Introduction - Electromyogram

It is possible to record the action of skeletal muscle in the body using either strain gage sensors monitoring the displacements and forces produced by the muscle or biopotential electrodes sensing electrical activation. Direct force measurements require intimate contact between the muscle and strain gage. For body surface recordings, this presents a problem. However, the electrical activity of skeletal muscles can be recorded by applying electrodes to the skin above the muscle in question. The pattern produced by the combined action potentials of many motor units is called an electromyogram. In this chapter we will restrict our discussion to biopotential surface recordings of this type.

To understand the origin of the electromyographic signal we will first look at some basic principles of cellular electrophysiology, then the characteristics of electrically excitable tissue, and finally how electromyography works. To get a better intuitive feeling for the electrochemistry of the cell membrane of excitable tissue, please look at Web Site from UCLA (<http://pb010.anes.ucla.edu/>). This is one of the best sources for this information. It includes computer simulations and great graphics. You should download some of the graphics from that Web Site to enhance these notes.

6.1 Electrophysiology of the Cell

The electrochemical properties of a typical cell are dominated by electrolyte transport across the cell membrane. The simplest model of a cell membrane is a semipermeable barrier to ionic exchange between intracellular (within the cell) and extracellular (outside the cell) spaces. Consider the simple system below.

Panel A shows a two-sided system separated by a semipermeable membrane allowing cations (positive ions) to go through but **not** allowing negative ions (anions) to permeate. Consider the case where a concentration of KCl is placed in aqueous solution on the left side and water is placed on the right. Both K^+ and Cl^- ions will attempt to diffuse across the membrane, but only K^+ will make it to the right side. The instant a K^+ ion moves through a pore, it necessarily leaves behind a Cl^- ion. The space charge buildup at the membrane surface produces an electrostatic potential across the membrane. This potential opposes diffusion of the K^+ ion across the membrane.

Panel B shows the case where a smaller amount of KCl is placed in the right side of the boundary. Again, diffusion drives some K^+ ions across the boundary, but the charge imbalance produces a counteracting potential. These two concepts are combined in panel C showing the membrane between two solutions of different KCl concentration. At equilibrium, diffusion and electrical transport balance, as described by the Nernst equation (see chapter 3 to remember this relation). For the simple electrolyte system

considered in the figure above, the activity is simply proportional to the concentration such that E_K , the equilibrium potential for potassium becomes:

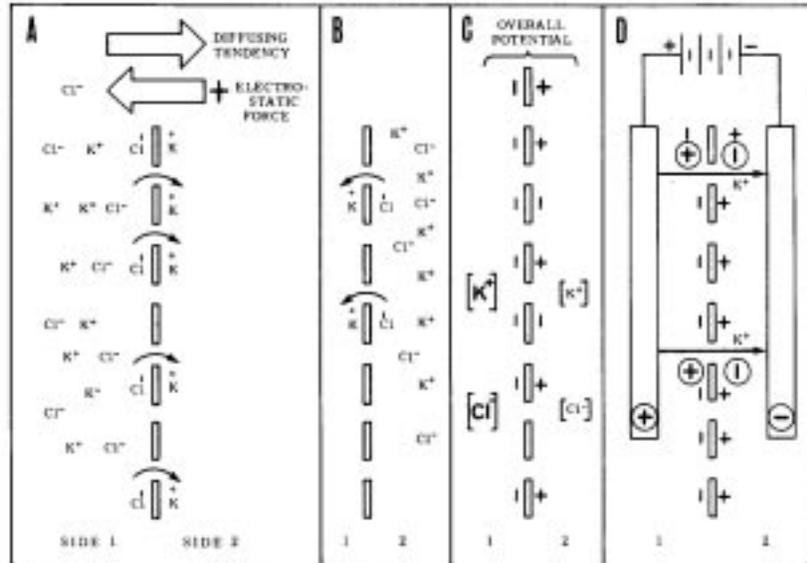


Figure 1-6
 Ion movements across semipermeable membranes. A. A relatively large amount of KCl is placed on side 1. The membrane allows only K^+ ions to diffuse, thereby establishing a voltage difference across the membrane. B. A relatively smaller amount of KCl on side 2 tends to cause a lower voltage to build up, which is opposite in polarity to the situation in A. C. If A and B are combined, an overall equilibrium potential results that will be negative on the side of the higher potassium concentration. Since this is an equilibrium condition, no net amounts of ions will flow. D. The equilibrium potential in C may be upset by altering the voltage across the membrane. In this case, net movements of ions will occur according to the manner in which the equilibrium condition is disturbed.

$$E_K = \frac{-RT}{F} \ln \frac{K_1}{K_2} = \frac{-RT}{F} \ln (10) \log_{10} \frac{K_1}{K_2}$$

where K_1 is the potassium concentration on side one, K_2 is the potassium concentration on side 2, and the ratio of thermodynamic constants, defined in Chapter 3, when multiplied by $\ln (10)$ reduces to the constant 60 mV at room temperature, i.e.,

$$E_K (\text{mV}) = -60 \log_{10} \left[\frac{K_1}{K_2} \right]$$

For example, if side 1 contains 100 mM K^+ and side 2 contains 10 mM K^+ , then the potassium potential across the membrane is:

$$E_K (\text{mV}) = -60 \log_{10} \left[\frac{100}{10} \right] = -60 \text{ mV}$$

In the body, the intracellular potassium concentration for most cells is about 130 mM and the extracellular potassium concentration is about 4 mM. This implies that if human cells were in equilibrium with respect to K^+ , a transmembrane potential of about -92 mV (inside negative, termed polarization) would exist. In fact, the true value is close to this but differs slightly because living membranes are not uniquely permeable to K^+ - they are also slightly permeable to Na^+ and Cl^- .

The transmembrane potential estimated above assumes no applied electric fields. The potential can be changed either by changing the ion concentration or by applying a field across the membrane. Consider panel D of the figure above. A potential is applied with external electrodes to produce an additional positive potential (repolarization) from side 1 to 2. Diffusion forces potassium from left to right to counterbalance this potential. As soon as the field is removed, diffusion will drive the ion concentrations back to their original values yielding the original potential difference across the membrane.

A more complicated model of ionic transport across a cell membrane is presented below. The cell is bounded by a semipermeable membrane that is highly permeable to K^+ , but is also somewhat permeable to both Na^+ and Cl^- . This means that potassium ions move through the membrane with relative ease compared with chloride and sodium ions, which permeate with comparative difficulty.

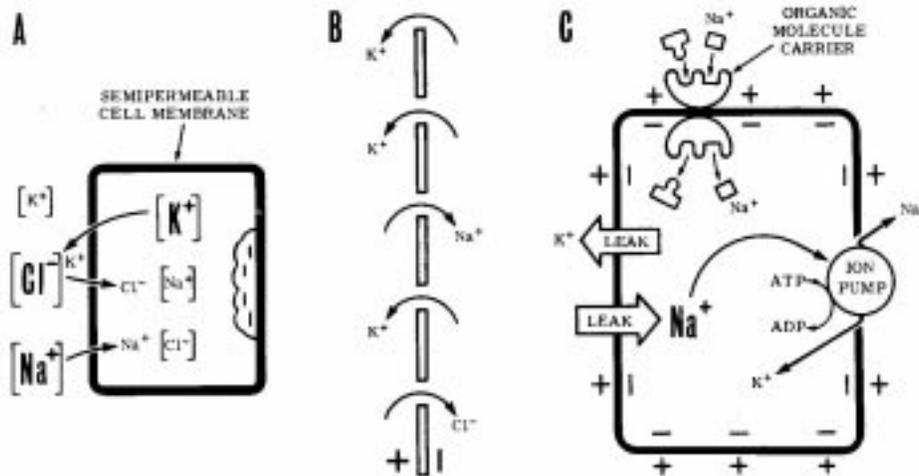


Figure 1-10 Maintenance of electrophysiologic steady state in cell system. A. Relative ion gradients across a representative cell membrane. Arrows designate diffusing tendencies of the ions. Internal protein makes up a large part of the anion inside, causing a low internal chloride concentration. "Donnan condition" is characterized by opposite nature of K^+ and Cl^- levels inside and out. B. Membrane is more permeable to K^+ than to either Na^+ or Cl^- . Thus an overall inside negative "resting potential" is set up. This potential is mainly related to potassium. C. A complete system is shown. Ions tending to "leak" (i.e., Na^+ in and K^+ out) are transported back via an energy-dependent ion pump. The pump activity is related to the level of internal Na^+ . Thus the steady state ion concentrations are maintained. This ion gradient is utilized by the organic molecule carrier for the uphill transport of glucose.

The intracellular and extracellular concentrations for these ions in a typical cell are tabulated below

Ion	Intracellular Conc. (mM)	Extracellular Conc. (mM)	E(mV)
K^+	130	4	-92
Na^+	15	140	+58
Cl^-	3	120	-96

Note that positive and negative charges aren't balanced either inside or outside the cell. Outside the cell other ions contribute to the electrolyte charge concentration. Inside the cell, immobile bound proteins with a net negative charge help balance the apparent electrolyte imbalance.

In a real membrane there are several types of ionic channels, each of which selects a specific ion, such as Na^+ or K^+ . The total transmembrane potential, therefore, must be related to the individual ionic fluxes. The solution when the sum of all flows is zero gives the Goldman-Hodgkin-Katz equation, a variant on the Nernst equation:

$$E_K = \frac{RT}{F} \ln \frac{P_{\text{Na}}[\text{Na}]_o + P_{\text{K}}[\text{K}]_o + P_{\text{Cl}}[\text{Cl}]_o}{P_{\text{Na}}[\text{Na}]_i + P_{\text{K}}[\text{K}]_i + P_{\text{Cl}}[\text{Cl}]_i}$$

where the permeabilities for the ion k is written as P_k and the concentrations of the ion are given by its chemical symbol in brackets followed by the subscript indicating the side of the membrane, with i for inside and o for outside. Thus, according to this equation, the voltage across the membrane is determined by the concentrations of all the ions and is most affected by the ion with the highest permeability. If E , as computed from this equation equals the E of the Nernst equation for one particular ion, we say that that ion is in equilibrium.

Real channels are not perfectly selective. The selectivity of ion channels is not perfect and, for example in K^+ channels, for every 20 K^+ ions that flow through the channel, one Na^+ ion can get through. This means that we cannot apply the Nernst equation to compute the potential that produces zero flow across the channel because more than one ion is involved. Instead, we could use the Goldman-Hodgkin-Katz equation (with $P_{\text{K}}/P_{\text{Na}}=20$, in the case of the K channel) and the potential predicted by the equation would be called reversal potential, instead of equilibrium potential, at which the net flow of charge through the channel is zero.

Channels are specialized membrane proteins that let ions pass through at a high rate when open. They are thought to have a specialized pathway allowing ion conduction. This pathway may be open or closed depending on the conformation of the channel molecule. Thermal motion will cycle the protein between closed and open conformations; consequently, this transition is a random event. This means it is not possible to predict at any given time whether the channel will be open or closed. The laws of probability, however, allow us to make certain predictions of the average behavior of the channel. Thus, by observing the channel operation for a long period, we can compute the total time the channel is open (O) as the sum of all the individual openings ($O = O_1+O_2+\dots$) and the total time the channel is closed (C) as the sum of the individual closed times ($C = C_1+C_2+\dots$). We can estimate the probability of being open, P_o , as $P_o = O/(O+C)$, equivalent to the fractional open time.

For membrane channels in excitable tissue, the P_o depends on the membrane potential, thus they are called voltage-dependent channels. Na and K channels in these types of tissues are voltage dependent and their open probability is low at negative (hyperpolarized) potentials and is high at positive (depolarized) potentials. The summation of a large population of these voltage dependent channels will produce a voltage dependent conductance in the cell membrane.

To understand how voltage dependent conductance changes for Na and K channels can lead to an action potential, first consider the transmembrane potentials for two hypothetical membranes having greatly different permeabilities for K and Na ions, as illustrated in the figure below. The first membrane has a relative K permeability of 0.9 and a relative Na permeability of 0.1. In contrast, the second membrane has a relative Na permeability of 0.9 and a relative K permeability of 0.1

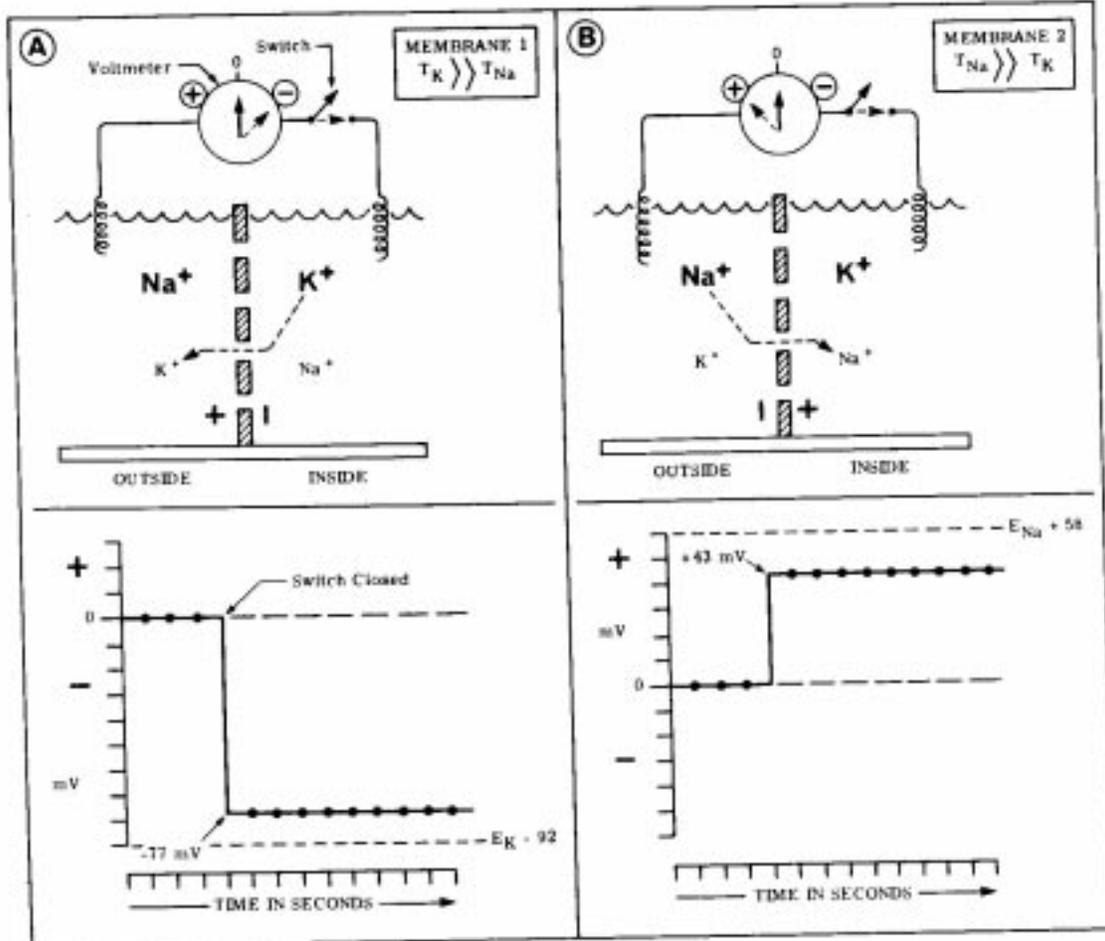


Figure 2-1
 Transmembrane potentials of hypothetical membranes with opposite ionic selectivities. A. Potassium-selective membrane shows an inside-negative potential. B. Sodium-selective membrane shows inside-positive potential.

The upper left panel shows two solutions, representing the inside and outside of the cell, separated by membrane 1. Because of its high K^+ permeability, this membrane allows K^+ ions to move more easily through the membrane than Na^+ ions. The inside-to-outside movement results in the outside-positive, inside negative electrical potential measured by the voltmeter at top. When the voltmeter is placed in the circuit, a -77 mV potential is measured.

The upper right panel shows the case for membrane 2. When the voltmeter is placed in this circuit, the transmembrane potential is dominated by the flow of Na^+ ions across the membrane, resulting in the measured +43 mV potential. The actual case for cell membranes in excitable tissue is a combination of these two cases where the permeability of the membranes to ion flows depends on the instantaneous transmembrane potential.

This phenomena is illustrated in the cartoon below, where again the left and right regions correspond to extracellular and intracellular spaces, respectively, with their corresponding ionic concentrations. The white blocks in the membrane represent

membrane one, and the cross-hatched blocks represent membrane two. In panel A, potassium selective membrane one is open while membrane two is covered. This means the voltmeter should read the resting potential for membrane 1, i.e., -77 mV.

If the opening and closing of channels can be thought of as voltage dependent gates, consider what happens when the gates are opened with a stimulus and both types of membranes contribute to the transmembrane potential, as illustrated in panel B. Applying the Goldman-Hodgkin-Katz equation for the assumed permeabilities for both membranes leads to a new transmembrane potential of -17 mV. At this potential, the voltage-dependent potassium gates close, producing the situation in panel C. The sodium gates completely control ionic flow across the membrane, so the transmembrane potential moves to the resting potential for membrane 2, i.e., +43 mV. At this positive potential the sodium gates close and potassium gates begin to open, returning the potential to its resting state.

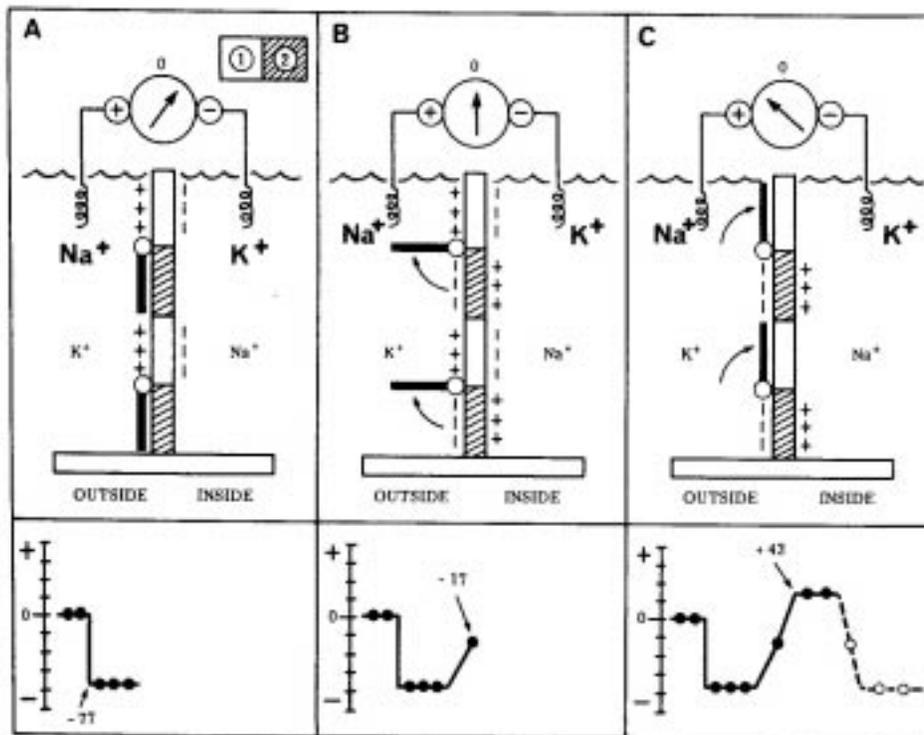


Figure 2-3
Electrical behavior of mosaic membrane in biologic fluids. Electrical events of mosaic membrane are shown as covers swing from potassium-selective to sodium-selective patches.

The timing of these events are illustrated in the next figure, where a simplified version is presented on the left and the characteristics of the true action potential for a nerve cell are shown on the right. Note the stimulus is presented to the cell at time $t=2$ msec. The events can be summarized as follows:

- 1.) A stimulus initiates sodium activation. Many sodium channels open simultaneously, producing the rapid potential increase with the peak approach E_{Na} . Sodium ions tend to move down the concentration gradient from outside to inside during this phase.

2.) Next, Na^+ inactivation and K^+ activation commence. Sodium channels close and more potassium channels open. K^+ ions tend to move down their gradient from inside to outside during this stage.

3.) Last, some of the K^+ channels close, returning to the number originally open in the resting condition. This brings the membrane potential back to the resting value.

4.) An active Na-K pump which requires energy (usually provided by ATP) reestablishes the original ion concentration.

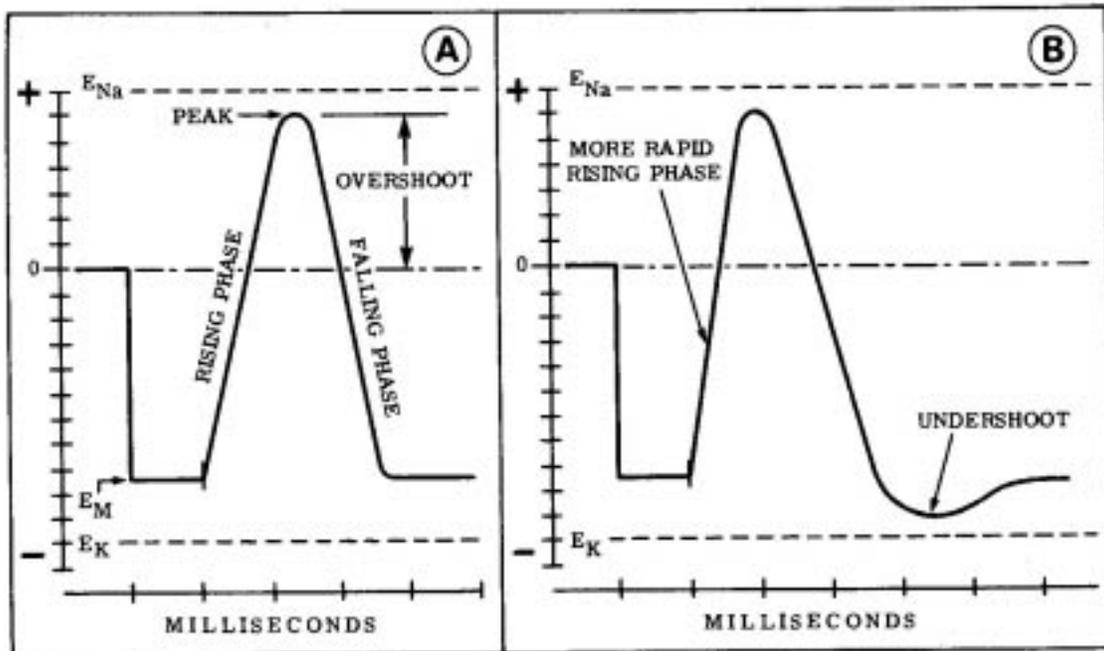


Figure 2-5
Comparison of simplified and real action potentials. (A) Simplified action potential is compared with (B) characteristics of true action potential of nerve.

Sodium and potassium channels act as voltage dependent gates on the flow of ions across the membrane. They can be simply modeled as charged, spring loaded arms, as illustrated in the figure below for a Na^+ gate. The inside potential electrostatically attracts the polar end of the gate, keeping it shut. Now consider a positive potential stimulus applied across the membrane. Some positivity is added to the membrane, slightly depolarizing the cell and allowing some Na^+ ions to flow across the membrane, as shown in panels B and C. The gate is not fully open for this potential difference. Once the stimulus is increased above a threshold, the gate fully opens allowing complete exchange of Na^+ ions across the membrane and flow of these ions in the direction of their concentration gradient. This initiates the action potential pulse and voltage dependent gate process described above. Note that an action potential is not produced for subthreshold stimulation. This is an important point that guards all excitable tissue from random activation.

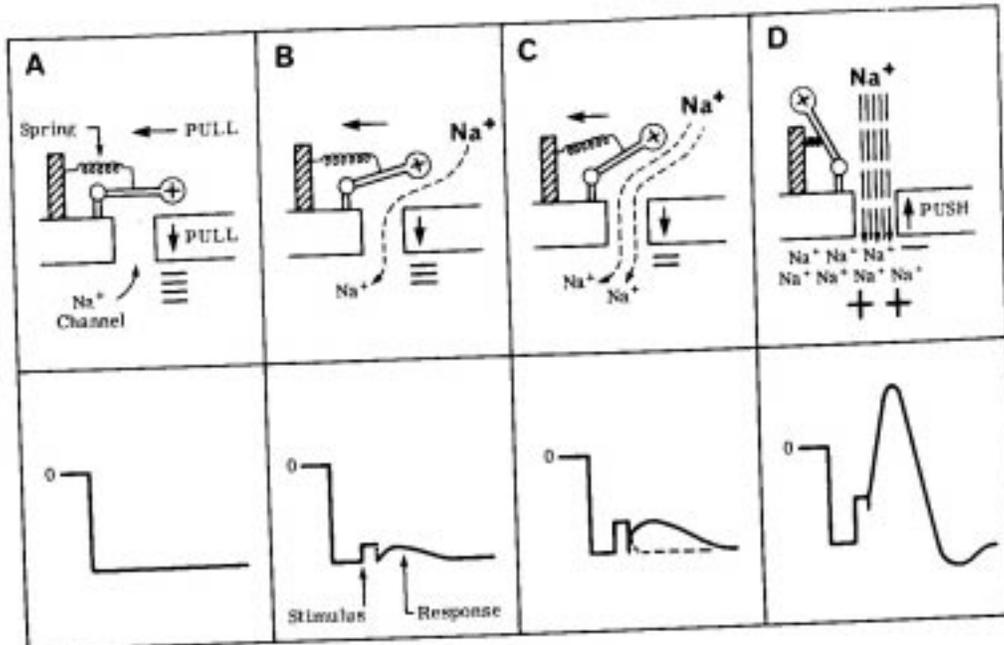


Figure 2-7
Gating process underlying the subthreshold response and the all-or-none response. A. Resting condition – gate is closed. B and C. Depolarization causes gates to open partially, allowing some Na^+ entry. D. Gate fully open at threshold, giving rise to action potential.

What happens when another above-threshold stimulus is delivered immediately following the first? The excitable tissue does not respond because the Na channels are already fully open. This lack of response is called the refractory period of the membrane, as illustrated in the figure below. The refractory timing can be understood by the position of the gates during the action potential period. This is illustrated in the lower row of this picture. Typical refractory periods are on the order of a msec in most excitable tissue.

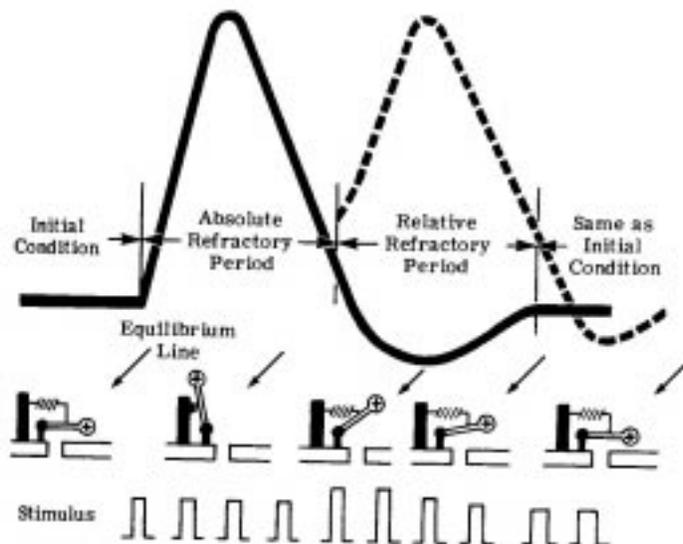


Figure 2-8
Refractory periods of the action potential and the condition of gates. Condition of the gates correlates with states of absolute and relative refractoriness in the nerve membrane.

6.2 Muscle Stimulation

The contraction of striated muscle is under neurological control - that is, electrical stimulation of excitable muscle cells to produce mechanical force is controlled by nerve-muscle junctions. The normal mode of activation is via a structure called the myoneural junction, or motor end plate. It is a special type of “synapse” between the ending of the motor nerve axon and the muscle cell membrane, as illustrated in the figure below:

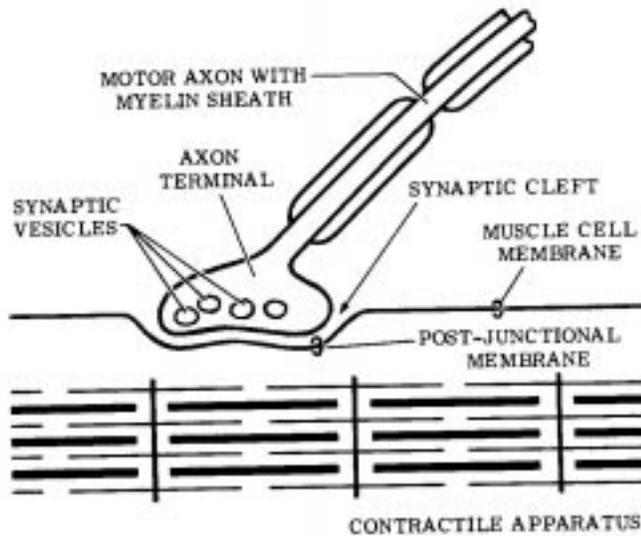


Figure 8-10
The nerve-muscle junction, or motor end plate, a specialized and simplified synapse (see Chap. 3).

The motor axon contains small synaptic vesicles housing the neurotransmitter biochemical acetylcholine (ACh). On the other side of a narrow synaptic cleft lies a specialized postjunction region of the muscle cell membrane sensitive to ACh. When a nerve action potential arrives at the motor axon terminal, the polarized presynaptic membrane is forced to release a small amount of ACh for a short period. This transmitter substance diffuses across the synaptic cleft, where it combines with a specific receptor molecule on the postjunctional membrane. The action of the ACh on the postjunctional membrane causes an increase in permeability to both potassium and sodium ions; this in turn produces a small membrane depolarization in the immediate area of the end plate called the end plate potential (EPP). When the EPP reaches a threshold, local excitatory current causes stimulation of the adjacent nonjunctional muscle membrane, and a propagating action potential travels down the length of the fiber. This process can be repeated multiple times under neurological control.

Multiple stimuli are required for continuous force from a muscle. A twitch is a single, brief muscular contraction produced by the arrival of a single nerve action potential. For a given muscle fiber under constant temperature, fiber length, and other physiologic conditions, the magnitude of the twitch is a constant, all or nothing property. If the fiber is stimulated multiple times before it has a chance to recover, as illustrated in the figure below, then the muscle produces a state of continued contraction, or tetanus. A tetanus produces more force than a twitch; the usual physiologic pattern of activation of a muscle fiber is tetanus of the appropriate duration.

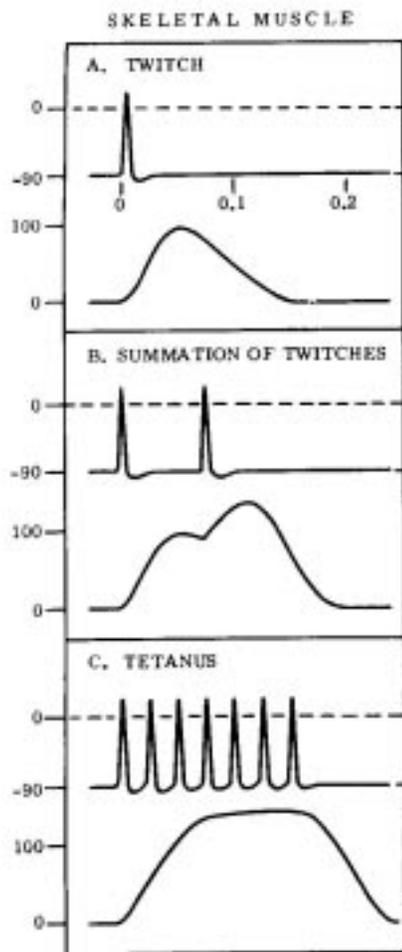


Figure 8-11
 Membrane and contractile events in skeletal muscle. A. The upper trace displays the muscle membrane potential in millivolts. The single action potential shown leads to the single contractile event, a twitch, shown in the lower trace. (The action potential trace time for all the traces is marked off in tenths of a second, and the amplitude of all of the contraction tracings are with reference to the peak twitch tension). It is important to note that the duration of the twitch is much longer than that of the action potential that triggered it. B. Because the refractory period of the muscle cell membrane is over long before the mechanical activity ceases, the muscle may be restimulated while some contractile force is still present. This leads to mechanical summation of the two twitches, and more force is produced than in a single twitch. C. If action potentials follow each other quickly enough (but not too quickly – why?), no relaxation occurs between successive twitches, and a fused tetanus results.

The motor axons innervating a muscle undergo varying degrees of branching before terminating in a number of end plates. A single axon may have terminals on many separate muscle fibers. A single axon, together with all the muscle fibers it innervates, is called a motor unit. Since activation of a muscle fiber via the motor end plate produce an all-or-nothing contraction, the size of a typical motor unit is related to the fineness of control required of the whole muscle. For muscles in which a high degree of control is exercised over fine movements, the motor axon will control only a few muscle fibers. In contrast, muscles specialized for large, rapid movements with little fine control typically have large motor units so that a single motor axon will control a large portion of the whole muscle. The ratio of muscle fibers to motor axons is called the innervation ratio.

6.3 Electromyogram (EMG)

It is possible for diagnostic or experimental purposes to observe the electrical activity of skeletal muscle in the intact body by applying biopotential recording electrodes to the skin above the muscle in question. Alternately, subsurface recordings can be obtained using needle electrodes, as illustrated in the figure below. The pattern produced by the combined action potentials of many motor units is called an electromyogram (EMG). In the rest of this chapter, and in the corresponding lab, we will only consider surface recordings with conventional biopotential electrodes applied to the skin.

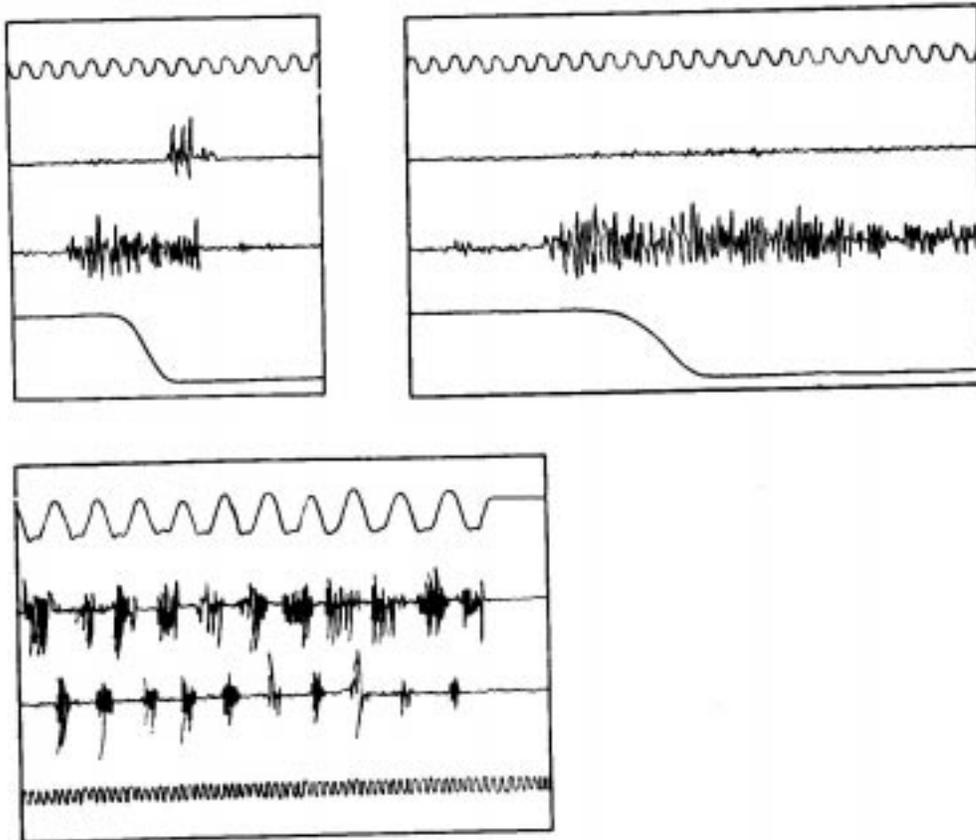


Figure 8-12

Electromyographs of human muscle. In these actual experimental records the subject had one needle electrode in the biceps (flexor) and one needle electrode in the triceps (extensor) of the upper arm. In the top two panels the four traces (reading downward) represent: time, 0.05 second per cycle; triceps muscle electrical activity; biceps muscle electrical activity; and movement of the forearm. The top left panel is from a rapid, maximal flexion of the arm with no load attached. The biceps is activated prior to, during, and following the rapid movement. At the end of the movement a burst of action potentials shows that the triceps is briefly activated to fulfill its role as antagonist to the biceps and check the movement of the forearm. In the upper right panel, the subject flexed his arm to lift a load that he continued to hold up after the movement stopped. Because the biceps was still activated, action potentials continued; but in this case, the antagonistic action of triceps was not needed, and its electrode showed that it remained inactive. The lower panel shows the antagonistic action more clearly (the positions of the timing and movement traces have been reversed; movement is now at the top). Here, the forearm was moved rapidly back and forth, and the biceps and triceps show electrical activity in an alternate fashion. The anatomic relationships between the antagonistic muscles are presented in Figure 8-1. (From D. R. Wilkie, *J. Physiol.* 110:249, 1950.)

The evoked extracellular field potential from the active fibers of a single motor unit is triphasic and of brief duration (3-15 msec) with an amplitude of about 20-2000 μV , depending on the size of the motor unit. It differs in form considerably from the classic neuron action potential presented in section 6.1. The frequency of discharge usually varies from 6 to 30 per second. The phases of this combined response are illustrated in the figure below - the frequency and amplitude range associate with the EMG is compared to other biopotential in the final figure on this page.

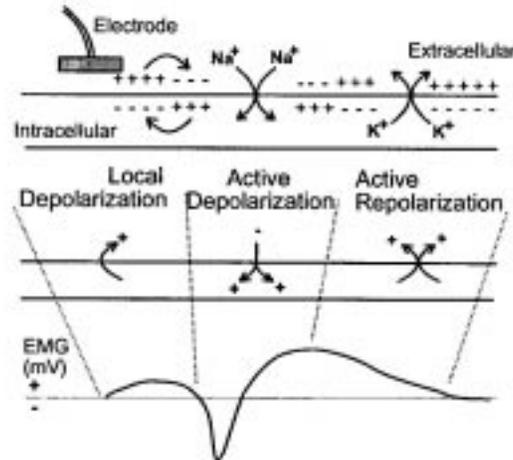


Figure 1. Ions move across the sarcolemma (top row), creating the current flow illustrated in the middle row, and measured as the extracellular potential (bottom). Notice that the electrode records the potential as it 'moves' by from right to left. (Adapted from Enoka, 1994).

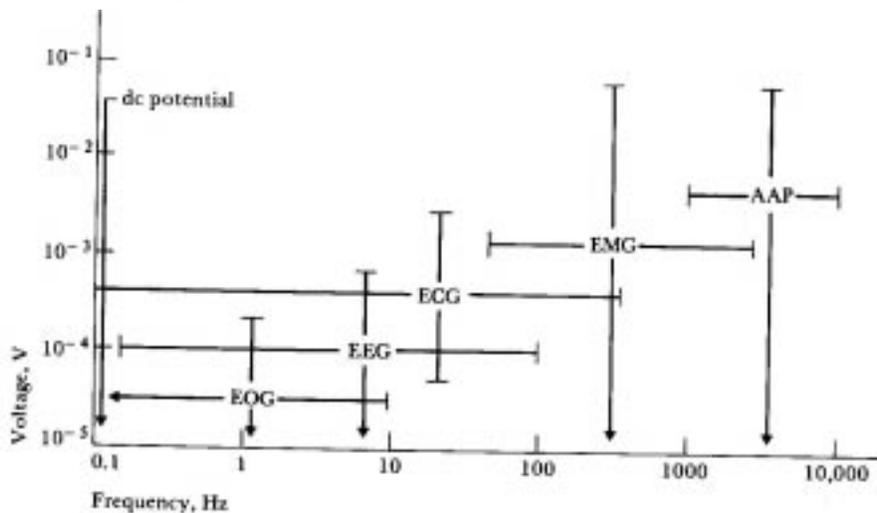


Figure 6.16 Voltage and frequency ranges of some common biopotential signals; dc potentials include intracellular voltages as well as voltages measured from several points on the body. EOG is the electrooculogram, EEG is the electroencephalogram, ECG is the electrocardiogram, EMG is the electromyogram, and AAP is the axon action potential. (From J. M. R. Delgado, "Electrodes for Extracellular Recording and Stimulation," in *Physical Techniques in Biological Research*, edited by W. L. Nastuk, New York: Academic Press, 1964.

A typical EMG recording is presented in the final figure below. This figure illustrates motor unit potentials from the normal dorsal interosseus muscle under graded levels of contraction. At high effort levels (panels c and d), many motor units are firing giving rise to the complicated response (often called an interference pattern since the timing between motor unit firings is complicated) in which individual units can no longer be distinguished. Note that as the a muscle contracts progressively under volition, active motor units increase their firing rate and new motor units are recruited.

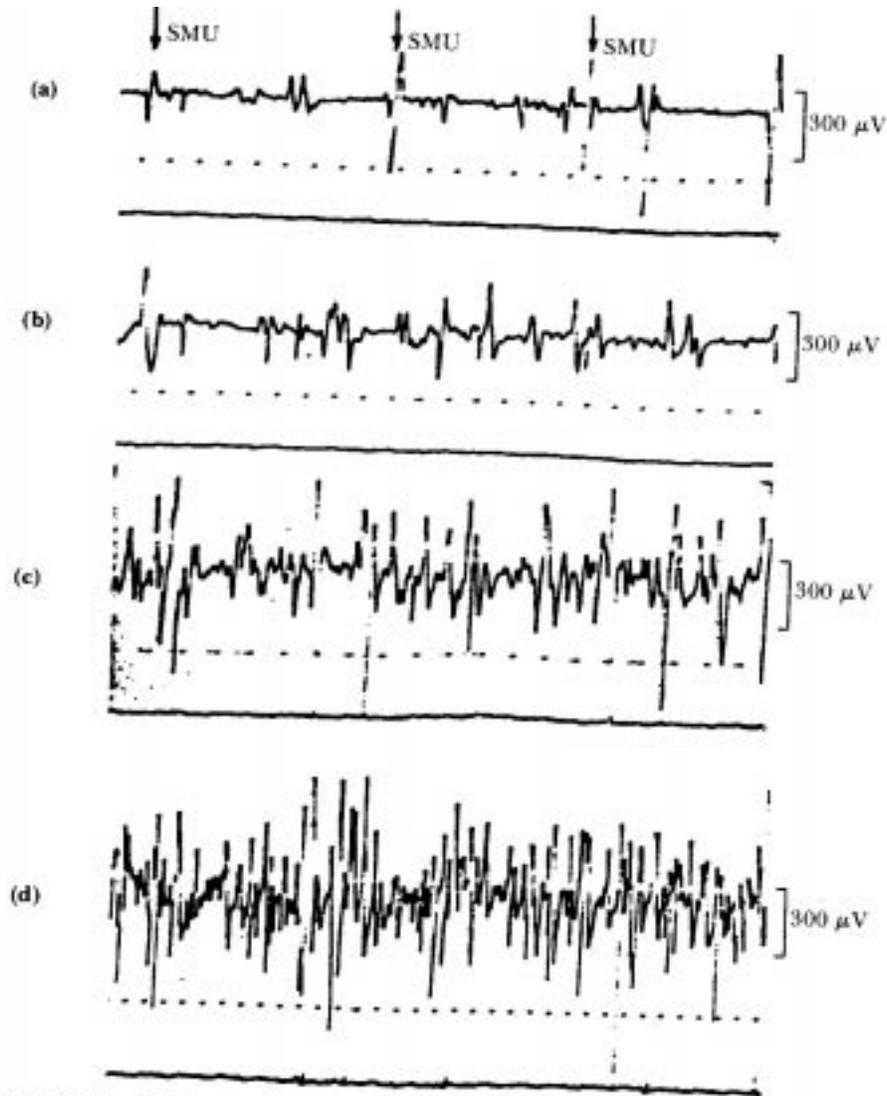


Figure 4.11 Motor unit action potentials from normal dorsal interosseus muscle during progressively more powerful contractions. In the interference pattern (c), individual units can no longer be clearly distinguished. (d) Interference pattern during very strong muscular contraction. Time scale is 10 ms per dot. (From J. A. R. Lenman and A. E. Ritchie, *Clinical Electromyography*, 2nd ed., Philadelphia: Lippincott, 1977; reproduced by permission of the authors.)