

13

Bioelectricity

This chapter is an introduction to the fascinating and complex subject of **bioelectricity**. Voltages are created by almost all types of animal cells and are largest in nerve and muscle cells. In fact, a surprisingly large fraction of the energy used by cells, and hence a large fraction of the energy requirement of the body, is needed to create and maintain **cell potentials** (voltages). At least 25% of the energy used by cells goes toward the creation and maintenance of bioelectricity.

Cell potentials may remain constant for long periods of time but can also be changed by various internal or external stimuli. Changes in cell potentials can amount to voltage pulses (nerve impulses, for example), with effects that depend on the type of cell involved. Nerve cells use these voltage pulses as communication signals for a variety of purposes, including gland control, vision, and muscle control. Nerve potentials are sometimes measured in individual nerves, indicating a specific type of nerve activity. The combined activity of many cells is also measured, such as in an *electroencephalogram* (EEG), a recording of the entire brain (billions of nerve cells) that is used as a gross indication of its activity.

Changes in cell potentials can cause muscle cells to contract. Such potentials are sometimes measured to diagnose muscle activity, most often for the heart in recordings called *electrocardiograms* (ECGs).

There are many other examples of bioelectricity. The infamous electric eel stuns its prey with electrical shocks. Some fish use electric fields to detect objects around them. Another example is that the healing of broken bones may involve electric currents.

The brief treatment of bioelectricity in this chapter concentrates on two of its major aspects. The first is the generation and transmission of bioelectricity at the cellular level. The second is the detection of bioelectricity and its use as a diagnostic indicator.

13.1 GENERATION AND TRANSMISSION OF BIOELECTRICITY

Generation; the Resting Potential

Bioelectricity is a cellular phenomenon. Although the fluids inside and outside cells are essentially neutral, there are differences in ionic concentrations that result in an electric potential across cell boundaries. Those cells that create electric potentials (most living cells in animals do) generate a thin layer of positive charge on their outside surface and a thin layer of negative charge on the inside surface of their boundary—a membrane (see Figure 13.1). Taken together, these two layers of charge are called a **dipole layer**. The two factors to be considered in explaining the creation of cell potentials are

13.1 GENERATION AND TRANSMISSION OF BIOELECTRICITY

1. the diffusion of ions across a semipermeable* membrane (the cell boundary);
2. the Coulomb force, in which like charges repel and unlike charges attract.

Differences in Ionic Concentrations and Semipermeability of the Cell Boundary There are many types of ions in the fluids inside and outside cells. The most important of these in creating cell potentials are Na^+ , K^+ , and Cl^- ions. There are large differences in the concentrations of these ions inside and outside cells, as Figure 13.2(a) shows. Negative ions other than Cl^- are designated as A^- . Note that the total charge inside and out is zero; that is, the fluids are electrically neutral.

To see how the cell potential is established, consider what happens in the example of an initially neutral cell membrane having the ionic concentration differences shown in Figure 13.2(a). The cell membrane is normally permeable to K^+ and Cl^- ions; it is about 100 times less permeable to Na^+ and is highly impermeable to other ions. Only K^+ and Cl^- diffuse across the cell membrane in significant numbers. As usual, the direction of net diffusion is from regions of high concentration to regions of low concentration. Therefore, K^+ and Cl^- ions diffuse in opposite directions, as shown in Figure 13.2(b). The K^+ and Cl^- ions have opposite charges, so there is a very strong attractive Coulomb force between them that causes them to form two thin layers of charge right on either side of the cell membrane, as seen in Figure 13.2(b).

Diffusion of K^+ and Cl^- continues until both attractive and repulsive Coulomb forces stop it. As the charge layers build up on the cell membrane, the Coulomb forces grow. The attraction of unlike charges acts to pull K^+ and Cl^- ions back to their regions of high concentration. Furthermore, the repulsion of like charges acts to keep further ions from moving out of their regions of high concentration. A balance is quickly reached between diffusion from high to low concentration and the opposing Coulomb forces. Once this balance is reached, the cell is in its **resting state**. If more K^+ and Cl^- ions diffuse across the cell membrane, the Coulomb forces grow and move some ions back. The net transfer is zero and the equilibrium is thus stable.

The Coulomb force is quite strong, and only about 1 out of 100,000 of the K^+ and Cl^- ions move across the membrane. This is such a small fraction that the overall ion concentrations are not altered. In addition, the fluids inside and outside the cell remain essentially neutral although some charge has been separated. That small separation of charge is very important, however, because it is the source of bioelec-

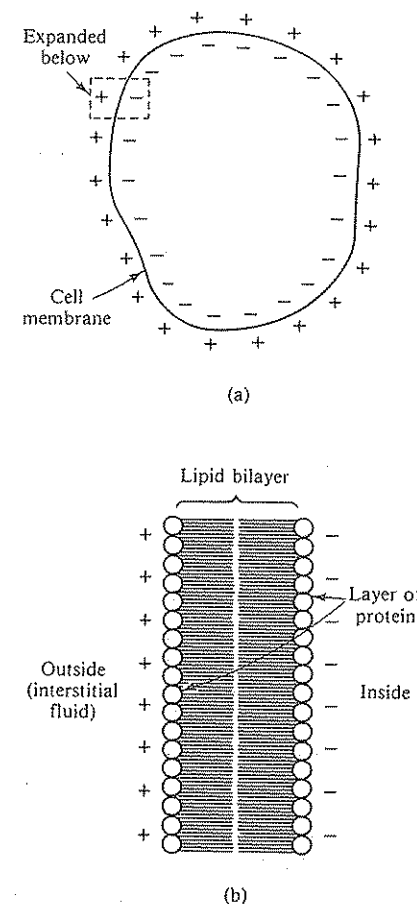


Figure 13.1 (a) Animal cell with a layer of positive charge on its exterior surface and a layer of negative charge on its interior surface. (b) Enlarged view of a section of cell membrane with its layers of positive and negative charges. The membrane itself consists of a lipid bilayer sandwiched between two layers of protein molecules. Not shown are holes through the membrane and large protein molecules that may penetrate the membrane.

*For other effects caused by the semipermeability of membranes, see Section 7.2.

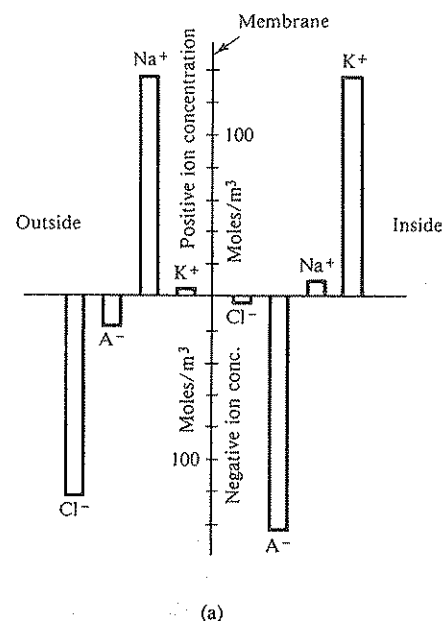


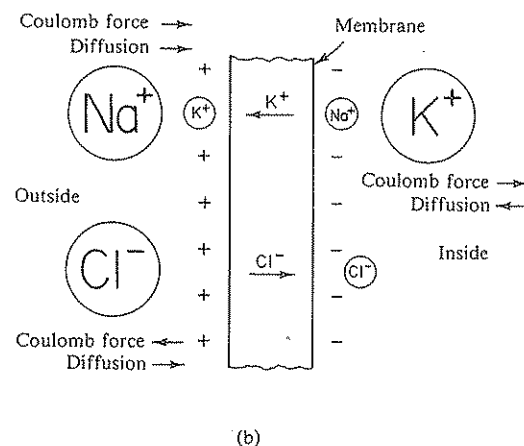
Figure 13.2 (a) Ion concentrations outside and inside a cell. Positive ion concentrations are graphed above the line and negative ion concentrations below to help illustrate that the fluids are electrically neutral. (Adapted from John R. Cameron and James G. Skolnick, *Medical Physics*, Wiley-Interscience, 1978, by permission.) **(b)** The membrane is permeable to K^+ and Cl^- ions, which diffuse in opposite directions as shown. Small arrows indicate that the Coulomb force resists the continued diffusion of both K^+ and Cl^- ions. If the membrane becomes permeable to Na^+ ions, both the diffusion gradient and the Coulomb force act to move Na^+ into the cell.

tricity. The inside of cells is at a potential 70–90 mV less than the outside, being about 90 mV in nerve and muscle cells. The outside of the cell is usually taken to be at 0 V, so the interiors of nerve and muscle cells have a *resting potential* of about –90 mV.

Quantitative Description of Potential Due to Concentration Differences; The Nernst Equation The balance between the concentration gradient and the Coulomb force is also an energy balance. Electrical potential energy just balances the potential energy due to the concentration difference—that is, the ability of the concentration gradient to do work by moving ions against the electric potential. The equation describing this energy balance is the Nernst equation. The Nernst equation gives the voltage that will be created by a concentration difference, but it does so only for a membrane that is completely permeable to one type of ion and completely impermeable to all others. Under those circumstances the Nernst equation is

$$V = V_{in} - V_{out} = -2.30 \frac{kT}{Ze} (\log C_{in} - \log C_{out}) \quad (13.1)$$

where V is the potential difference (inside minus outside), C_{in} and C_{out} are the concentrations of the ion to which the membrane is permeable, k is the Boltzmann constant, T is the absolute temperature, and Ze is the charge on the ion in multiples of electron charge (Z is the valence of



the ion). The minus sign indicates that an excess of diffusable positive ions in the neutral fluid inside a cell produces a negative voltage inside the cell. The most important feature of the Nernst equation is that the potential difference is proportional to the concentration difference.

Now, using the concentrations given in Figure 13.2(a), we can calculate what potential each type of ion would create if the membrane were permeable to it alone. For Na^+ the result is an inside potential of +59 mV. Since the actual potential in nerves is about –90 mV, this indicates that the membrane is not very permeable to Na^+ . For K^+ and Cl^- the results are –88 and –70 mV, respectively. These are close to actual values in many types of cells, but that does not necessarily imply the membranes are permeable to K^+ and Cl^- . It only implies that if the membranes are permeable to them, then there will be little movement under resting conditions.

Effect of Resting Potential on Membrane Structure The structure and characteristics of membranes are topics of intense research efforts. Much is as yet not understood, but it can be stated with certainty that the resting potential must have a major influence on membrane structure and characteristics. Although the resting potential is only 90 mV, it exists across membranes averaging only 8 nm thick. The voltage per meter is thus extremely large—on the order of 11 MV per meter. Such large voltages per meter can align molecules and have effects on pores and membrane permeability. For example, if the potential across a membrane is reversed, the permeability of the membrane changes drastically, suddenly becoming about 1000 times more permeable to Na^+ than it had been. No one understands exactly why the permeability changes, but one reason is the effect of the potential on the structure of the membrane. Nature has found a way to use such permeability changes to transmit bioelectrical signals.

Transmission of Bioelectrical Signals: The Action Potential

Depolarization and Repolarization The ability of nerve cells, also called **neurons**, to transmit bioelectrical signals is, of course, crucial to life in higher animals. When a neuron fires, it sends out a bioelectrical signal that consists of a temporary reversal in the potential across the neuron membrane. This reversal in potential starts in some localized region of the neuron and propagates along the cell membrane to other locations. The reversal in potential is called **depolarization**. The cell potential soon returns to its normal or resting polarity of negative inside and positive outside. The return to the resting state is thus called **repolarization**.

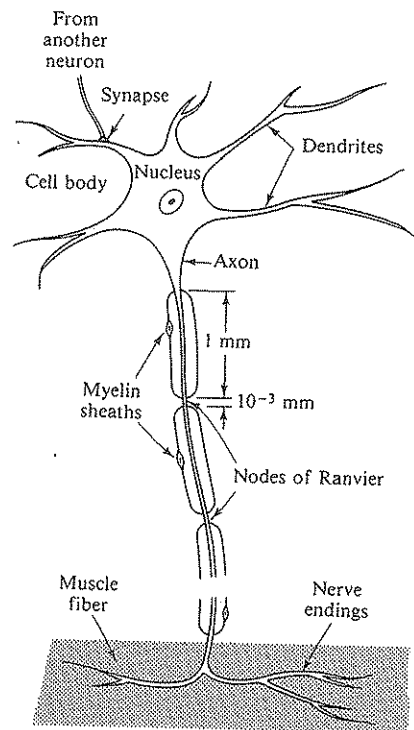


Figure 13.3 The neuron: Nerve impulses can be generated in the cell body by various stimuli. Once generated, the impulses travel down the axon, in this case stimulating a muscle.

Figure 13.3 shows the structure of a neuron. It is notable for its very long tail, called an **axon** or nerve fiber. Axons, which may be as long as 1 m, carry bioelectrical signals or nerve impulses away from the cell body to muscles, glands, or other neurons. Dendrites can also be long and tend to transmit signals from sensors inward to the body of the neuron. Several types of stimuli can trigger a neuron to fire and transmit a nerve impulse to some other location. Among these are pressure, temperature changes, electrical signals from other neurons, externally applied electric currents, and chemicals sent across junctions between neurons (called *synapses*).

The process by which a nerve fires and propagates a nerve signal can be understood in terms of the same two factors considered above to describe the resting potential of cells: namely, diffusion through a semipermeable membrane and the Coulomb force. In addition, active transport processes must be included to maintain the cell potential over long periods of time.

When a stimulus causes a nerve to fire, it does so by greatly increasing the permeability of the cell membrane to Na^+ . The cell membrane becomes about 1000 times more permeable to Na^+ than normal, making it about 10 times more permeable to Na^+ than K^+ . Because of the large concentration difference of Na^+ , a rapid diffusion of Na^+ ions into the cell now occurs. The influx of Na^+ makes the interior of the cell positive instead of negative. The potential inside the cell goes from its resting potential of -90 mV to about $+40$ mV. This is depolarization, the first step in the entire process. It is illustrated in Figure 13.4(a). The dipole layer on the membrane has essentially reversed, but only a small fraction of the ions need move to reverse the potential.

The reversal of potential across the cell membrane (depolarization) apparently alters membrane structure in such a way that two further changes in permeability take place. First, the permeability to Na^+ returns to normal (very small), and second, permeability to K^+ increases temporarily by a factor of 30. This first change cuts off further influx of Na^+ , and the second change allows a rapid outward diffusion of K^+ . The membrane potential is thus returned to its normal resting value; that is, it is repolarized. The dipole layer has been reestablished with only a small net loss in the concentration gradients driving the system. Finally, permeability to K^+ returns to normal, ending the process. These events are illustrated in Figure 13.4(a), and a graph of the permeability of the membrane to Na^+ and K^+ as a function of time is shown in Figure 13.4(b). Although the changes in permeability are known to occur as described, their exact cause is not well understood.

The change in cell potential from negative to positive and back during depolarization-repolarization amounts to a voltage pulse. This voltage pulse is the nerve impulse and is called the **action potential**. It is graphed for a nerve in Figure 13.4(a). The action potential can occur

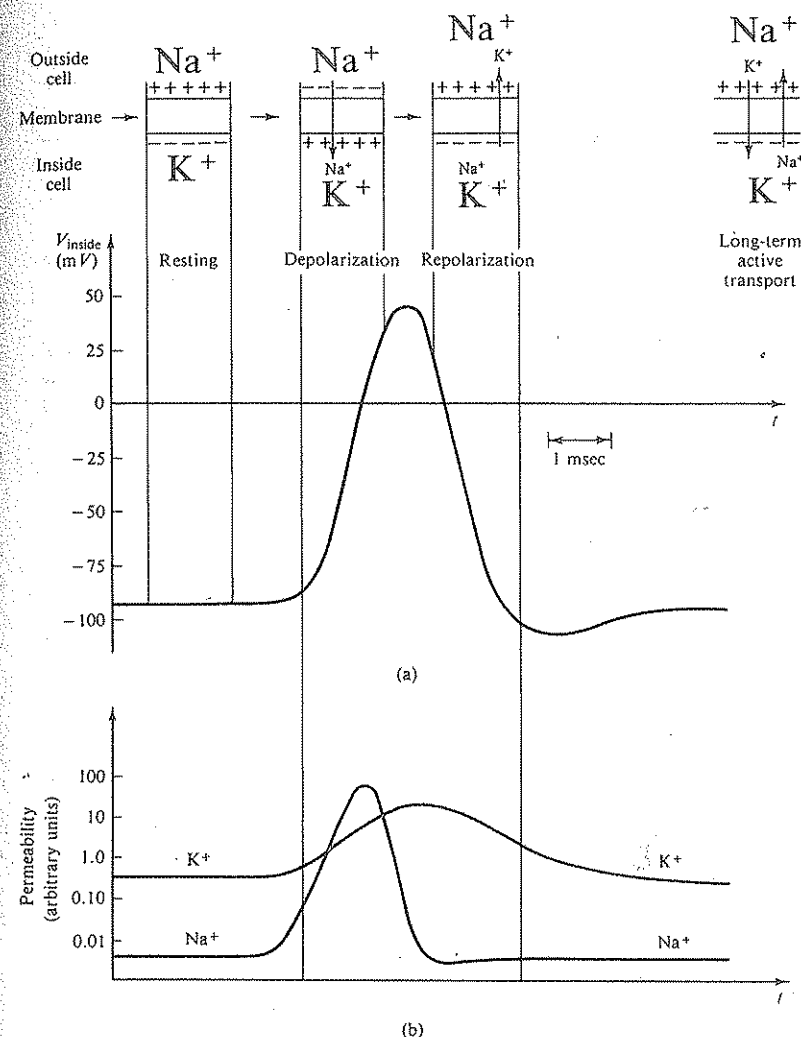


Figure 13.4 (a) Action potential at a fixed location on a nerve. Voltage inside the nerve is plotted as a function of time. Rapid movement of ions takes place only during depolarization and repolarization. Active transport is used over the long term to maintain concentration gradients. (b) Associated changes in membrane permeability during the action potential. Note the logarithmic scale for permeability.

in most animal cells, but it has some of its most interesting effects in nerve and muscle cells. Neurons can transmit the action potential to other locations, creating a variety of responses. In muscle cells the action potential causes a muscle contraction. The action potential in muscle cells, as in neurons, can originate in the cell itself or be induced by an outside source.

The Sodium-Potassium Pump Each time a cell fires there is a net loss of Na^+ and K^+ ions from their respective regions of high concen-

tration. However, it is important to note that the number of these ions crossing the cell membrane during each firing is a very small fraction of those available. Remember that only one in 100,000 of the available Na^+ and K^+ ions are needed to create the resting potential, and a similarly small fraction moves across the cell membrane during each firing. Nerves can thus fire rapidly and repeatedly (many hundreds of times) before the concentrations of Na^+ and K^+ are depleted enough to make a difference.

Over the long term the cell must find a way to move Na^+ out of its interior and K^+ back into its interior to maintain the concentration differences that create the resting potential and drive the action potential. It must utilize active transport to do this (see Section 7.2) because it is moving both Na^+ and K^+ against their concentration gradients and Na^+ against the Coulomb force (see Figure 13.2). Tracer studies have shown that the cell moves one Na^+ ion out for every K^+ ion it moves in. The active transport doing this is thus called the **sodium-potassium pump**. Active transport requires energy and a sizeable fraction of a cell's metabolism is devoted to maintaining the resting potential and driving the action potential.

Active transport is not needed to maintain the concentration gradient for Cl^- in neurons. Neuron membranes are very permeable to Cl^- and the Coulomb force moves it back and forth across the membrane during depolarization–repolarization. There is thus a small influx of Cl^- when the interior becomes positive during depolarization and a similar outflux of Cl^- when the interior returns to negative.

Propagation of the Action Potential The description of the action potential and the graphs in Figure 13.4 tell what happens at a fixed location on a cell membrane. How is the action potential transmitted to other locations? The answer is that the voltage change during depolarization of one region is sufficient to change the permeability and thereby depolarize adjacent regions of cell membrane. The depolarization–repolarization process shown in Figure 13.4 is thus stimulated in adjacent cell membrane a short time after starting at some point. These adjacent regions in turn stimulate additional cell membrane farther away, and the action potential is propagated along the cell membrane as shown in Figure 13.5. Of course, the propagation of action potentials is not limited to the cell in which they originate. Neurons can transmit them to other neurons, glands, and muscles. The brain is an extreme example of complex interconnections of neurons.

Myelinated and Unmyelinated Axons Figure 13.3 shows the axon of a nerve cell with segments covered by **myelin** sheaths and small gaps between the myelin sheaths called **nodes of Ranvier**. The axon in Figure 13.3 is myelinated; a bare axon without the sheaths is

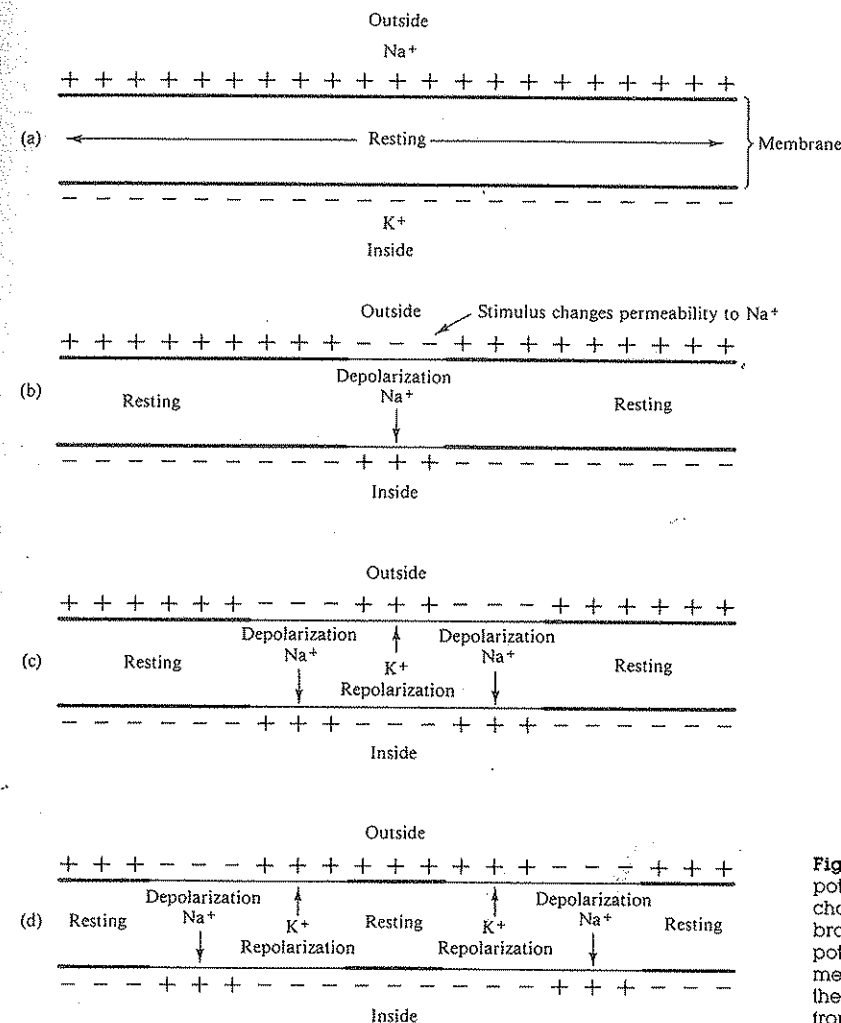


Figure 13.5 Propagation of an action potential along a membrane. A stimulus changes the permeability of the membrane to Na^+ ions, triggering the action potential. This in turn changes the permeability of adjacent membrane, and the action potential propagates outward from the starting point as shown.

called unmyelinated. Myelinated axons have certain advantages over unmyelinated axons. The myelin sheath electrically insulates the axon from being fired by other axons. This allows bundles of nerves to carry signals without “cross talk” between axons of different nerves. The speed of propagation in myelinated axons is much higher than in unmyelinated axons—as high as 130 m/sec versus as low as 0.5 m/sec in some unmyelinated axons. Furthermore, the energy required to send a signal down a myelinated axon is much less than for an unmyelinated axon. Both types of axons exist throughout the body, and it has been

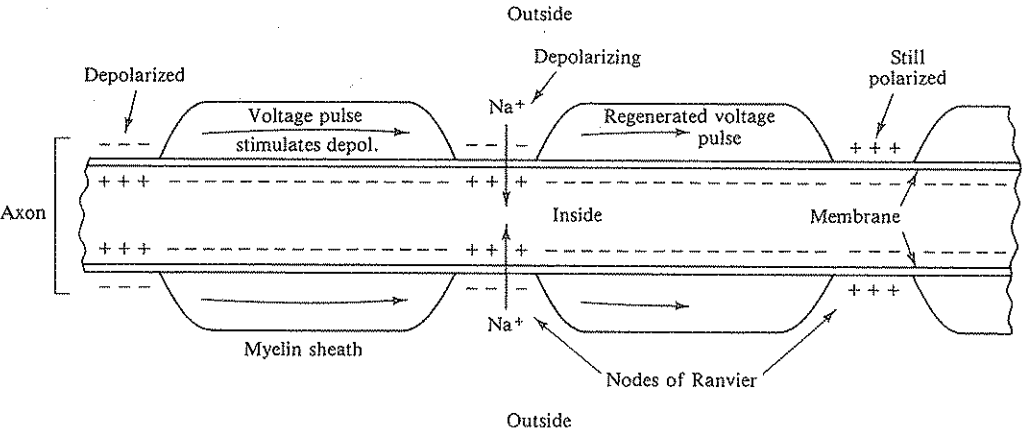
speculated that the development of the myelin sheath was an important evolutionary step.

The reason that myelinated axons conduct signals at larger speeds and using less energy has to do with the insulating properties of myelin. Consider Figure 13.6, which shows the propagation of a nerve impulse along a myelinated axon. When the nerve impulse or action potential reaches the myelin sheath, it does not cause many ions to cross the axon membrane because the myelin sheath separates the axon from the fluid outside. The action potential travels along the myelin sheath much as any voltage pulse would travel through an ordinary resistor, losing voltage as it goes along (recall that $V = IR$ is the voltage drop in a resistor) but moving very rapidly.

The voltage pulse would eventually become too small to stimulate anything at the end of the axon, so it is periodically regenerated in the nodes of Ranvier. When the voltage pulse reaches a node of Ranvier, it is still large enough to stimulate the depolarization–repolarization cycle, generating a full voltage action potential that is sent on down the axon to be regenerated at each successive gap. The nodes of Ranvier act as small amplifiers along the axon. The energy-consuming sodium–potassium pump does not have to work as hard to maintain the concentration gradients, because so few charges cross the axon membrane in the myelinated regions. The speed of propagation is larger than in unmyelinated axons because most of the axon is covered with myelin. In fact, the speed in the myelin sheaths is so large compared to the gaps that the action potential seems almost to leap from one gap to the next. Such propagation is called *saltatory* propagation.

Both the generation and propagation of bioelectricity are complex processes involving several simultaneously occurring physical phenomena, such as diffusion, Coulomb forces, and active transport. Nev-

Figure 13.6 Propagation of a nerve impulse down a myelinated axon. The nerve impulse travels very fast in the myelinated regions but loses voltage. It is regenerated in each gap.



ertheless, many of the basic properties of bioelectricity on the cellular level can be understood in terms of principles of physics. Of course, a detailed understanding requires a great deal of chemistry and biology and a dollop of more advanced physics, but some features are still mysterious.

13.2 DETECTION OF BIOELECTRICITY AND ITS USE IN MEDICAL DIAGNOSTICS

Action potentials created by *individual* cells can be measured using very small probes. This usually is done for research purposes, such as investigating the function of a particular bundle of nerve fibers. It also can be done for therapeutic purposes. For example, such measurements can help determine which nerves an artificial eye should stimulate to be most effective. Coordinated electrical activity of *entire systems* of cells, as in the brain or heart, can also be measured. Large-scale electrical activity is much easier to observe than that of individual cells and is consequently more commonly measured.

Measurement of Bioelectricity in Individual Cells

Nerve Cells Action potentials in nerve cells appear as graphed in Figure 13.4(a). To obtain such data a tiny probe is inserted into the interior of the nerve cell and another is placed in the fluid between cells. The potential difference between these probes is graphed in Figure 13.4(a), with the exterior probe taken to be at zero volts. Care must be exercised that the probes themselves do not alter the behavior of the cell they monitor. For example, one variety of probe inserted into cells is filled with a conducting solution of potassium chloride (KCl), so that ion concentrations inside the cell are not disturbed. Many early measurements of nerve potentials were made on axons removed from squids because those axons are exceptionally large. Today it is possible to measure individual nerve potentials in other animals, including humans, and it is not necessary to remove the nerves from the organism.

A variety of information has been gained by monitoring individual nerves. For example, the response of nerves in the hand has been investigated by monitoring nerve bundles carrying signals from the hand to the brain. It was found that some nerves respond to touch, rub, or pinch, others to temperature changes. Some of this information "was expected because of earlier, less direct measurements, but measurement of individual nerves provides invaluable direct evidence.

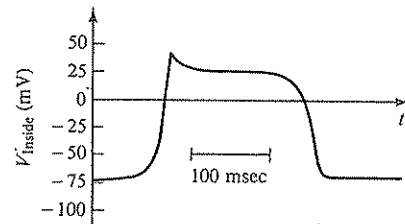


Figure 13.7 Action potential in one type of muscle cell. Note the much longer time required and the difference in shape from nerve action potentials (compare with Figure 13.4(a)).

Firing characteristics of nerves are found to differ. Some nerves fire continuously while stimulated, and others fire only when stimulation starts or stops; in some the firing rate is proportional to the strength of the stimulus. Alternatively, the probe can be used to stimulate specific nerves electrically and have the subject relate what he or she feels. And, as has been mentioned, the basic characteristics of nerve action potentials, graphed in Figure 13.4(a), were determined from observations of individual nerves.

Muscle Cells Action potentials in individual muscle cells exhibit some important differences from action potentials in nerve cells. Depending on the type of muscle cell, the action potential may last much longer than in nerve cells—from a few milliseconds to as long as several hundred milliseconds. A representative pulse for one type of heart cell is shown in Figure 13.7. Permeabilities of muscle cell membrane to various ions differ from those in nerves, and the details of action-potential generation in muscle cells are not as well known. However, one major characteristic is the same. An action potential consists of depolarization of the cell membrane followed by its repolarization.

An action potential in a muscle cell causes it to contract. Muscle action potentials may be generated inside a cell or triggered from the outside, such as by a nerve or another muscle cell. Large-scale muscle contraction sometimes occurs because there is good electrical contact among cells within a group of cells. The action potential of one muscle cell in a group can thus be transmitted from cell to cell within the group.

Measurement of Bioelectricity in Large Collections of Cells

Muscle Activity: The Electromyogram It is easy to measure electrical activity associated with muscle action by inserting probes into the muscle or by placing probes (often called *electrodes*) on the skin. This is because the coordinated action of many thousands of individual muscle cells is involved. Such a recording is called an **electromyogram** (EMG). One use of an EMG is to train a person to use diseased or injured muscles. Because the EMG is very sensitive to muscle movement, patients can determine whether they are successful in moving a muscle by watching the EMG output. Athletes are sometimes trained to develop specific muscles by using an EMG.

Electromyograms are used in a variety of research areas. Because the EMG is more sensitive than touch or vision in determining which muscles are used in a particular activity, they are useful in *kinesiol-*

ogy, the study of human motion. Electrical stimulation of muscle movement is also used in conjunction with EMGs to diagnose damaged muscles, to determine which muscles are controlled by which nerves, or to explore the tiring rate of muscles.

The Electrocardiogram The most common and most important of all measurements of bioelectricity is the recording of electrical signals from the heart, called an **electrocardiogram** (ECG). A great deal can be determined about the heart from ECGs; however, the electrical nature of the heart is complex, and its electrical signals are not simple. (A detailed treatment of ECGs can easily occupy an entire book.) Only the basics of how the ECG is measured and some of its implications are discussed here.

First, it is important to note that electrical activity can start in one part of a muscle and spread out like a wave on a pond. This is particularly true of the heart. An array of electrodes on the skin of a person can determine the timing, magnitude, and direction of motion of these electrical patterns. Figure 13.8 shows how this is done and how the addition of more electrodes gives more complete information. In Figure 13.8(a), depolarization of the muscle has started at the top of the muscle and is moving downward. When cells depolarize (the first step in the action potential), their exteriors go from positive to negative. Electrode B is therefore positive compared to electrode A; that is, the potential difference between B and A is positive. Conversely, if the potential difference between B and A is positive, then the depolarization is moving toward B.

The exact direction of the electrical wave is not determined by two electrodes. Only the component of the arrow representing the magnitude and direction of the electrical wave (a vector) is determined. Adding a third electrode, as in Figure 13.8(b), helps because the components of the vector along three directions are observed. Vector addition is used to gain further information. However, three points determine a plane and the muscle is three-dimensional, so more electrodes are necessary to obtain complete information.

Standard ECGs are measured using a triangular configuration of electrodes like that in Figure 13.8(b). This configuration is called the **Einthoven triangle** after Willem Einthoven, a Dutch physiologist who pioneered its use at the beginning of this century. The electrodes sometimes are placed on the right and left arms and the left leg and are traditionally labeled RA, LA, and LL. The potential differences between the three pairs of electrodes are traditionally labeled I, II, and III, as shown in the figure. All three are usually recorded, and the measurements are often called leads I, II, and III. (Each *lead* is the potential difference between two electrodes.) It is now common in a detailed ECG to have six additional electrodes attached across the

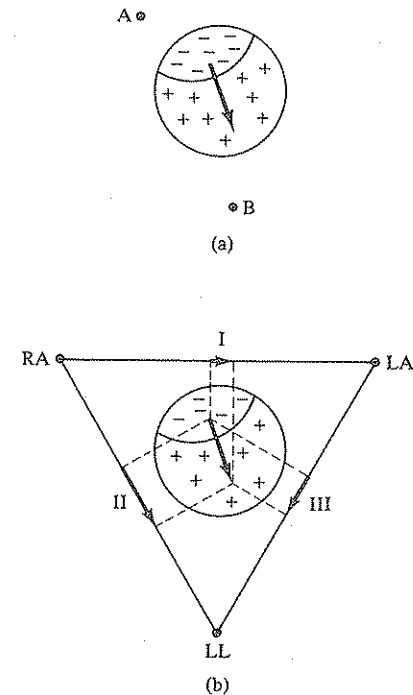


Figure 13.8(a) Wave of depolarization spreading across the heart is represented by an arrow pointing in the direction of motion of the wave and having a length proportional to the strength of the wave (a vector). An electrode placed at B will be positive compared with an electrode at A. (b) The Einthoven triangle configuration of electrodes for measuring electrical signals from the heart in an ECG. Each pair of electrodes measures one component of the voltage wave.

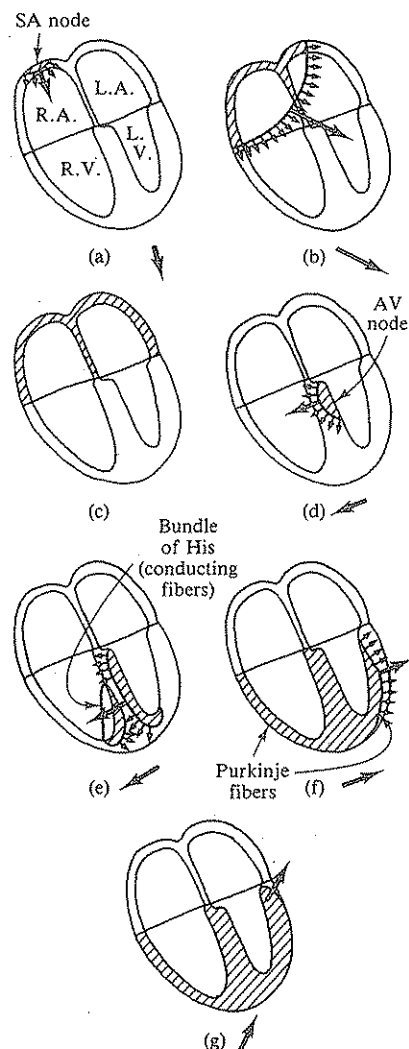


Figure 13.9 In (a)–(c), depolarization begins in the SA node and sweeps across the atria, producing the feature labeled P in Figure 13.10. (d) The AV node is triggered. (e) Depolarization is transmitted down the middle of the heart. (f), (g) Depolarization moves up the sides and back of the heart. Depolarization of the ventricles in (d)–(g) produces the feature labeled QRS in Figure 13.10. (Adapted from Hobbie, *Intermed. Phys. for Med. and Biol.*, Wiley, 1978, by permission.)

chest of the subject to give three-dimensional information. Typically, skin potentials on the order of 1 mV are observed.

Figure 13.9 shows how electrical patterns spread out on the heart. Each heartbeat starts with the right and left atria pumping blood into the right and left ventricles. The ventricles then pump blood through the lungs and the rest of the body. The electrical signal that starts this sequence of events originates in an area of the upper left part of the heart called the *sinoatrial* or **SA node**. This wave travels across the atria, depolarizing muscle cells and causing the atria to contract. See Figure 13.9(a)–(c).

When this depolarization wave reaches the bottom of the atria, it stimulates an area called the *atrioventricular* or **AV node**. The AV node then fires and begins the depolarization of the bundle of His, which carry the depolarization to the Purkinje fibers, a special conducting system of muscle fibers. These muscle fibers have lost the ability to contract and act as a rapid conduction system, bringing depolarization to all parts of the ventricles nearly simultaneously and causing their contraction [see Figures 13.9(d)–13.9(g)].

After the heart muscle has depolarized and contracted, it repolarizes and is then ready for the next beat. The SA node is the natural pacemaker for the heart and will cause it to beat about 72 times a minute without outside stimulation. However, there are nerves leading into the SA node that can adjust the heart rate in response to bodily needs such as during exercise, anxiety, or sleep.

Figure 13.10 shows an ECG trace for lead II (the potential difference between the left leg and right arm) and a graph of the corresponding arterial blood pressure. The major features of the ECG are labeled P, Q, R, S, and T. The **P wave** corresponds to the depolarization and contraction of the atria. The **QRS complex** accompanies the depolarization and contraction of the ventricles. The **T wave** is the repolarization of the ventricles to prepare them for the next contraction. Repolarization of the atria occurs earlier and is not observed because it is masked by the large QRS complex.

Traces from other pairs of electrodes differ from the lead II trace shown, because they get different components of the electrical waves moving across the heart. Taken together, the several traces (three to twelve) of an ECG can give very detailed information on the functioning of the heart. For example, dead regions of the heart, called *infarcts*, reflect the electrical waves. In an ECG such reflections can be observed, and the affected parts of the heart sometimes can be identified.

An **AV block** can also be observed in an ECG. In an AV block the depolarization wave from the atria is blocked by dead or scarred tissue from reaching the AV node. In terms of the ECG this means the P wave has trouble triggering the QRS complex. This can appear as an

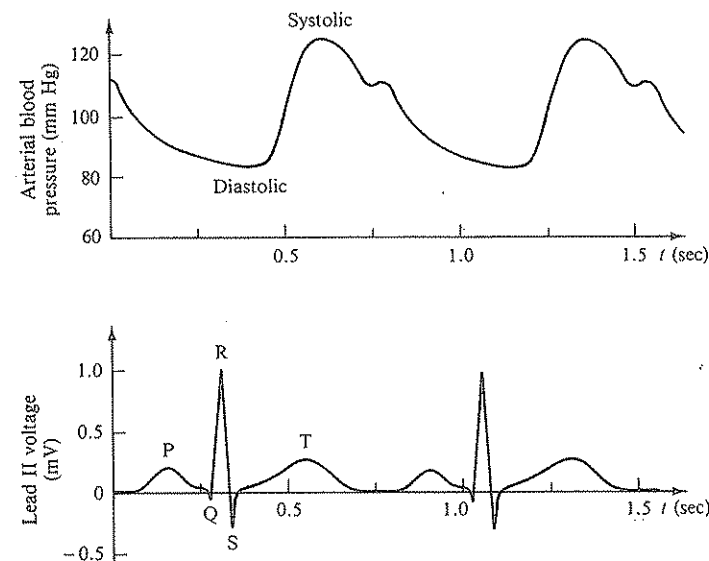
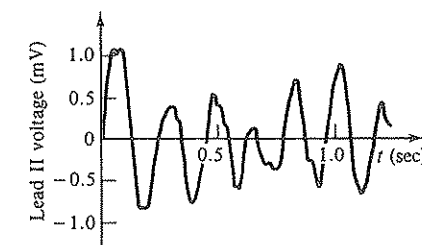


Figure 13.10 An ECG with corresponding blood pressure. The P wave is the depolarization of the atria. The QRS complex is the depolarization of the ventricles, followed shortly by ventricular contraction as indicated by the subsequent maximum in blood pressure. The T wave is the repolarization of the ventricles to ready them for the next beat. See also Figure 7.14.

abnormally long time between the P wave and the QRS complex. A severe AV block prevents triggering of the ventricles by the electrical wave from the atria. The AV node then takes over, and acts as its own pacemaker, but the heart rate becomes unacceptably low. The remedy is the implantation of an artificial pacemaker. The artificial pacemaker has an electrical connection to the ventricles and stimulates them to contract at an acceptable rate. Artificial pacemakers are rather common and enable many people to lead nearly normal lives instead of being greatly incapacitated.

Another heart problem easily diagnosed with an ECG is ventricular fibrillation, a malfunctioning of the signals from the AV node to the ventricles that causes the ventricles to contract chaotically and ineffectively. It is fatal within minutes if uncorrected, so ECGs are useful in detecting ventricular fibrillation only when a patient is already being monitored, as during surgery or in intensive care. An ECG taken during ventricular fibrillation, such as that in Figure 13.11, shows complete distortion of normal P, QRS, and T features. The remedy is electrical defibrillation by passing a large current through the heart. This apparently completely depolarizes the heart muscle, so that the heart is often able to restore its normal electrical patterns. Defibrillators are now constructed with microcomputers that initiate defibrillation at the time of a QRS, if any are observable. It is also interesting that some artificial pacemakers have built-in defibrillation capabilities. These pacemakers use a microcomputer to recognize when the heart is fibrillating and to administer the defibrillating shock.

Figure 13.11 An ECG taken during ventricular fibrillation, showing complete distortion of the normal heart signals. The victim is unconscious and without a pulse.



The interested reader is referred to the voluminous literature on ECGs. A few such references appear at the end of this chapter.

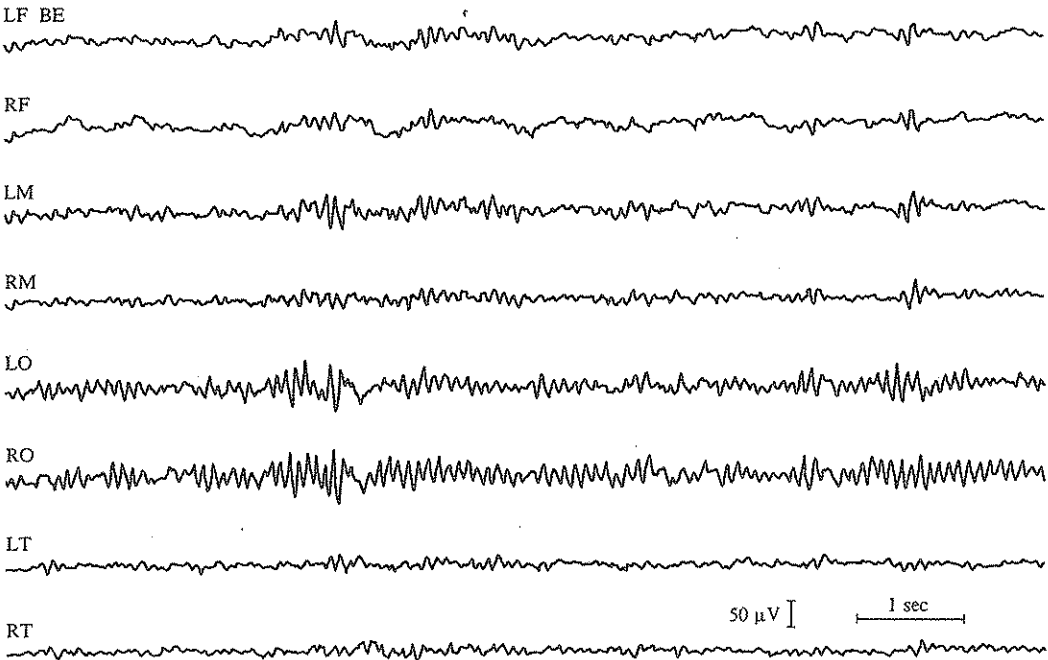
Brain Activity: The Electroencephalogram Electrodes placed on the scalp can detect complex electrical patterns associated with nerve activity in the cortex of the brain. The recording of these potentials is an **electroencephalogram** (EEG) and is a valuable tool both in research and in medical diagnostics.

More than twenty electrodes are placed on the scalp to record a detailed EEG. Potential differences between various pairs of electrodes are recorded, and a reference electrode is attached to one ear. Signals from the left and right sides of the brain are often compared because asymetry in brain activity is frequently an indication of brain disease.

Voltages of about 50 μV are typically observed on the scalp. These are small compared to the millivolt potentials found on the skin associated with muscle movement. Electrical signals from eye movements, heart activity, and noise from electrical devices in the room are problematic but can be overcome. Figure 13.12 shows a typical EEG for a normal subject.

Electroencephalogram traces are obviously more complex and appear to be less regular than ECG traces. This is not surprising, to say

Figure 13.12 An EEG for a normal patient. The letters on the left identify the locations of electrodes. The voltage and time scales are given in the lower right. (From K. Kooi, R. Tucker, and R. Marshall, *Fundamentals of Electroencephalography*, 2nd ed. Harper & Row, 1978, by permission.)



the least, since hundreds of millions of neurons are involved and their activity is neither as simple nor as coordinated as that of heart muscle cells. Analysis of EEG traces shows that there are certain basic frequencies present in the electrical signals. The presence or absence of certain frequencies is strongly correlated with certain types of brain activity. Consequently, frequency ranges are given the names listed in Table 13.1. A skilled observer can detect the presence of the various **brain waves**, as they are called, in an EEG. The advent of computers has made that task somewhat easier.

Mental alertness is one thing that can be detected with an EEG. *Beta waves* become dominant during concentrated mental activity. *Alpha waves* decrease and disappear as a person falls asleep and sometimes are monitored to determine the depth of anesthesia during surgery. *Delta waves* are indicative of deep sleep and become dominant for a person in a coma. Lack of brain activity as indicated by an EEG is sometimes used as a criterion for determining when a person is dead.

Certain brain disorders, such as epilepsy, and diseases that affect brain function can be detected with an EEG. The presence of brain tumors can be determined because there is less brain activity in the region of the tumor. In fact, any disease that affects blood flow or glucose supply to the brain, as do sickle-cell anemia and hypoglycemia, may cause an abnormal EEG. The presence of certain psychoactive drugs also causes an abnormal EEG.

The correlation between EEG and specific brain activities or malfunctions is not perfect. For example, a significant percentage of epileptics, depending on the type of seizure they suffer, have normal EEGs between seizures. EEG technology is still in the developmental stage, partly because the brain itself is so poorly understood and partly because so many hundreds of millions of neurons are involved.

TABLE 13.1 / BRAIN WAVE FREQUENCY RANGES

Band	Frequency Range (Hz)
Delta (δ)	0.5–4
Theta (θ)	4–8
Alpha (α)	8–13
Beta (β)	13–22
Gamma (γ)	22–30

REVIEW: CONCEPTS AND EQUATIONS

- Bioelectricity

Cell Potential

Cell Membrane Permeability

Dipole Layer

Resting State

Action Potential

Depolarization

Repolarization

Sodium–Potassium Pump

Neurons
- Axons

Myelin

Nodes of Ranvier

Electromyogram

Electrocardiogram

Einthoven Triangle

P, QRS, T Waves

SA Node, AV Node

Electroencephalogram

Brain Waves

$$V = V_{in} - V_{out} = -2.30 \frac{kT}{Ze} (\log C_{in} - \log C_{out}) \quad (13.1)$$

QUESTIONS

- 13.1. Are the concentrations of the following ions greatest inside or outside of cells: Na^+ , K^+ , Cl^- ? What process is used to maintain the concentration gradients of Na^+ and K^+ ions over long periods of time?
- 13.2. In what way is energy expended to create bioelectricity?
- 13.3. Note that in Figure 13.2(b) both the concentration gradient and Coulomb force tend to move Na^+ ions into the cell. What prevents significant amounts of Na^+ from moving into the cell?
- 13.4. If the cell membrane is permeable to both K^+ and Cl^- ions, why don't they diffuse across the membrane until their concentrations are equal on either side?
- 13.5. How can a cell (and the fluid around it) be electrically neutral and still create a potential difference across the cell membrane?
- 13.6. Explain why a large concentration of K^+ ions inside a cell causes it to become negative inside.
- 13.7. Define *depolarization*, *repolarization*, and *action potential*.
- 13.8. Describe the sequence of events occurring in an action potential in terms of diffusion, the Coulomb force, and membrane permeability.
- 13.9. Why can a cell fire many times before needing active transport to maintain K^+ and Na^+ concentration gradients?
- 13.10. Explain why there is a minimum time interval between firings of a cell. Does that minimum time depend on the type of cell—such as nerve or muscle?
- 13.11. Estimate the maximum firing rates for neurons and for heart cells. *Hint*: Consider the graphs of their action potentials.
- 13.12. What is the relationship between the maximum firing rate of nerves and the hearing mechanism as discussed in Chapter 9? Similarly, what is the relationship between the maximum firing rate of heart cells and the maximum heart rate?

- 13.13. Note in Figure 13.4(a) that the potential in a nerve cell overshoots a bit when it returns to its resting potential, briefly becoming -110 mV. Explain this overshoot in terms of the increased permeability of the membrane to K^+ ions graphed in Figure 13.4(b).
- 13.14. Draw a cell membrane as it would appear if the sequence shown in Figure 13.5 is carried one step further.
- 13.15. What is the function of the nodes of Ranvier, and why are these gaps between myelin sheaths necessary?
- 13.16. Explain the advantages of myelinated nerves in terms of the insulating properties of myelin.
- 13.17. Give an example of an action potential that originates inside a cell. Give another example in which an action potential is transmitted to another cell, causing it to fire.
- 13.18. How can the direction in which a depolarization wave moves across a muscle be measured?
- 13.19. Why are more than three electrodes needed in an ECG to obtain three-dimensional information about the heart?
- 13.20. Explain in terms of efficient use of a muscle why simultaneous depolarization of all parts of the ventricles is desirable.
- 13.21. Depolarization of a muscle cell causes it to contract a few tenths of a second later. Is this consistent with Figure 13.10? Explain.
- 13.22. What physical quantities (e.g., current, voltage, resistance, velocity, time, mass, etc.) are actually measured in EMGs, ECGs, and EEGs?
- 13.23. Which yield more specific types of information, ECGs or EEGs? Explain why.
- 13.24. Why do altered blood flow and blood glucose levels affect EEGs?
- 13.25. What is the physical distinction between the various types of brain waves (alpha, beta, etc.) measured in EEGs?

problems is to verify the potentials given for K^+ , Na^+ , and Cl^- ions in the text. The value of Boltzmann's constant is $k = 1.38 \times 10^{-23}$ J/K. A temperature of 310K (37°C) is assumed.

PROBLEMS

The Nernst equation (equation 13.1) gives the membrane potential that would result from the concentration gradient of a single type of ion in the presence of a membrane perfectly permeable to it. The purpose of the following three

- 13.1. (II) Show that the membrane potential for K^+ ions is -88 mV, given the concentrations of K^+ ions to be 140 mol/m^3 inside the cell and 5 mol/m^3 outside.
- 13.2. (II) Show that the membrane potential for Na^+ ions is $+59$ mV, given the concentrations of Na^+ ions

to be 15 mol/m^3 inside the cell and 140 mol/m^3 outside.

- 13.3. (II) Show that the membrane potential for Cl^- ions is -70 mV, given the concentrations of Cl^- ions to be 9 mol/m^3 inside the cell and 125 mol/m^3 outside.

REFERENCES AND ADDITIONAL READING

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