Textbook of Electrocardiography

DAVID LITTMANN

TEXTBOOK OF
ELECTROCARDIOGRAPHY

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PREFACE

Electrocardiography, from its earliest beginnings, has been an empiric, never completely objective discipline. Modern practice and improved techniques notwithstanding, much remains that cannot be measured or described and must be interpreted. And, fortunately or not, depending on the point of view, it is still impossible to digitize and computerize all the subtleties of the electrocardiogram. Electrocardiographic theory, too, in some areas remains uncertain and speculative. Nevertheless, valuable, even precise diagnoses are being made by means of empiric observation and pattern recognition. This includes the practice of vectorcardiography, which is even more dependent on pattern identification than classic electrocardiography.

In its present state, clinical electrocardiography must in large measure be taught by example and the recorded electrocardiogram. Accordingly, in order to minimize personal bias as well as obscurity, meticulous care has been used to select the illustrations for this book. Only those records which are above technical criticism have been chosen—those which illustrate beyond any reasonable doubt the disorders they are meant to represent; in many instances the diagnoses are supported by laboratory studies, physical examination, x-ray studies, and autopsy findings. This material, collected during more than 25 years of hospital practice, contains examples of most normal and abnormal electrocardiograms. All the "textbook" examples are shown, plus a sufficient number of more usual though less classic forms to approximate the range of material as it comes from the laboratory. All else in this book is secondary to the illustrations; only enough text is offered to provide adequate introduction, coherence, and continuity.

Most of the electrocardiograms used to illustrate this book were made on photographic recorders. This was done to preserve minute details and subtleties possible with this method but sometimes obscured by hot-stylus instruments. Direct-writer electrocardiograms were used only when photographic tracings could

not be obtained. The photographic electrocardiograms, which normally display a white trace on a black background, have been reversed to show a black trace on a white background in order to conform with the current, almost universal, directly written electrocardiograms and to improve their readability. Faint QRS complexes which in reproduction became still fainter or actually lost were intensified (retouched) before printing.

I confess at the outset my extensive use of imprecise qualifying terms like *often, frequent, common, occasional, rare, exceptional,* and the like. I do not concede, however, that they are inferior to percentages or other numerical expressions merely because the latter can be so positively defined; I firmly resist the seduction of numbers in an area where quantitation is beset by such uncertainties. Observations on the electrophysiology of individual cells and the order of ventricular depolarization, although derived from animal studies and often incompletely understood, are applied to the clinical electrocardiogram in this book. There is little evidence that these are valid extrapolations, but they are made in the interest of simplicity and practical gain. Such instances are identified as teaching devices or simplifications.

Theoretic limitations notwithstanding, electrocardiography has so expanded that some component subject areas have attained book size. This is true of electrophysiology, pediatric electrocardiography, and disturbances of rhythm. It is not my intention to consider these areas in extensive detail. Each, however, is presented in sufficient degree for adequate continuity and for most ordinary purposes. Vectorcardiography is described and briefly discussed, but applied in only the most limited degree.

Finally, the electrocardiogram is a laboratory test, albeit a special one from which diagnostic inferences are drawn. It has been suggested that the electrocardiogram, like many nonspecific tests, should be interpreted only in its clinical context and in the presence of all "pertinent" information. I disagree with this point of view and feel that an electrocardiogram should be interpreted for itself, like a bone marrow or liver biopsy, and, to prevent bias, without knowledge of its source. Only after the initial judgment (which must be recorded) should it be compared with previous records and considered in its clinical setting. It has long been my practice, for myself as well as for my house staff, to interpret each electrocardiogram by and for itself as a pure exercise, before noting the name of the patient and any clinical diagnoses and before comparing it with prior tracings. This has led to a growth of intellectual honesty as well as to the detection of cardiac and metabolic disorders that had not been suspected. It has also dispelled many important but erroneous diagnoses that had been made on purely clinical grounds. The illustrations shown with many cardiac and metabolic disorders are not offered as the sole electrocardiographic findings of those disorders. In many instances it is not even clear that the findings result from the diseases they accompany. They are shown, however, as examples of what may be found without excluding other possibilities.

Much of the material in this book as well as its manner of presentation has been successfully used to train a whole generation of medical students and residents. Judging from its reception in the past, it can be expected to have general

appeal to the medical student and particularly to the resident in internal medicine. The illustrations are sufficiently inclusive to serve as an atlas of electrocardiography for the cardiologist and internist.

West Roxbury, Massachusetts

D.L.

CONTENTS

CHAPTER 1

Electrophysiology: The Normal Electrocardiogram

ELECTROPHYSIOLOGY OF THE HEART

THE RECORDER

The recorder, or electrocardiograph machine, resembles a radio receiver that detects and amplifies vastly attenuated signals broadcast from a remote transmitter. Despite the transmitter's enormous output, the energy dwindles to a minute fraction of its original value by the time it is intercepted at a distance. Similarly, electrocardiograms (ECGs) recorded from large animals like elephants or whales, despite their large hearts and proportionately large electrical emissions, are disappointingly small. Like the radio station, the heart diffuses its electrical output into a surrounding volume (three-dimensional) conductor. The electrical emissions are radiated in concentric, isopotential spheres, with the energy level, as measured along a radius, diminishing inversely with the square of the distance. In its healthy state, the human body is a reasonably homogeneous electrical conductor despite its heteromorphic and discontinuous composition. Accordingly, conduction of electrical events away from the heart may be thought of as proceeding along relatively straight

lines. Were the heart a symmetrical muscular sphere, simultaneously and equally depolarized from its center, almost complete cancellation would ensue, and an electrode would intercept only a minute force simply because it is closer to one surface of the heart than to the remainder. It is the anatomic asymmetry and asynchronous depolarization of the heart that make recording of the ECG possible.

THE CELL

The intracellular ionic potential (electrical pressure) of a myocardial fiber is electrically negative in relation to the exterior and of surprising magnitude. In sharp contrast to body surface levels of 1 to 3 mv generated by the whole heart, transmembrane potentials of −80 to −90 mv are regularly observed by means of paired intra- and extracellular microelectrodes. It is apparent that the relatively large electrical forces of the myocardium must be cancelled or otherwise dissipated within the heart, its contents, or the surrounding tissues before they attain the body surface.

ACTIVATION OF THE CELL

The description which follows is a simplified account of electrophysiologic activities derived from observations on a variety of excitable cells. It is believed to be in reasonable accord with the actual events. The live cell existing in an environment of 140 mEq/L of sodium and 5 mEq/L of potassium contains cytoplasm with levels of 5 to 10 mEq of sodium and 130 to 150 mEq of potassium (Fig. 1-1). This separation of electrolytes is achieved by the expenditure of metabolic energy and maintained by the integrity of the cell membrane. The fully polarized cell is pictured as being lined by a series of electrical couplets, or dipoles, with the negative charges on the inside and the positive charges on the outside of its semipermeable membrane, which displays electrical capacity as well as resistance to the flow of electrical charges. The membrane is believed to consist of a thin layer of lipid sandwiched between two protein films and is normally impervious to penetration by water solutes except through widely separated intermolecular pores that are somewhat smaller than sodium ions and slightly larger than potassium and chloride ions. The partition of electrolyte on the two sides of the membrane depends, in large measure, on the differential resistance of the membrane to the passage of ions. The observed intracellular voltage (electrical pressure) of the fully polarized cell is in agreement with anticipated theoretical values and is the result of the approximately 30-times greater concentration of potassium within the cell. When a myocardial cell has been stimulated by the applica-

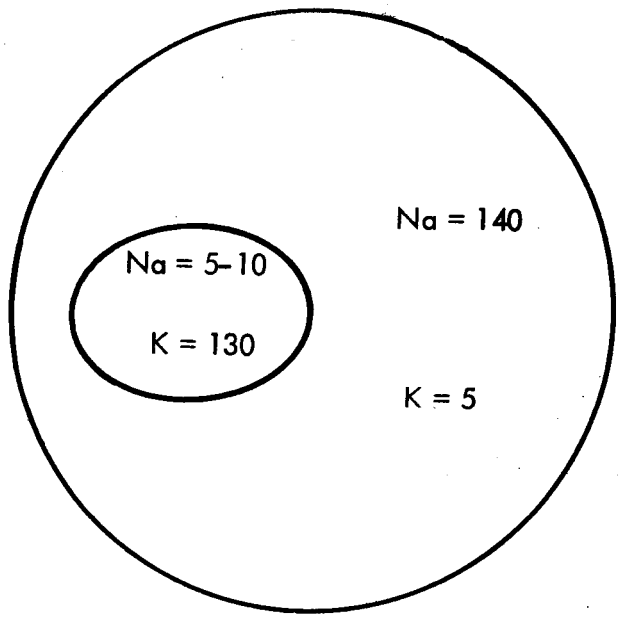


FIG. 1-1. Intra- and extracellular cation content.

tion of a positive voltage (about +20 to +30 mv) sufficient to reduce its intracellular negativity to the neighborhood of −60 mv, a threshold is crossed beyond which depolarization proceeds to completion without further stimulation. Attainment of the threshold effects a change in membrane permeability to sodium, increasing it as much as 200 times over the resting level. This permits the influx of sodium ions that neutralize the remaining negative charges lining the cell and further reduce the insulating capacity of the membrane. The additional penetration of sodium which follows is sufficient to reverse the intracellular polarity to a level of +20 to +30 mv. The flow of sodium and the increasing membrane permeability attain simultaneous peaks, then decline together. Following the raised permeability to sodium and coincident with its return toward normal, a similar change in potassium permeability permits an outward movement of potassium ions. Reconstitution of intracellular negativity begins when the efflux of potassium exceeds the inward flow of sodium, and slows (phase 2, below) when they are balanced during an interval of low permeability to both cations. The faster phase of recovery which follows is believed to accompany a further increase in potassium permeability with more rapid effusion and extracellular accumulation of potassium ions. The final ionic reconstitution of the cell is achieved largely during the resting phase (phase 4) of the myocardium by the "ionic pump" mechanism, which exchanges sodium for potassium

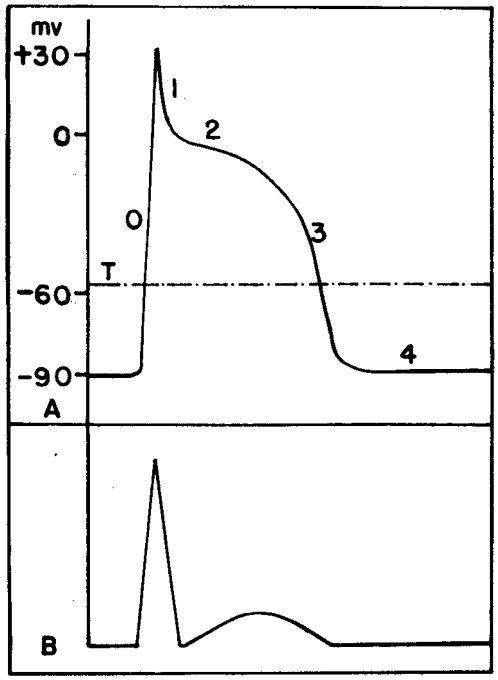


FIG. 1-2. Activation of the cell A. Intracellular potentials during depolarization and repolarization. Phases 0 to 4 (see text). T = threshold above which depolarization proceeds without further stimulation. *B.* Simultaneous surface electrogram.

across the cell membrane—possibly by means of carrier substances. The process of repolarization is more complex and difficult than depolarization and takes more time for completion. It is performed at a cost of metabolic energy and is more vulnerable than depolarization to interference by abnormal levels of extracellular electrolyte, particularly potassium.

The phases which together constitute the cycle of cellular activity are diagrammed in Figure 1-2 and are designated 0, 1, 2, 3, and 4. The period of depolarization coincides with phase 0. The phase of rapid repolarization, phase 1, is followed by the slower plateau, phase 2. Phase 3, coincident with the period of slow recovery, merges with phase 4, during which the maximal resting potential (MRP) is attained. Anatomic contraction of the fiber begins with phase 0 and persists through phase 3. On a simultaneously recorded surface electogram the QRS complex coincides with phase 0 and the T wave with phase 3.

COMMENT

The activity of a myocardial cell may be likened to the action of a semiautomatic firearm that discharges, reloads, and resets the firing mechanism at a single trigger-pull, and is reconstituted for the next contraction as it completes the first. The cell itself is more like a crossbow that has been wound up

and prepared for discharge. The arrow is fired by light finger pressure, and the weapon immediately readied for another cycle by turning the crank, fitting the dart, and completing certain time-consuming procedures. Little force is needed to discharge the bow, but a practiced and coordinated effort is required to prepare it for the next firing. Once the crossbow has been made ready, only a major disorder of the mechanism could interfere with discharge (depolarization). On the other hand, any impediment in the crank, rack, pinion, or the bowman's muscle could result in faulty preparation of the weapon for subsequent use.

SUPERNORMALITY

The U Wave

Once rapid discharge has been initiated, the cell is completely resistant to further stimulation (wholly refractory) until reconstitution is almost complete. Some preparations exhibit a slow termination of phase 3 prior to its junction with phase 4 (Figure 1-3). This takes the form of a diastolic afterpotential corresponding in time to the U wave of the clinical ECG and coincident with the period of supernormality. The muscle is not only capable of being stimulated at this time, but because it has not attained its maximum diastolic (resting) potential (MRP) can be brought up to threshold level with *less* than ordinary stimulus voltage. It is the time when premature beats are most likely to take place. They could originate from a focus of ectopic

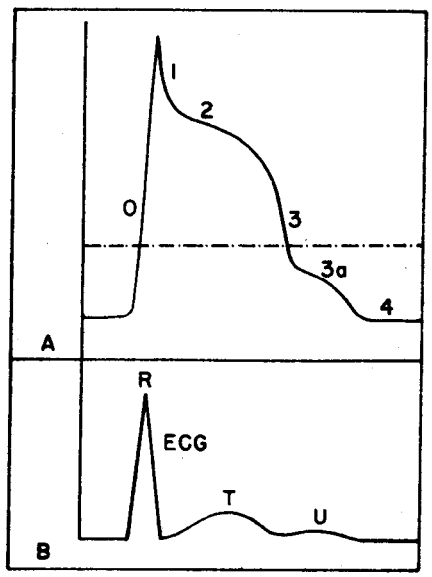


FIG. 1-3. Supernormality. Schema as in Fig. 1-2. *A*. Diastolic afterpotential; 3a = slow termination of phase 3 prior to phase 4. *B*. Clinical ECG illustrating temporal similarity of the U wave to the afterpotential of the intracellular curve.

activity insufficiently potent to stimulate the heart at any other phase of the cycle. The mechanism of clinical U wave genesis, however, is poorly understood. It has been suggested that extracellular accumulation of potassium ions released during phase 1 somehow impedes final reconstitution of the cell until the ions have been either completely resorbed or diffused. This explanation, however, is not well supported by experimental evidence. In any event, extensive differences in U wave size and configuration are seen during clinical shifts in serum potassium. Probably, a more valid hypothesis attributes the U wave to Purkinje fiber repolarization recorded from the body surface. The activation time of these fibers is much longer than that of the ventricular myocardium, which allows the Purkinje recovery deflections to appear *after* the more prominent ventricular T waves and thus escape cancellation and envelopment. Potassium variations are also known to have more effect on the Purkinje fibers than on the ventricular myocardium.

AUTOMATICITY

Studies of cells from the sinus node, the atrium, the atrioventricular node, the conducting bundles, the Purkinje fibers, and the ventricular myocardium indicate that each behaves somewhat differently from the others in the contour of the activation curve and in its response to various pharmacologic agents. Cells from the sinus node, certain areas within the atria, the His bundle and its branches, and the Purkinje system are capable of automatic rhythmic discharge. They differ from the rest of the myocardium by exhibiting continuous slow diastolic depolarization with gradually lessening intracellular negativity until the threshold level is attained, at which rapid discharge occurs (Fig. 1-4). The slope of the slow depolarization curve, the threshold height, and the maximum potential attained at the end of repolarization all determine the frequency of its rhythm. Acetylcholine, which decreases the slope of diastolic repolarization and increases the level of electronegativity (transmembrane potential) at the end of repolarization, slows the rate. It is by such means that intense vagal stimulation, for example, may stop all sinus and atrial activity. The slope is diminished by lowered temperature and increased by fever. Hypoxia, increased P_{CO_2} tension, and anatomic stretching exaggerate the slope and with it the rate. High calcium concentration in the extracellular fluid raises the threshold for rapid discharge and slows the rate. A lower concentration of calcium reverses this trend. The current of injury which develops in the vicinity of damaged myocardium can contribute part or all the voltage needed to attain the threshold level and may thus initiate ectopic beats. Only cells that exhibit diastolic depolarization are capable of automaticity, but other cells not normally so endowed may acquire this property under abnormal and usually stressful circumstances.

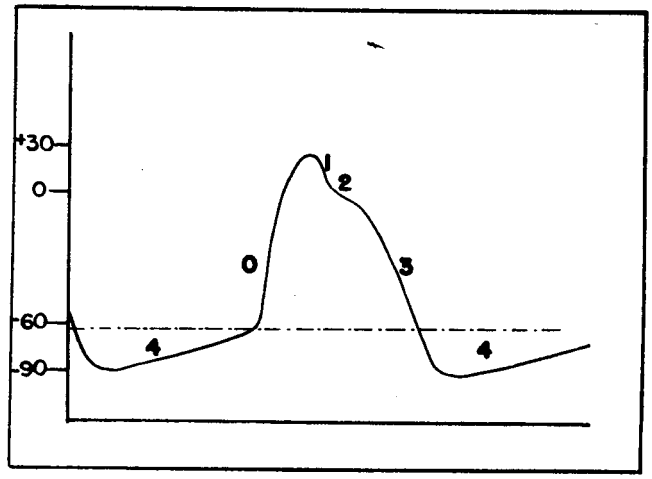


FIG. 1-4. Automaticity. Slow diastolic depolarization in sinoatrial cell capable of automaticity. When the rising potential of phase 4 exceeds the threshold level, the cell depolarizes without further stimulation.

SURFACE MANIFESTATIONS

The polarized cell, despite the known electronegativity of its interior, exhibits no external voltage or activity when investigated by means of nonpenetrating or extracellular electrodes. The potential level at such electrodes will not deviate from zero (Fig. 1-5*A*). On stimulation, however, the region which is first to depolarize becomes negative (or less positive) in relation to its environment.

If the exploring electrode has been situated at a point 180° away from the point of stimulus, it will record electropositivity. (An electrode lying in the path of an advancing wave of negativity, facing the depolarization front, will sense electropositivity.) Since, by convention, positive voltage is indicated by a baseline ascent, the recording voltmeter will exhibit an upward deflection (Fig. 1-5*B*). The level of positivity will continue to rise as depolarization progresses until one-half the cell (or muscle preparation) has been activated (Fig. 1-5*C*); then, as the midpoint is passed, the voltmeter—although still indicating positivity—will do so at a lesser level. A spike will be inscribed at the point of reversal (Fig. 1-5*D*). The level of positivity will continue to wane with further progress until complete depolarization has been achieved when it will have diminished to zero (Fig. 1-5*E*). With the onset of repolarization the electrode, now in an area of negativity, will record a minus potential (Fig. 1-5*F*) and inscribe a downward deflection as recovery progresses (Fig. 1-5*G*). It will finally return to its original isoelectric level when the cycle has been completed (Fig. 1-5*H*). As noted previously, repolarization is a slower process than depolarization, and the deflection it produces is of longer duration than the depolarization spike. The movement of ions required for cellular recovery is of the same order of magnitude as that which occurs dur-

ing depolarization. Accordingly, the waves of depolarization and repolarization are equal in area but opposite in direction.

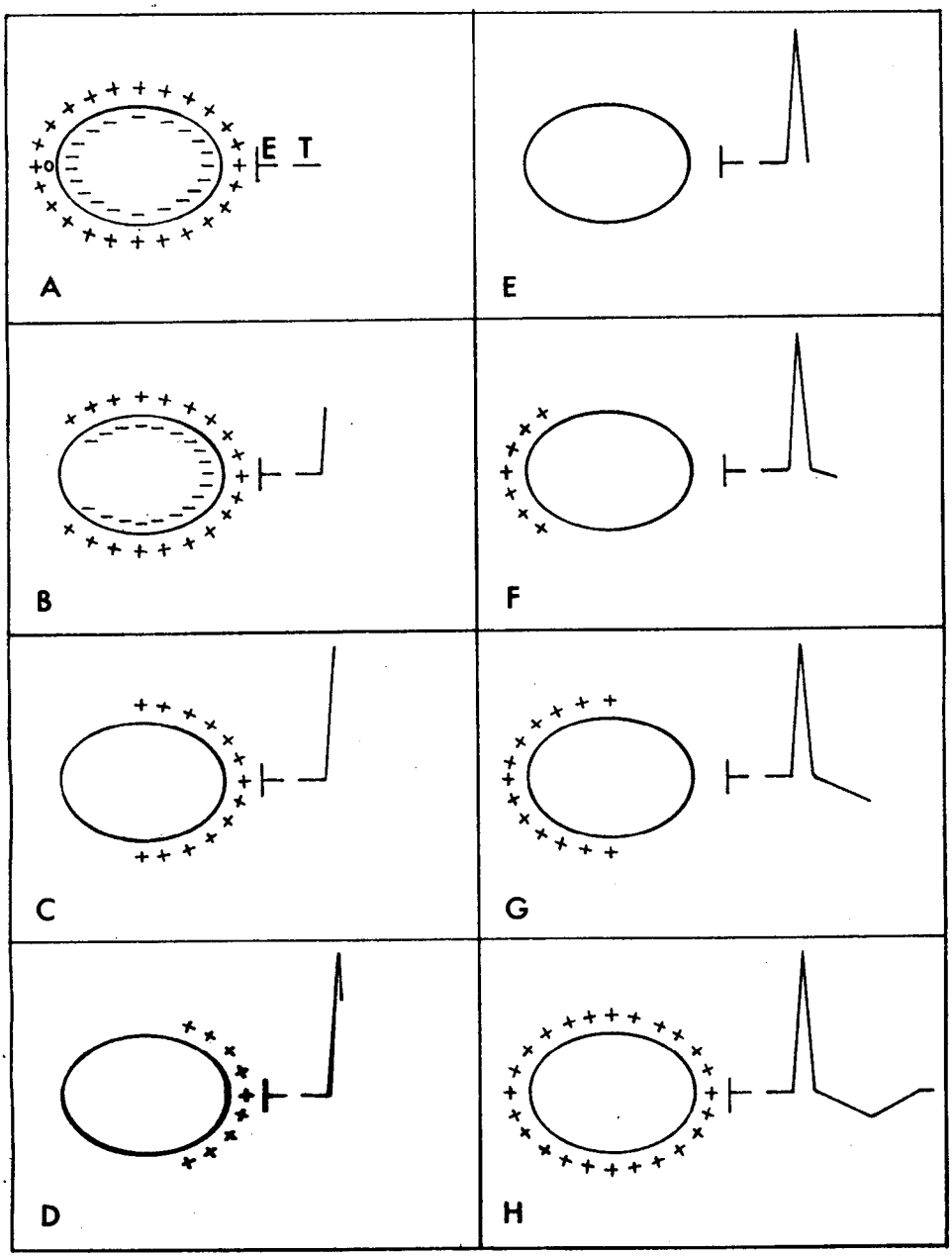
MODIFICATION OF THE ACTIVATION PATTERN

It is possible in some preparations of adequate size to inactivate (by freezing) one part of the cell and render it unresponsive to stimulation despite being fully polarized (Fig. 1-6*A*). If this is done on the side facing the electrode and the cell is stimulated as before, depolarization will proceed to the boundary between uninhibited and inert sections and stop at that point (Fig. 1-6*B*). The galvanometer will record ascending positivity as the unmodified portion is activated, but will remain at an elevated level when the inert section is reached. When recovery begins the voltage difference will diminish, (Fig. 1-6C) slowly as before, until the cell is reconstituted, at which point the isoelectric level will once more be attained (Fig. 1-6*D*). The resulting curve will be monophasic and can be attributed to the activity (discharge and recovery) of a normal segment of myocardium, here called the **a** half, for the sake of convenience, balanced against the inert **b** half. If the cell is rendered inert on the **a** side and stimulated at the junction, a curve will be inscribed which is also monophasic but opposite in sense to the **a** curve and attributable to the **b** segment (Fig. 1-6*E*, *F*, and *G*). When the two curves are recorded in their normal order and temporal sequence (**b** following **a** by the time required for depolarization of **a**), they may be algebraically added to achieve resynthesis of the original biphasic pattern (Fig. 1-6*H*).

Components

This exercise is introduced for the sole purpose of demonstrating alterations in form of the *total curve* resulting from modifications of one of its *com-*

FIG. 1-5. Surface electrical manifestations of cellular depolarization and repolarization. 0 = point of stimulation, E = unipolar electrode, T = tracing or recording. *A.* Cell fully polarized, prior to application of stimulation at left; total electrical balance. *B.* Beginning loss of electrical charges where first stimulated; electrode now in an area of relative positivity (by convention, a positive (+) influence causes a rising trace). *C.* Cell one-half depolarized; T now at its greatest positivity, since the largest difference exists between the right and left sides of the cell. *D.* Further loss of electrical charges; electrode still in area of positivity but less so than in C; this results in a diminution in voltage and peaking of the trace. *E.* Cell completely depolarized; electrode now at electrical zero, and T returns to the baseline. *F.* Beginning repolarization where cell was initially depolarized; electrode now in an area of relative negativity. Repolarization is a slower process than depolarization; the slope of T is more gradual than the initial rise. *G.* Cell one-half repolarized; T attains its greatest negativity. *H.* Complete repolarization; T returns to the baseline. Minus signs omitted after 1-5B in the interest of simplicity.


E
T
A
E
B
F
C
G
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H

ponents. For example, if, with the **b** half uninhibited, the **a** side of the preparation is subjected to cooling, reduced oxygen tension, or mechanical pressure, both depolarization and recovery of **a** will be slowed (Fig. 1-7). Repolarization, however, will exhibit a greater total delay than depolarization. Algebraic summation of the curves will reveal a barely perceptible difference in the discharge spike but a total change in direction of the recovery deflection.

It seems likely that the mechanism responsible for the normally upright T waves of most mammalian hearts is dependent on this phenomenon. The myocardium of the endocardial side of the left ventricle is the first to be depolarized but is delayed in its recovery—probably by the intraventricular systolic pressure—and consequently has diminished blood flow and oxygen tension compared with the epicardial layers. This takes place during inscription of the T wave (Fig. 1-8). Stated otherwise, the **a** (endocardial) side of the myocardium is the first to depolarize and the last to recover.

Repolarization

The "normal" sequence of repolarization and T wave sense may be reversed in susceptible individuals by the ingestion of ice water. The intimate relation of esophagus to the dorsal aspect of the heart permits cooling of its outer surface but probably has little effect on the deeper layers. It seems likely that T wave inversion occurs as the result of inhibition and slowing of the chilled outer layers and tends to restore the endocardial-to-epicardial order of recovery. Something similar may also be responsible for the negative T waves of left ventricular dilatation, an anatomic distortion of a muscular chamber in which the outer myocardial fibers are stretched more than the inner. This disparity results from the geometric observation that the area of a sphere varies with the square of its radius. The difference is greater with thick-

FIG. 1-6. Derivation of components of the surface activation curve. Schema as in Fig. 1-5; unipolar electrode present but omitted for simplicity. The **b** half of the cell has been rendered inert by freezing. *A*. Cell fully polarized before application of stimulus; tracing flat. *B*. Cell is one-half depolarized (as in Fig. 1-5*C*); tracing has attained its maximal height. However, wave of depolarization unable to proceed to the inert **b** half. *C*. Beginning (slow) repolarization of the **a** half; **T** begins to descend as the difference in electrical potential diminishes between **a** and **b**. *D*. With complete repolarization of the **a** half, **T** returns to the baseline. *E*. Same as *A* except that the **a** half is inert. Stimulus can no longer be applied to the left extremity because it will not be transmitted across the inert **a** half. It is applied at the junction between **a** and **b** indicated by **0**. *F*. Appearance of the tracing with **b** half depolarized and **a** half remaining polarized but inert. *G*. Appearance of complete curve obtained from depolarization and recovery of the **b** half with **a** half inert—the reverse of Fig. 1-6*D*. *H*. Algebraic summation of the **a** and **b** curves. Curve **b** is displaced to the right (later in time) by the interval **I**, the time needed to depolarize the **a** half; **S** = the reconstituted curve of the **a** and **b** components. It resembles Fig. 1-5*H*.