The effects of anesthesia on the action potential of the heart tissues
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A review of the cause/effect relationship that anesthetic agents have on the action potential of the myocardial and automatic cells of the heart.

What is the exact mechanism responsible for the arrhythmias observed and sometimes expected with anesthesia? In order to answer this question, one must delve into the alterations incurred by anesthetic agents on the action potential of the myocardial and automatic cells of the heart.

Action potential is merely the electrical potential which exists across a cell membrane. Curve A, Figure 1, demonstrates the change in electrical activity of a myocardial cell membrane upon stimulation. The resting membrane maintains a potential of $-90 \text{ MV}$, with a predominance outside the selective semi-permeable membrane of sodium, and a predominance inside the membrane of potassium.²

This membrane is only slightly permeable to sodium, but very permeable to potassium, which is retained intracellularly by the negative intracellular potential. When the cell membrane is excited, it becomes more permeable to sodium which, in turn, rushes into the cell to reverse the transmembrane potential to $+30 \text{ MV}$, (phase 0), allowing potassium ions to leave the cell (phase 1, 2).³ Sodium is then removed from the cell by active membrane transport (or that ever-famous sodium pump²). Potas-

In the normal automatic cells of the heart (which are the pacemaker or Purkinje cells), a slow “leaking”³ of sodium into the cell occurs during diastole (Curve B, Figure 1), causing a slow depolarization³ and decrease in mem-

Figure 1
Normal action potential (Myocardial cell and Purkinje cell)

Transmembrane potential of myocardial cell (A) and Purkinje cell (B).

MRP=membrane resting potential; TP=threshold potential; AR=absolute refractory period; RR=relative refractory period; SN=supernormal period. 0, 1, 2, 3, 4=phases of depolarization-repolarization.
brane resting potential. This occurs until a threshold potential of about -60 MV is reached, when rapid depolarization occurs.

During depolarization, the cell membrane is absolutely refractory to other stimuli and will not accept another stimulus until repolarization reaches the threshold potential. At this point, the membrane becomes relatively refractory, requiring an unusually strong stimulus to bring about depolarization. As repolarization continues toward resting potential, the membrane becomes hypersensitive to stimulation.

So as to study the effects of anesthetic agents on the action potential, an abbreviated approach is to divide these effects into sympathetic- and parasympathetic-like effects.

With the sympathetic-like effects (Figure 2), any one or all of the following may occur:

1. An increased rate of spontaneous diastolic depolarization (a more vertical phase 4 slope), in which impulse formation in the affected cell occurs more rapidly.

2. A reduction of membrane resting potential which causes the cell to be more excitable and impulse formation to occur sooner.

3. An increase in the threshold potential, which causes the cell to be more irritable and spontaneous depolarization to occur more readily.

Anesthetic agents which would alter phase 4 depolarization in the aforementioned manner would include cyclopropane, methoxyflurane, and ether. Other causative factors for sympathetic-like effects on the action potential of the heart tissue during the administration of anesthesia might include acidosis and hypoxia, which cause the cell membrane to become unstable and increase spontaneous depolarization.

If a patient has a rapid decrease in extracellular potassium, the membrane resting potential is reduced and the slope of phase 4 is increased, at which time one might see ventricular ectopic beats or ventricular tachycardia. Cardiac glycosides, such as digoxin, increase the rate of phase 4 depolarization. Hyperthermia and, of course, sympathetic stimulation also may cause the same responses.

For parasympathetic-like effects (Figure 3), vagal activity decreases the phase 4 slope, decreases the threshold potential, and increases the membrane resting potential, slowing the rate of discharge. Sinus activity is most affected, allowing lower centers to function as pacemakers. Atrial, A-V junctional, or idioventricular rhythms may occur. Halothane depresses spontaneous phase 4 depolarization, causing the frequently observed bradycardia. Reserpine, a parasympathomimetic agent, produces catechol depletion and probably produces bradycardia by increased vagal activity. Hypothermia decreases the phase 4 slope to slow the sinus rate, allowing the "escape" of pacemakers in the atria or A-V junctional areas.

Anectine causes depolarization of
1. Decreased rate of spontaneous diastolic depolarization (Less vertical phase 4 slope)
2. Increased membrane resting potential (MRP)
3. Decreased threshold potential (TP)

*Halothane, Anectine, Reserpine, Hypothermia

Striated muscle, which is maintained and, thereby, increases the serum potassium levels. This increased potassium may decrease the resting membrane potential, lengthen the phase 4 slope, and raise the threshold potential.\(^5\)

Bigeminy in an anesthetized patient is often the cause of "any coincidal PVC's in the attending anesthetist." Carnes elaborates on this arrhythmia in a manner which leads one to include it with the parasympathetic effects. He states that: "slowing the rate of conduction or decreasing the amplitude of action potential will decrease the rate of conduction. If there is a progressive decrease in rate and amplitude, a progressive impairment of conduction called 'decremental conduction' occurs. This type of conduction is responsible for the normal delay of impulse conduction at the A-V node."\(^3\)

If, however, this decremental conduction continues, failure of conduction may occur. This is usually unidirec-}

To conclude, anesthesia is capable of altering the action potential of the myocardium and automatic cells of the heart. Anesthetists should be knowledgeable of not only the possibility of detrimental and positive effects of anesthetic agents, but also the curative measures one must take to correct the alterations.

REFERENCES

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