

Figure 10-1 Sinus node and the Purkinje system of the heart, showing also the A-V node, atrial internodal pathways, and ventricular bundle branches.

interior of the fiber. Then the “plateau” of the ventricular action potential is caused primarily by slower opening of the slow sodium–calcium channels, which lasts for about 0.3 second. Finally, opening of potassium channels allows diffusion of large amounts of positive potassium ions in the outward direction through the fiber membrane and returns the membrane potential to its resting level.

But there is a difference in the function of these channels in the sinus nodal fiber because the “resting” potential is much less negative—only -55 millivolts in the nodal fiber instead of the -90 millivolts in the ventricular muscle fiber. At this level of -55 millivolts, the fast sodium channels mainly have already become “inactivated,” which means that they have become blocked. The cause of this is that any time the membrane potential remains less negative than about -55 millivolts for more than a few milliseconds, the inactivation gates on the inside of the cell membrane that close the fast sodium channels become closed and remain so. Therefore, only the slow sodium–calcium channels can open (i.e., can become “activated”)

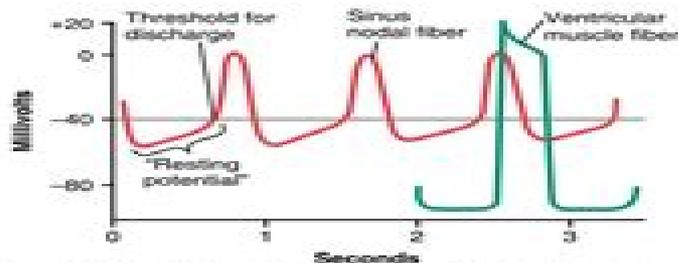


Figure 10-2 Rhythmical discharge of a sinus nodal fiber. Also, the sinus nodal action potential is compared with that of a ventricular muscle fiber.

and thereby cause the action potential. As a result, the atrial nodal action potential is slower to develop than the action potential of the ventricular muscle. Also, after the action potential does occur, return of the potential to its negative state occurs slowly as well, rather than the abrupt return that occurs for the ventricular fiber.

Self-Excitation of Sinus Nodal Fibers. Because of the high sodium ion concentration in the extracellular fluid outside the nodal fiber, as well as a moderate number of already open sodium channels, positive sodium ions from outside the fibers normally tend to leak to the inside. Therefore, between heartbeats, influx of positively charged sodium ions causes a slow rise in the resting membrane potential in the positive direction. Thus, as shown in Figure 10-2, the “resting” potential gradually rises and becomes less negative between each two heartbeats. When the potential reaches a threshold voltage of about -40 millivolts, the sodium–calcium channels become “activated,” thus causing the action potential. Therefore, basically, the inherent leakiness of the sinus nodal fibers to sodium and calcium ions causes their self-excitation.

Why does this leakiness to sodium and calcium ions not cause the sinus nodal fibers to remain depolarized all the time? The answer is that two events occur during the course of the action potential to prevent this. First, the sodium–calcium channels become inactivated (i.e., they close) within about 100 to 150 milliseconds after opening, and second, at about the same time, greatly increased numbers of potassium channels open. Therefore, influx of positive calcium and sodium ions through the sodium–calcium channels ceases, while at the same time large quantities of positive potassium ions diffuse out of the fiber. Both of these effects reduce the intracellular potential back to its negative resting level and therefore terminate the action potential. Furthermore, the potassium channels remain open for another few tenths of a second, temporarily continuing movement of positive charges out of the cell, with resultant excess negativity inside the fiber; this is called *hyperpolarization*. The hyperpolarization state initially carries the “resting” membrane potential down to about -55 to -60 millivolts at the termination of the action potential.

Why is this new state of hyperpolarization not maintained forever? The reason is that during the next few tenths of a second after the action potential is over, progressively more and more potassium channels close. The inward-leaking sodium and calcium ions once again overbalance the outward flux of potassium ions, and this causes the “resting” potential to drift upward once more, finally reaching the threshold level for discharge at a potential of about -40 millivolts. Then the entire process begins again: self-excitation to cause the action potential, recovery from the action potential, hyperpolarization after the action potential is over, drift of the “resting” potential to threshold, and finally re-excitation to elicit another cycle. This process continues indefinitely throughout a person’s life.

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