Effects of Low and High Concentrations of Potassium on the Simultaneously Recorded Purkinje and Ventricular Action Potentials of the Perfused Pig Moderator Band

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ABSTRACT
The use of the perfused pig moderator band has allowed us to study the rapid simultaneous changes in Purkinje and ventricular action potentials induced by perfusing solutions of high (10 to 12 mM) and low (0.6 to 0.8 mM) K concentrations. High K shortened the plateau more in Purkinje fibers than in ventricular fibers and decreased the difference between the action potential durations of the two fiber types. Low K prolonged the plateau in Purkinje fibers but shortened it in ventricular fibers and increased the difference between the action potential durations. Low K initially hyperpolarized both Purkinje and ventricular fibers. However, the resting potential of the Purkinje fiber subsequently decreased as phase-4 depolarization increased. The decreased maximum repolarization potential associated with low K-induced pacemaker activity was time rather than voltage dependent. When the perfusate was changed from low K to control (K = 4.8 mM), phase-4 depolarization was rapidly suppressed and the action potential of the Purkinje fiber was shortened to less than that produced by high K, and then changes in amplitude of the resting and action potentials occurred. Our observations help to explain some of the effects of low and high K on rhythm and conduction.

ADDITIONAL KEY WORDS
resting potential, action potential plateau, phase-4 depolarization, maximum repolarization potential, Zwaardemaker-Librecht effect, pacemaker, arrhythmia

Purkinje fibers have different electrophysiological properties than ventricular myocardial fibers (1-4). Spontaneous diastolic depolarization occurs normally in the Purkinje fibers but not in the ventricular fibers. In addition, the transmembrane action potentials of the Purkinje fibers have a greater amplitude, larger spike, more negative resting membrane potential, more rapid upstroke velocity, and longer duration than the action potentials of ventricular fibers. These differences may be responsible for abnormalities in impulse formation and conduction in the ventricle (5) and may be modified by agents which have different effects on the action potentials of the two types of fibers (6). Therefore, a study of factors which alter the differences between the Purkinje and ventricular action potentials may contribute to a better understanding of the membrane properties of the two types of fibers and to a better understanding of the mechanisms of ventricular arrhythmias and conduction disturbances.

Several investigators have studied the effects of changes in extracellular K concentration on the action potentials of Purkinje and ventricular fibers (7-13). Ventricular fibers
have been studied in bathed and perfused preparations (7-10), but the Purkinje fibers have been studied only in bathed preparations (11-13). When the extracellular K concentration is increased above ca. 5.0 mM, the amplitude, upstroke velocity, and duration of both Purkinje and ventricular action potentials are decreased, the resting potential of both types of fibers becomes less negative, and diastolic depolarization is suppressed in the Purkinje fiber (7-12). When the extracellular K concentration is decreased below ca. 3.0 mM, the duration of both Purkinje and ventricular action potentials is increased and diastolic depolarization is enhanced in the Purkinje fiber (9,10,12,13). The effect of a decreased extracellular K concentration on the duration of phase 2 and on the resting potential is different in the Purkinje and ventricular fibers. The duration of phase 2 is increased in the Purkinje fiber of the bathed sheep and calf preparation (12,13) but is decreased in the ventricular fiber of the perfused rabbit heart (10). The resting potential of the Purkinje fibers in the bathed sheep and calf preparation becomes more negative or remains unchanged when the extracellular K concentration is decreased from 5.4 to 2 mM (11-13), but becomes less negative when the extracellular K concentration is decreased to less than 1.35 mM (11). However, the resting potential of the ventricular fibers in the perfused rabbit heart becomes more negative when the extracellular K concentration is decreased from 4.8 to 0.8 mM (10).

We questioned whether the differences between the effects of the decreased extracellular K concentration on the action potentials of the Purkinje and ventricular fibers reflect differences between the electrophysiologic properties of the two types of fibers or between the techniques of supplying the K-deficient solution. Changes in action potential occur ten times more slowly in bathed than in perfused preparations (8). Thus, the bathed fibers are studied after a longer exposure to K-deficient solution than are the perfused fibers and may be subject to a greater loss of intracellular K. It is possible that changes in the action potential of the perfused preparation may reflect more accurately the effect of changes in the extracellular K concentration.

To compare the effect of a change in the K concentration on the action potentials of Purkinje and ventricular fibers, it is desirable to study both fibers simultaneously. There are two studies (14,15) in which reference is made to the effects of changes in K concentration on simultaneously recorded Purkinje and ventricular action potentials. However, the investigators used bathed preparations and reported only the effect of the varying K concentrations on the total action potential durations.

This paper presents a study of the effects of changes in extracellular K concentration on the simultaneously recorded Purkinje and ventricular action potentials of the perfused pig moderator band. This preparation is...
similar to the perfused calf and sheep moderator band used by Delèze to study ventricular action potentials (8) and was chosen because both Purkinje and ventricular fibers lie close to the surface and because the artery perfusing the band is large enough to be cannulated (16).

**Methods**

Pigs weighing from 20 to 50 kg were anesthetized with sodium pentobarbital, 50 mg/kg, injected intravenously or into the heart. The heart was removed rapidly, and the moderator band with a portion of interventricular septum and free wall of the right ventricle was placed in a plexiglass chamber of 300-ml capacity in which solution was continuously exchanged at a rate of 15 ml/min. The preparation was also perfused at a rate of 2 to 10 ml/min through a cannula inserted into the septal branch of the anterior descending coronary artery (Fig. 1). All branches of the septal artery except that supplying the moderator band were ligated, and the nonperfused septal muscle was removed. Figure 2 demonstrates that the Purkinje fibers were located near the surface of the moderator band.

We injected 0.2 ml of a dilute Evans blue dye solution (3 mg/100 ml H₂O) into the cannula. When the perfusion was adequate, there was immediate staining of the entire preparation. We
also injected 0.5 ml of a $10^{-5}$ solution of epinephrine into the bathing solution. As a rule, the addition of this amount of epinephrine to the bath did not produce spontaneous rhythms in the quiescent preparation. We then injected into the cannula 0.2 ml of the bathing solution into which the epinephrine had been added. When the perfusion was adequate, rapid spontaneous activity occurred immediately.

The ends of the band were immobilized with pins on a piece of flat cork which could be raised or lowered by a piston. The surface of the band was positioned just below the surface of the bathing solution.

The Krebs-Henseleit solution (control) contained 143 mM Na, 4.8 mM K, 2.4 mM Ca, and 1.2 mM Mg in 1 liter as well as phosphate, bicarbonate, and dextrose. The control solution was altered in two ways: (1) by decreasing the K concentration to 0.6 to 0.8 mM (low K), and (2) by increasing the K concentration to 10 to 12 mM (high K). All solutions were maintained at 37°C and were equilibrated with 95% O$_2$ and 5% CO$_2$. The solution perfusing the band through the arterial cannula and the solution in the bath were the same. When solutions were changed, the bath was rapidly emptied by suction and refilled with the solution perfusing the band through the cannula.

The preparation was driven at rates ranging from 1 to 3/sec by a Grass S-4 stimulator and isolation unit. Stimuli of 2-msec duration and 1.5 X diastolic threshold strength were applied through a pair of stainless steel hook electrodes inserted into the septal end of the preparation. In some experiments, premature stimuli from the same stimulator were also introduced.

Transmembrane action potentials were recorded simultaneously from Purkinje and ventricular fibers with either flexibly or rigidly

![Figure 3](image-url)

Simultaneously recorded action potentials from the ventricular fiber (upper trace) and Purkinje fiber (lower trace) before (A), during (B,C), and after (D) perfusion with a solution of 0.6 mM K. In this and subsequent figures, the numbers above the action potentials designate the action potential amplitudes in mv. The intersection of the tangents to the plateau and to the phase of rapid repolarization define the durations of phases 2 and 3. On the right are shown the superimposed tracings of action potentials of ventricular fibers (upper trace) and Purkinje fibers (lower trace) from A (solid line) and B (broken line). In C, the rhythm is spontaneous. Note that in both Purkinje and ventricular fibers, the amplitudes of the takeoff potential and action potential are decreased when depolarization occurs from incompletely repolarized fibers.
The amplitude of the action potential was measured from the maximum repolarization potential to the peak of the action potential spike. The amplitude and duration of the depolarization following the action potential spike were measured from the nadir of the spike to the point where the slope of the plateau changed from positive to negative (Fig. 4). The duration of phase 2 was measured from the nadir of the spike to the intercept of tangents to the flattest portion of the plateau and to the steepest portion of phase 3 (Fig. 3). The duration of phase 3 was measured from the end of phase 2 to the maximum repolarization potential. The effective refractory period was assumed to equal the duration of the action potential from its onset to repolarization at the level of −60 mV because propagation of an impulse is not possible until the membrane potential reaches this level (11). The relative refractory period was measured as the interval from −60 mV to the maximum depolarization potential. The velocity of phase-4 depolarization was determined from measurements of the potential difference and the time between the maximum depolarization potential to the onset of the subsequent action potential.

Statistical significance was evaluated by the Student t-test.

Results

Action potentials of Purkinje and ventricular fibers were recorded simultaneously during perfusion with control solution in 17 experiments, during perfusion with low K solution in ten experiments, and during perfusion with high K solution in seven experiments. Table 1 shows the action potential amplitudes, maximum repolarization potentials, and durations of the phases of repolarization in preparations stimulated at a rate of 1/sec. During perfusion with control solution, the action potential amplitude was greater and the maximum repolarization potential was more negative in Purkinje than in ventricular fibers in all experiments. In the Purkinje fiber, the spike was followed by a phase of depolarization which averaged 35 mV in amplitude and 16 msec in duration. In the ventricular fiber, depolarization after the spike was either absent or less than 5 mV in amplitude and less than 5 msec in duration. Phase 2 and the effective refractory period were always longer in Purkinje than in ventricular fibers. Phase 3 and the relative refractory period were longer in Purkinje than in ventricular fibers in 15 experiments and were slightly shorted in Purkinje than in ventricular fibers in the remaining three experiments.

Perfusion with Low K Solution

In all experiments, the amplitude of the action potential increased and the maximum repolarization potential became more negative within 1 minute and spontaneous rhythms occurred within 5 to 45 minutes of starting the low K perfusion. Figure 3 and Table 1 show the maximal changes produced by the low K perfusion prior to the onset of spontaneous activity. In both the Purkinje and ventricular fibers the maximal increase in amplitude of the action potential and maximum repolarization potential occurred within the first 5 minutes of the low K perfusion. Thereafter, the low K perfusion produced no further changes unless the membrane potential at the onset of depolarization,
Superimposed tracings of action potentials of a Purkinje fiber recorded before (solid line) and after 25 minutes (broken line) of perfusion with a solution of 0.6 mm K. The vertical lines mark the end of the phase of depolarization which follows the spike. Low K has prolonged all phases of repolarization. Note particularly the prolongation of the phase of depolarization which follows the spike. See text for discussion.

i.e. the takeoff potential, became less negative than control. When this occurred, the amplitude of the action potential decreased (17).

Changes in the repolarization of both Purkinje and ventricular fibers developed continuously throughout the entire period of perfusion and were therefore more pronounced in the preparations with the more delayed onset of spontaneous activity. In the Purkinje fiber, low K prolonged the average duration of phases 2 and 3, the effective refractory period, the relative refractory period, and the total duration of the action potential. The prolongation of phase 2 was due in part to prolongation of the phase of depolarization which followed the action potential spike. This phase was prolonged in seven of ten experiments by 5 to 28 msec and remained unchanged in the remaining three experiments. Typical changes are shown in Figure 4.

In the simultaneously recorded ventricular fiber, low K shortened the duration of phase 2 and the effective refractory period and prolonged the duration of phase 3 and the
relative refractory period. The net change in action potential duration was determined by the sum of these opposing effects. The absolute increase in phase 3 and the relative refractory period was less in the ventricular than in the Purkinje fiber in all experiments.

Low K caused a variable increase in the slope of phase 4 in the Purkinje fiber. Figure 5 illustrates an experiment in which the amplitude of the action potential increased and the maximum repolarization potential became more negative. However, the slope of phase 4 increased only slightly and, as a result, the resting potential also became more negative. In two experiments, low K progressively increased phase-4 depolarization and the fibers acquired pacemaker characteristics. As phase-4 depolarization increased, the takeoff potential and the maximum repolarization potential became less negative, and the amplitude of the action potential and upstroke velocity decreased. When the rate of stimulation was increased and the interval between the action potentials was shortened, the takeoff potential and the maximum repolarization potential became more negative and the amplitude of the action potential and the upstroke velocity increased. The more negative maximum repolarization potential associated with the increased rate of stimulation could be related to either the more negative takeoff potential or the decreased interval between the action potentials. To separate these two parameters we induced premature action potentials in Purkinje fibers with low K-induced pacemaker activity. Figure 6 shows two such experiments. In both A and B of this figure, the premature action potentials have more negative maximum repolarization potentials than the nonpremature action potentials. In A, the premature (third) action potential begins after repolarization of the preceding action potential has been completed. Thus, the takeoff potential of the premature action potential is more negative than the takeoff potential of the nonpremature (first and second) action potentials. In B, however, the premature (third) action potential begins before repolarization has been completed, and its takeoff potential is the same as that of the nonpremature (first and second) action potentials. These observations suggest that the changes in the maximum repolarization potential are determined.

**Figure 5**

Action potentials of the Purkinje fiber recorded during the continuous perfusion of the same fiber before (A) and after 25 minutes (B) of perfusion with a solution of 0.8 molar K. Low K has caused the resting potential to become more negative. See text for discussion.
FIGURE 6
Action potentials of a Purkinje fiber recorded during perfusion with low K solution in two different experiments. AP = action potential amplitude; TOP = takeoff potential; MRP = maximum repolarization potential. In A, the maximum upstroke velocity is represented by the amplitude of the upward directed spike below each action potential. The third action potential is premature and begins 625 msec after the second. The fourth action potential is spontaneous. It begins after the longest interval (1400 msec) and has the least negative takeoff potential and maximum repolarization potential. In B, the third action potential is premature and begins 600 msec after the second. See text for discussion.

FIGURE 7
Action potentials of the Purkinje fiber recorded during the continuous penetration of the same fiber (lower trace) and bipolar electrogram (upper trace) before (A), during (B), and after (C-F) perfusion with a solution of 0.8 mM K. Note that in C-E, the Q-T interval of the electrogram is shorter than in A, B, and F. See text for discussion.
by the interval between the action potentials rather than by the takeoff potential.

PERFUSION WITH CONTROL SOLUTION AFTER PERFUSION WITH LOW K SOLUTION

Figure 7 shows that changes in the action potential of the Purkinje fiber occurred within the first minute of changing the perfusate from the low K to the control solution, and that the action potential returned to its control configuration within 2 minutes. The changes occurred in the following sequence: (1) suppression of phase-4 depolarization, (2) shortening of phases 2 and 3 and total action potential duration to less than control, (3) reduction of the action potential amplitude and of the maximum repolarization potential, and (4) lengthening of phases 2 and 3 until the shape and duration of the action potential returned to control. Figure 7 also shows that the shortening of the duration of the action potential to less than control was accompanied by a shortening of the Q-T interval of the electrogram to less than control. This Q-T shortening suggests that repolarization was shortened in the entire preparation.

PERFUSION WITH HIGH K SOLUTION

Changes in the amplitude and duration of the action potential and in the maximum repolarization potential occurred within the first minute of starting the high K perfusion. Table 1 and Figure 8 show that in both Purkinje and ventricular fibers, the amplitude of the action potential decreased, the maximum repolarization potential became less negative, and the durations of phase 2, the effective refractory period, and the action potential became shorter. The absolute shortening of phase 2 and of the total duration of the action potential was greater, however, in the Purkinje than in the ventricular fibers. The duration of phase 3 and of the relative refractory period was either shortened slightly or unchanged in both Purkinje and ventricular fibers. High K also suppressed phase-4 depolarization in the Purkinje fiber.

EFFECT OF PERFUSION WITH LOW AND HIGH K SOLUTIONS ON THE DIFFERENCES BETWEEN THE ACTION POTENTIALS OF PURKINJE AND VENTRICULAR FIBERS

Table 2 and Figures 8 and 9 show the effect of perfusion with the low and high K solutions on the differences between the action potentials of Purkinje and ventricular fibers.

**FIGURE 8**

Superimposed action potentials of Purkinje (P) and ventricular (V) fibers recorded during the continuous penetration of the same Purkinje and ventricular fibers before (A) and after 3 minutes (B) of perfusion with a solution of 10 mM K. Note that the difference between the durations of the Purkinje and ventricular action potentials decreases as the rate increases during both the control and high K perfusions, but that at each rate, the difference between the durations of the Purkinje and ventricular action potentials is less during the high K perfusion than during the control perfusion.
Superimposed action potentials of Purkinje (P) and ventricular (V) fibers recorded during the continuous penetration of the same Purkinje and ventricular fibers before (A) and after 15 minutes (B) of perfusion with a solution of 0.6 mM K. Note that the difference between the durations of the Purkinje and ventricular action potentials decreases as the rate increases in A and B, but that at each rate, the difference is greater during the low K perfusion than during the control perfusion.

TABLE 2

Differences Between Purkinje and Ventricular Fibers During the Control Perfusion and the Effects of Perfusion with Low and High K Solutions on These Differences

<table>
<thead>
<tr>
<th>Difference between Purkinje and ventricular fibers</th>
<th>Control</th>
<th>Effect on difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Action potential amplitude</td>
<td>P &gt; V</td>
<td>No change*</td>
</tr>
<tr>
<td>Maximum repolarization potential</td>
<td>P &gt; V</td>
<td>No change*</td>
</tr>
<tr>
<td>Action potential duration</td>
<td>P &gt; V</td>
<td>Increased</td>
</tr>
<tr>
<td>Phase-2 duration</td>
<td>P &gt; V</td>
<td>Increased</td>
</tr>
<tr>
<td>Phase-3 duration</td>
<td>P &gt; V</td>
<td>Increased</td>
</tr>
<tr>
<td>Effective refractory period</td>
<td>P &gt; V</td>
<td>Increased</td>
</tr>
<tr>
<td>Relative refractory period</td>
<td>P &gt; V</td>
<td>Increased</td>
</tr>
<tr>
<td>Phase-4 velocity</td>
<td>P &gt; V</td>
<td>Increased</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Low K</th>
<th>High K</th>
</tr>
</thead>
<tbody>
<tr>
<td>No change*</td>
<td>No change</td>
</tr>
<tr>
<td>No change*</td>
<td>No change</td>
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<tr>
<td>Increased</td>
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<td>Increased</td>
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</table>

P = Purkinje; V = ventricular.

*Unless the takeoff potential of the Purkinje fiber became less negative than control; †Not statistically significant.

Our study has demonstrated that the perfused pig moderator band is a useful preparation for the study of the rapid, simultaneous action potential changes in Purkinje and ventricular fibers. At all rates studied, low K increased the differences between the effective refractory periods, the relative refractory periods, and the total action potential durations of the Purkinje and ventricular fibers, while high K decreased the differences between the effective refractory period and total action potential durations in the two fiber types. The differences between the relative refractory periods of the Purkinje and ventricular fibers were also decreased by high K, but this decrease was not significant.

Discussion

Our study has demonstrated that the perfused pig moderator band is a useful preparation for the study of the rapid, simultaneous action potential changes in Purkinje...
and ventricular fibers. In this study, the changes in the Purkinje and ventricular action potentials induced by the altered extracellular K concentrations occurred as rapidly as did the changes in the ventricular action potentials of the rabbit heart perfused by the Langendorf method with nearly identical solutions (10, 18, 19).

We have shown that the various changes in the extracellular K concentration produced similar effects on the Purkinje and ventricular action potentials of the pig moderator band as on the Purkinje fibers of the bathed sheep and calf preparation (11-13) and on the ventricular fibers of the perfused rabbit heart (10). In addition, we have made several previously unreported observations which may be helpful in the understanding of the effects of high and low K on rhythm and conduction. We have shown that the variable effects of low K on the resting potential of the Purkinje fiber is caused by the variations in the magnitude of the associated increase in phase-4 depolarization. We have also shown that the decrease in the maximum repolarization potential of the Purkinje fiber which occurs when low K increases phase-4 depolarization is time rather than voltage dependent. By recording the action potentials of the Purkinje and ventricular fibers simultaneously, we have shown that low K increases and high K decreases the differences between the action potentials of these two types of fibers. By recording the action potential of the Purkinje fiber during the change from the low K to the control perfusion, we have shown that shortening of the duration of the action potential and suppression of phase-4 depolarization precede changes in action potential amplitude, resting potential, and maximum repolarization potential.

Perfusion with the high and low K solutions induced changes in the differences between the durations of the Purkinje and ventricular action potentials because both high and low K had a different effect on the plateau of the Purkinje fiber than on the plateau of the ventricular fiber.

The observation that high K shortened the plateau more in the Purkinje fiber than in the simultaneously recorded ventricular fibers suggests that the plateau of the Purkinje fiber was more sensitive than the plateau of the ventricular fiber to changes in gK (potassium conductance) (7, 12, 20-22).

Low K increased the rate of repolarization following the spike and shortened the plateau in the ventricular fiber but decreased the rate of depolarization following the spike and prolonged the plateau in the Purkinje fiber. It is reasonable that a process which accelerates repolarization in the ventricular fiber would slow depolarization in the Purkinje fiber. The changes in the initial portion of the plateau of the Purkinje and ventricular fibers induced by low K may be attributed to an increase in the K outward current or to a more rapid decrease in the Na inward current (23). An increase in K outward current would occur if perfusion with the low K solution increased gK or the electrochemical driving force for K. The latter would be expected since the decrease in extracellular K concentration would cause a more negative K equilibrium potential and increase the difference between the transmembrane potential and the K equilibrium potential. We cannot exclude the possibility that changes in extracellular K concentration altered gNa or the activity of the electrogenic Na "pump" (11).

Our study has confirmed and extended the observations of Moore et al. (14). These authors reported that the differences between the durations of the action potentials and functional refractory periods of canine ventricular and Purkinje fibers decreased when the heart rate was increased. They also reported that the durations of the action potentials of both fiber types were increased when the K concentration of the bathing fluid was lowered from 2.7 to 0.67 mM and were decreased when the K concentration was raised to 8.1 mM. We have shown that at all K concentrations studied, the differences between the durations of the action potentials of the Purkinje and ventricular fibers decreased with increasing rate, but that at all rates studied, low K increased, and high K decreased the
differences between the action potential durations and the effective refractory periods of the two fiber types.

In man, hypopotassemia increases the incidence of ectopic beats and rhythms and may produce ventricular fibrillation (24-27). Our studies have shown that low K not only increases phase-4 depolarization but also increases the difference between the durations of the action potentials of the Purkinje and ventricular fibers. Hoffman and Cranefield (1) have suggested that the differences in action potential durations of the Purkinje and ventricular fibers may facilitate re-entry. The increase in these differences which we have observed would increase this possibility. Conversely, the antiarrhythmic effect of K (28) may be attributed, not only to the suppression of phase-4 depolarization, but also to the decrease in the differences between durations of the Purkinje and ventricular action potentials.

In the present study, the maximum repolarization potential of both Purkinje and ventricular fibers became more negative when the K concentration of the perfusate was 0.6 to 0.8 mM. In the ventricular fibers phase-4 depolarization did not occur and the resting potential also became more negative. The resting potential of the Purkinje fiber depended on the magnitude of the associated increase in phase-4 depolarization. When the increase in phase-4 depolarization was small, the resting potential also became more negative. When the increase was large, the resting potential remained unchanged or became less negative. Our results suggest that the inconsistent effects of low K on the resting potential of Purkinje reported in previous studies (11-13) may be explained by the variable effects of low K on phase-4 depolarization.

When low K increased phase-4 depolarization, the maximum repolarization potential became less negative. A similar change in maximum repolarization potential has been observed by others when pacemaker activity was induced by isoproterenol (29), digitalis (6), and hypoxia (30). Singer et al. (30) showed that the decrease in takeoff potential which occurred when the rate of stimulation was slow, and which was associated with abnormalities in conduction, was caused by the less negative maximum repolarization potential as well as by the increase in phase-4 depolarization. These authors attributed the less negative maximum repolarization potential to the time- and voltage-dependent decrease in gK which occurs during phase-4 depolarization (31). Our study suggests that the change in maximum repolarization potential in Purkinje fibers perfused with low K solution is time rather than voltage dependent (Fig. 6). When depolarization occurred during incomplete repolarization, the maximum repolarization potential became more negative even though the takeoff potential was decreased. The practical importance of the latter effect requires further investigation. It is possible that the more negative maximum repolarization potential of early premature beats may contribute to the slowing or suppression of ectopic rhythms.

The rapid change of the perfusing solution from low K to control rapidly abolished phase-4 depolarization and shortened the duration of the action potential of the Purkinje fiber. The shortening was more than that produced by perfusion with the high K solution and was similar to the shortening of atrial and ventricular action potentials in the isolated perfused rabbit heart which occurred after a similar change in perfusing solution from low K to control (19). In the rabbit heart experiments, the change in perfusing solution was immediately followed by a cardiac arrest which lasted from 10 to 30 seconds. We postulated that this arrest was due to inhibition of pacemaker activity rather than to depression of excitability. The rapid suppression of phase-4 depolarization observed in this study supports this hypothesis.

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References


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