Effect of Acute Coronary Artery Occlusion on Local Myocardial Extracellular K+ Activity in Swine

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SUMMARY We studied the time course, magnitude and homogeneity of the change in extracellular myocardial potassium activity after acute ligation of the left anterior descending coronary artery in pigs using potassium-sensitive electrodes made from a valinomycin-polyvinyl chloride matrix membrane. We also studied the relationship between the changes in potassium activity and the simultaneous changes in ventricular activation using the reference barrel of the K+ electrode to record ventricular electrograms. We found that the K+ rose sooner, more rapidly and to higher levels than previously reported. The K+ changes occurred in three phases: a phase of rapidly rising K+ that began within seconds of the ligation and lasted 5-15 minutes, a plateau phase that lasted approximately 15 minutes and a phase of slowly rising K+ that extended throughout the longest occlusion (60 minutes) used in this study. The K+ changes were reversed by release of the occlusion during the rapidly rising and plateau phases, but were not reversed by release of the occlusion during the phase of slowly rising K+. Inhomogeneities in the K+ rise appeared between the center and lateral margins of the midmyocardial ischemic zone, between the subendocardium and the subepicardium in the center of the ischemic zone, and between closely spaced electrodes located in the midmyocardial center of the ischemic zone. Thus, the change in K+ activity, as recorded by our electrodes, can be considered an excellent marker of ischemia. Changes in ventricular activation paralleled the K+ rise, the inhomogeneities of K+ rise and the reversal of the K+ rise after release but could not be entirely explained by the change in K+.

THE ACCUMULATION of substances released by the ischemic myocardium is thought to play an important role in the genesis of ischemia-related arrhythmias. Harris and co-workers demonstrated that the ischemic myocardium lost K+ into the extracellular space after acute coronary artery ligation. They also showed that the intracoronary injection of KCl produced electrocardiographic changes and ventricular arrhythmias similar to those induced by coronary artery ligation. Harris et al. concluded that the rise in extracellular K+ was a major cause of ischemia-induced ventricular fibrillation. These observations have been confirmed by other investigators. Recently, Downar et al. recently presented data that suggest that factors other than or in addition to the ischemia-induced K+ rise cause the associated electrophysiologic changes.

In all prior experiments, K+ was measured in the vein draining the ischemic zone. Thus, the K+ values were only an approximation of the actual myocardial extracellular K+ and the evidence for or against a
primary etiologic role of K⁺ was indirect. The development of flexible K⁺-sensitive electrodes in our laboratory¹⁴,¹⁵ permits the direct assessment of the changes in myocardial extracellular K⁺ activity (ak⁺) during acute ischemia. The studies reported in this article were designed to determine the magnitude, rate, time course, homogeneity and reversibility of the changes in myocardial extracellular ak⁺ after acute coronary occlusion. By simultaneously recording ventricular electrograms using the same electrodes, we could determine the relationship of the changes in myocardial extracellular ak⁺ to changes in intramyocardial conduction, and to the development of ventricular fibrillation. Preliminary results have been reported in abstract.¹³

Methods

Twenty-four domestic pigs of either sex weighing 25–40 kg and anesthetized with pentobarbital (30 mg/kg) were used in these studies. Respiration was maintained with room air supplemented with 95% O₂-5% CO₂ through an endotracheal tube by a Harvard pump at rates adjusted to maintain arterial blood saturation above 95% and pH at 7.40 ± 0.03. Core temperature was monitored using a Yellow Springs Instruments #403 rectal probe. Polyethylene catheters (1.5 mm o.d.) were placed in the femoral artery and femoral vein for blood pressure monitoring and blood sampling.

The heart was exposed through a midsternal thoracotomy and cradled in the pericardium. In five experiments, the sinus node was crushed and the right atrium paced at a constant rate of 90–180 beats/min using platinum bipolar plunge electrodes coupled to a Grass model S88 stimulator. In the remaining 19 experiments, the heart beat spontaneously at rates of 100–200 beats/min. A snare of 1-0 silk suture was positioned around the left anterior descending coronary artery just distal to the origin of the first diagonal branch.

Miniature K⁺-selective, double-barrel electrodes (0.25 mm o.d./barrel) (fig. 1) that could accurately measure in vivo myocardial extracellular ak⁺ were constructed from polyvinyl chloride (PVC) tubing and PVC-valinomycin matrix membrane and tested as previously described.¹¹ The K⁺ electrodes used in these experiments had a calibration slope of 57–61 mV per decade change in K⁺ activity, an impedance of 10–15 MΩ and a time constant of 40–50 msec.

In 22 experiments, two to five flexible K⁺ electrodes were inserted into the midmyocardium of the left ventricular anterior free wall (5–6 mm below the epicardial surface) using a plastic catheter introducer as a guide. The electrodes were positioned at various sites in the anticipated ischemic zone and in the anticipated nonischemic zone. In two experiments, two double-barrel K⁺ electrodes were fused together, with their tips positioned 8 mm apart. These electrodes were inserted into the anticipated center of the ischemic zone to measure the subendocardial and subepicardial ak⁺ change simultaneously. In six experiments, the reference barrel of the K⁺ electrode was used to record the local unipolar electrogram versus a central terminal. In eight experiments, an additional reference electrode (0.25 mm o.d.) was positioned within 1 mm of the K⁺ electrode tip, creating a triple-barrel electrode. The two reference barrels were then used to record local bipolar electrograms. Midmyocardial and epicardial temperatures within the anticipated ischemic zone were monitored using Yellow Springs Instruments model 511 (1 mm o.d.) flexible probes and #42112-4 surface probes, respectively. The DC voltage from the intramyocardial K⁺ probes, local ventricular electrograms (50–500 Hz), local myocardial temperatures, and the Y-lead surface ECG (0.05–20 Hz) were simultaneously displayed on an oscilloscope monitor and strip-chart recorder, and recorded on an oscillographic recorder and FM tape recorder.

The K⁺ electrodes were calibrated immediately

![Diagram](http://circ.ahajournals.org/)

**FIGURE 1.** A) Photograph of the double-barrel mini-electrode used to measure changes in extracellular K⁺ activity (ak⁺). The K⁺-sensitive barrel is prepared by dip casting a polyvinyl (PVC)-valinomycin membrane over the tip of a PVC barrel that has been filled with 0.5 M KCl. An identical PVC barrel is filled with 1 M NaCl and fused to the K⁺ barrel using tetrahydrofuran. Ag/AgCl wires are used to measure the DC potential between K⁺ and reference barrel tips. B) Photomicrograph of the mini-electrode tip illustrating the smooth K⁺ sensitive membrane on the K⁺ barrel, the adjacent reference electrode barrel, and the PVC-insulated stainless steel (s.s.) hook used to anchor the electrode in the myocardium. (With permission of the American Physiological Society.¹¹)
before and after each experiment as previously described. If the pre- and postcalibrations differed in any electrode, the data obtained from that electrode were disregarded. The electrodes were also calibrated in situ by injecting 2 mEq KC1 within 15 seconds into the femoral vein. Electrodes were replaced if the maximum rate of rise in response to the K+ bolus was less than 0.5 mM aK+ /min or 0.2 mM aK+ /min different than the other electrodes. They were also replaced if a baseline drift of more than 0.2 mM occurred during the pre-ischemic phase of the experiment. These problems were rarely encountered.

In 18 experiments, a 1.5-mm o.d. K+ electrode was inserted via the jugular vein into the right atrium to monitor systemic K+ activity.

The left anterior descending coronary artery (LAD) was acutely occluded by abruptly tightening the snare. Electrical potentials were recorded continuously throughout the duration of the occlusion and after release. In the five experiments using sinus node crush and atrial pacing, consecutive occlusions of 2–3 minutes duration with an interocclusion period of 45–60 minutes were performed. In these experiments, the pacing rate was either maintained at 90 beats/min (three experiments), changed from 90 to 140 beats/min between occlusions (two experiments), or increased from 90 to 180 beats/min during the occlusion (two experiments). The results are presented in mM K+ activity (aK+) and mM K+ concentration ([K+]) calculated from aK+ using an activity coefficient of 0.746. Statistical analysis was performed using t tests for paired and unpaired data.

**Results**

Insertion of the K+ electrodes into the myocardium caused an increase in the local aK+ to a maximum of 30 mM, which fell rapidly (t½ = 2 minutes) to steady-state levels. The mean difference between the simultaneously measured steady-state myocardial extracellular and intravenous aK+ values was 0.1 ± 0.02 mM. These K+ values remained stable for at least three hours after electrode insertion. After acute coronary occlusion, the myocardium supplied by the distal LAD became cyanotic, ceased to contract and began to bulge. The area of cyanosis was sharply demarcated and ranged from 20–30 cm². Epicardial surface temperature within the ischemic zone decreased 1–3°C within five minutes of the occlusion. However, the midmyocardial temperature recorded at the site of the K+ electrode tip fell 1°C or less during the entire occlusion. Thirteen of the 24 animals developed ventricular extra beats after coronary occlusion; two had short episodes of ventricular tachycardia without ventricular fibrillation, and 11 developed ventricular fibrillation within 2.25–23 minutes. The spontaneous sinus rate, arterial blood pressure, and Y-lead ECG changed little during the occlusion.

**Changes in Local Myocardial K+**

The time course, magnitude and inhomogeneity of changes in myocardial K+ activity induced by acute LAD occlusion are shown in figures 2–6. In figure 2 extracellular aK+ in the center began to rise within 15 seconds of the occlusion, reaching a level of 8.5 mM after 7 minutes ([K+] = 11.5 mM). At the margin of the ischemic zone, the extracellular aK+ rose more slowly and to a lower level (5.5 mM). No changes in aK+ were recorded in the nonischemic zone. After release, the K+ activity at both the center and margin of ischemic zone fell rapidly to levels near control.
In the experiment illustrated in figure 3, the increase in $aK^+$ recorded by the two electrodes positioned in the center of the ischemic zone rose more rapidly and to a higher level than that recorded in the marginal zone. A steady rate was achieved within 5–8 minutes and persisted for approximately 20 minutes. A second, slower rise in $aK^+$ then occurred at all three sites. The $aK^+$ recorded by the electrode located in the center of the ischemic zone reached 14.9 mM ($[K^+] = 20.0 \text{ mM}$) after 60 minutes. Release of the occlusion at 56 minutes failed to reverse the increase in extracellular $aK^+$. Systemic $aK^+$ measured by the $K^+$ electrode in the right atrium did not change by more than 0.2 mM during the entire occlusion period. In the two other experiments of this type, the $aK^+$ reached levels of 19.5 and 21.0 mM ($[K^+] = 26.1$ and 28.0 mM, respectively).

The results from the 22 experiments in which the midmyocardial probes were used are summarized in figure 4. The mean value of the extracellular $aK^+$ in all zones before occlusion was $3.5 \pm 0.1 \text{ mM}$. The difference in control $aK^+$ values recorded at different sites in any given experiment varied from 0.1–0.5 mM. These differences were not statistically significant. The time after occlusion at which $aK^+$ began to rise (figure 4A) was, on the average, less in the center than at the margin of the ischemic zone, although in some experiments similar onset times were observed. The
FIGURE 4. A) Summary of the results from 22 experiments in which midmyocardial extracellular K⁺ activity (aK⁺) was measured after acute occlusion of the left anterior descending coronary artery. These figures illustrate the onset (A), maximum rate (B), magnitude (C) and the plateau level achieved (D) of aK⁺ rise in the center of the ischemic zone (CZ), within 5 mm inside the margin of the ischemic zone (MZ₁), and within 5 mm outside the margin of the ischemic zone (MZ₀). The number of measurements in a given zone is shown on the abscissa. The mean ± SEM of the values in each zone are shown.

maximum rate of rise of aK⁺ (fig. 4B) was significantly greater in the center than at the margin of the ischemic zone. Rates as high as 2.9 mM aK⁺/min were recorded in the center of the ischemic zone. The highest aK⁺ level achieved after 2.75–31 minutes of occlusion, including data from the nine experiments in which early release or ventricular fibrillation precluded the attainment of the plateau phase (fig. 4C), was significantly greater in the center than at the margin, and significantly greater at the inside margin than at the outside margin. The data in figure 4C do not include the aK⁺ values recorded during the slow phase of the K⁺ rise that occurred after the plateau. In 13 experiments, a plateau level was observed (fig. 4D). In summary, the K⁺ changes occurred earlier, rose more rapidly and rose to higher levels in the center of the ischemic zone than at the margins. Our results also indicate that a slight increase in aK⁺ occurs at sites 0.5 cm outside the epicardial cyanotic margin.

The results shown in figures 2–4 indicate that significant inhomogeneities in the rate and magnitude of aK⁺ exist between the center and margin of the ischemic zone. Figures 3, 5, and 6 indicate that inhomogeneities of aK⁺ also existed within the center of ischemic zone. In the experiment shown in figure 5, two midmyocardial K⁺ electrodes were positioned 1

FIGURE 5. Changes in extracellular K⁺ activity (aK⁺) recorded from two midmyocardial electrodes positioned 1 cm apart in the center of the ischemic zone (CZ). Ventricular fibrillation (VF) occurred after 4.25 minutes of occlusion. NZ = nonischemic zone.
cm apart within the center of the ischemic zone. The experiment shown in figure 6 illustrates that the greatest degree of inhomogeneity occurred between the subendocardium and subepicardium of the center of the ischemic zone. The rate of K⁺ rise in the subendocardium was twice as fast as that recorded in the midmyocardium during any other experiment. Similar changes were observed in the other experiment of this type.

Figure 7 illustrates experiments designed to determine the reproducibility of the ischemia-induced changes in aK⁺ and the effect of heart rate on the aK⁺ change. Three occlusions separated by 45 minutes were performed (fig. 7A). The heart rate was 90 beats/min for the first two occlusions. Five minutes before the third occlusion, the heart rate was increased to 140 beats/min. The ischemia-induced change of aK⁺ was the same for all three occlusions. A transient rise in extracellular aK⁺ of 0.1 mM began within 10 seconds of the preocclusion increase in heart rate and lasted for less than 2 minutes. (fig. 7B).

Changes in Local Ventricular Activation

Figure 8 illustrates the bipolar electrograms and changes in aK⁺ recorded simultaneously from triple-barrel electrodes located in the center of the ischemic zone and in the nonischemic zone. In this experiment, slight changes in ischemic zone activation were noted within 1 minute of the coronary occlusion even though aK⁺ had increased by only 0.5 mM. The local activation spikes became progressively delayed and widened during the most rapid phase of the aK⁺ rise, with marked fractionation occurring at 5 minutes. Between minutes 6 and 14, the activation abnormalities lessened. In this interval aK⁺ continued to rise, but at a somewhat slower rate than in the preceding 6 minutes. By 18 minutes, the local activation spike was again more delayed. At this time, the plateau phase of the K⁺ rise had been reached and only slight changes in aK⁺ occurred. The aK⁺ and electrograms recorded from the nonischemic zone were essentially unchanged throughout this period. The changes illustrated in this figure are typical of the changes in the midmyocardium of the center of the ischemic zone recorded in other experiments.

Figure 9 illustrates that the changes in local ventricular activation reflected the inhomogeneity in the rate and magnitude of the changes in aK⁺. The figure demonstrates 2:1 conduction block in the central area.

The changes in activation associated with the acute occlusion were substantially greater than those produced by an equivalent steady-state elevation in aK⁺ (fig. 10). In this experiment aK⁺ was increased to 6.6 mM by the infusion of KC1 into the inferior vena cava and maintained at this level by the constant infusion of KC1 (center panel). The activation spike recorded after the steady state was reached was similar to control (aK⁺ = 3.4 mM). However, the rapid increase in aK⁺ to 6.6 mM induced by ischemia resulted in a marked delay in the local activation spike.

Ventricular fibrillation occurred in 11 experiments. In four it occurred during the phase of rapid K⁺ rise, in six during the plateau phase, and in one during reperfusion. None of the swine developed ventricular fibrillation during the phase of slowly rising aK⁺ observed during longer occlusion periods (three experiments). Although the changes in local ventricular activation paralleled the midmyocardial aK⁺ rise, there was no correlation between the rate or magnitude of the aK⁺ rise and the incidence of ventricular fibrillation.

Discussion

In the experiments of Harris et al. and in similar experiments by other investigators, [K⁺] determinations were performed on venous blood draining the ischemic zone or on blood drawn from the coronary sinus. Many of the [K⁺] values were determined during coronary occlusion and indicated that the vein was draining a portion of normally or partially perfused myocardium. Concentration values of up to 6.4 mM (an activity approximately equivalent to 4.8 mM) were reported after 5–10 minutes of occlusion. These rather modest elevations were thought to reflect significant accumulations of K⁺ in the extracellular space within the ischemic zone. Downar et al. studied
**Figure 7.** The effect of heart rate on the changes in midmyocardial extracellular $K^+$ activity ($a_K^+$) after acute occlusion of the left anterior descending coronary artery. A) Consecutive occlusions at the same (1,2) or increased (3) heart rate produced nearly identical rates of rise in extracellular $a_K^+$. Note the rapid fall in $a_K^+$ after release of the occlusion (arrow R). B) Increasing heart rate during an occlusion. Three minutes after occlusion the heart rate was increased from 90 to 180 beats/min. The rate of rise in $a_K^+$ did not increase but began to slow as in other experiments of constant heart rate. Ventricular fibrillation (VF) occurred at 5 minutes. CZ = ischemic zone; NZ = nonischemic zone.

**Figure 8.** A) Tracings of midmyocardial bipolar electrograms recorded from triple-barrel $K^+$ electrodes positioned in the center of the ischemic zone (CZ) and the nonischemic zone (NZ) during a 22-minute occlusion. The activation delay recorded from the ischemic zone progressively worsened after 2 minutes of occlusion, but was less severe after 6 minutes, even as the $a_K^+$ continued to rise. B) The corresponding time course of midmyocardial rise in extracellular $K^+$ activity. Ventricular fibrillation (VF) occurred after 22 minutes of occlusion.
this relationship in pig hearts, in which there was no flow during the occlusion in the vein draining the ischemic zone. They measured the K⁺ concentration of the first 2–4 ml of venous effluent collected upon release of a 10–15 minute coronary occlusion and found values of 4.6–16.2 mM (aK⁺ = 3.4–12.1 mM), with a mean of 8.0 mM (aK⁺ = 6.0 mM).

The K⁺ electrodes used in our experiments provide the means of determining directly the magnitude and time course of the changes in aK⁺ during acute ischemia at various locations within and without the ischemic zone. These experiments indicate that there are three distinct phases of change in aK⁺ after the acute ligation of a coronary artery. The first phase begins within 15 seconds of occlusion and lasts for 4–15 minutes. It is a period of rapid K⁺ increase in which aK⁺ rises as rapidly as 2.9 mM aK⁺ (3.9 mM K⁺) per minute to values as high as 12.4 mM ([K⁺] = 16.6 mM). The midmyocardial mean aK⁺ value of 7.6 ([K⁺] = 10.6 mM) is higher than reported by Dow-  

![Figure 9](http://circ.ahajournals.org/)

**Figure 9.** Heterogeneous changes in local ventricular activation recorded from the tip of midmyocardial K⁺ electrodes after left anterior descending coronary artery occlusion. Corresponding values of extracellular K⁺ activity (aK⁺) and the calculated equivalent [K⁺] are shown. A) Tracings of bipolar electrograms from electrodes positioned in the center (CZ) and inside margin (MZ₁) of the ischemic zone. By 2 minutes there is a significant delay in activation at CZ but only a slight delay at MZ₁. By 3 minutes, the activation delay at MZ₁ is more pronounced, but at CZ a marked fractionation of the electrogram and a 2:1 block in activation has occurred. B) Tracings of unipolar electrograms recorded from two electrodes positioned 1 cm apart in the center of the ischemic zone (CZ). Between 1.5 and 2.5 minutes, K⁺ activity rose faster and to a higher level at CZ₁. This was paralleled by a more marked delay in the local activation spike recorded at CZ₁ (arrows).
FIGURE 10. Comparison of the change in local ventricular activation when the steady-state extracellular K⁺ activity (aK⁺) is increased by intravenous infusion of KCl (middle panel) and when the same level of aK⁺ occurs during ischemic (lower panel). The traced electrogram during the K⁺ infusion was essentially unchanged from control (upper panel). The traced electrogram recorded when aK⁺ increased to the same level, 2.25 minutes after left anterior descending coronary artery occlusion, showed significant conduction delay.

The time course of the initial phase of aK⁺ change correlates well with the changes in extracellular PCO₂ recently described by Case et al. They reported that the rise in PCO₂ began within 7 seconds of acute coronary ligation. It is likely that the K⁺ changes we observed occur equally rapidly (figs. 5 and 6). However, for reasons that are not obvious, the rise in PCO₂ did not have a plateau phase. In this respect, the changes in extracellular aK⁺ may be a more useful index of ischemia because the end of the plateau may identify the end of the period of reversibility.

Our results indicate that significant inhomogeneities in the rate and magnitude of aK⁺ change exist between the center and lateral margins, between the endocardium and epicardium, and even in the central midmyocardial region of the ischemic zone. The fastest rise in aK⁺ was recorded in the subendocardium (fig. 6). These inhomogeneities are consistent with results obtained from microsphere studies of local perfusion and from studies of the anatomic, biochemical and electrical changes induced by ischemia. As such, our results provide additional evidence consistent with the concept of a border zone or zones. However, our results do not indicate whether the border is due to juxtaposed normal and ischemic cells or cells with varying degrees of ischemia because each would produce similar findings.

The possibility that changes in electrode response characteristics may have contributed to the inhomogeneities of K⁺ is unlikely because the characteristics of the electrodes were not altered by the experiments. The possibility that heterogeneous changes in muscle temperature might have influenced our results was also unlikely because no evidence of such heterogeneity was recorded in the midmyocardium where the temperature fell 1 degree or less throughout the occlusion period and the changes in epicardial temperatures were considered in the calculations of epicardial aK⁺. The possibility that a K⁺-independent change in DC electrical potential might have occurred between adjacent electrode tips was also considered and found not to exist.

Our study does not permit insight into the cause of the ischemia-induced rise in extracellular aK⁺. The possibilities include alterations in membrane-bound Na⁺-K⁺-ATPase, a change in membrane permeability to K⁺, or a H⁺-K⁺ exchange. Nor do our results establish a causal relationship between the changes in aK⁺ and ventricular activation. It is reasonable to assume that some of the conduction slowing, especially that seen during the plateau and slowly rising phases, is related to the changes in resting potential, action potential upstroke and conduction velocity induced by the change in the aK⁺.

However, three pieces of evidence support the conclusion of Downar et al., that changes in ventricular activation during the initial phase of the aK⁺ rise may not be the result of the magnitude of change in aK⁺ per se. These include: 1) Marked changes in local activation occurred when aK⁺ was 4–6 mM ([K⁺] = 5.8–8.0 mM), levels of K⁺ associated with either no change or even a slight speeding of conduction; 2) The delay in activation recorded during the occlusion-induced rapid rise of aK⁺ was more marked than that associated with similar steady-state levels of aK⁺ induced by the systemic infusion of KCl (fig. 10). 3) The local activation delay associated with the rapid rise in aK⁺ became less marked even though aK⁺ continued to increase slightly or did not change. The more marked changes in aK⁺ in the subendocardium might have caused the changes in electrical activity recorded in the midmyocardium. It is also possible that intramyocardial Purkinje fibers were more sensitive to changes in aK⁺ than the myocardial fibers or that the rate of the ischemia-induced aK⁺ rise contributed to the observed changes in activation. Also, other factors, such as changes in intra- and extracellular pH, PCO₂, PO₂ and the extracellular accumulation of metabolic end-products, should be considered. It is unlikely that the difference in activation observed during the infusion-induced and ischemia-induced changes in aK⁺ could be attributed to changes in intracellular aK⁺ because, on the one hand, it has been shown that increasing extracellular aK⁺ does not in-

![Heartbeat Diagram](image-url)
crease intracellular aK+ \(^{47}\) and on the other, that the ischemia-induced rise in extracellular aK+ can be accounted for by a 1–3% decrease in intracellular aK+.\(^{48}\) This change would make an insignificant difference in the extracellular-intracellular K\(^+\) gradient induced by a 100–200% change in extracellular aK+. The transient rise in extracellular aK+ that we recorded in the normal myocardium after the abrupt increase in heart rate is consistent with recent observations using K\(^+\)-sensitive microelectrodes.\(^{49, 50}\) However, the change in aK+ in the ischemic zone during the rapidly rising phase was independent of heart rate, perhaps because the rate of change in aK+ was too great to be influenced by the increase in heart rate. Thus, the rate-dependent slowing of conduction within the acutely ischemic zone observed by others\(^{51}\) cannot be attributed to a more rapid accumulation of extracellular K\(^+\). Rather, the rate-dependent changes probably reflect the prolonged recovery of the action potential upstroke\(^{48}\) and of conduction velocity that occurs in K\(^+\) depolarized fibers.\(^{48}\)

We could not correlate the development of ventricular fibrillation to the changes in midmyocardial aK+. The magnitude of the inhomogeneities in aK+ and activation throughout all areas of the ischemic zone may be more important than the individual changes we recorded. The ability to record the changes in aK+ and activation simultaneously from more areas than was possible in this study should permit more accurate assessment of these inhomogeneities and perhaps the identification of the factors critical to the development of ventricular fibrillation.

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Transluminal Angioplasty: Correlation of Morphologic and Angiographic Findings in an Experimental Model

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SUMMARY The morphologic consequences of transluminal angioplasty of stenotic atherosclerotic coronary arteries are unknown. This study describes the production of aortoiliac atherosclerosis in rabbits and reports the morphologic changes after transluminal angioplasty of stenotic arterial lesions. Atherosclerotic lesions were evaluated angiographically before and after transluminal angioplasty and were studied histologically and by electron microscopy after angioplasty. Moderately stenotic aortic segments showed demudation of endothelial cells and deposition of a carpet of platelets enmeshed in fibrin. Medial and intimal changes were not seen. Intimal plaque disruption and splitting of atheromatous plaques were observed in more stenotic vessels where dilatation during angioplasty is relatively greater. Transluminal angioplasty, therefore, acutely causes desquamation of endothelial cells and superficial plaque elements, splitting of atheroma and subsequent deposition of platelets and fibrin in the area of angioplasty. This experimental model may be useful to evaluate the morphologic changes after angioplasty and might be used in further studies to determine the long-term pathophysiological changes after transluminal angioplasty.

RECENT STUDIES by Grünzig¹,² indicate that percutaneous coronary transluminal angioplasty with a balloon-tipped catheter is effective in the treatment of stenotic coronary artery disease in humans. In follow-up, coronary angiograms of patients treated by this technique show improved lumen diameter at the angioplasty site, thallium-201 perfusion images reveal fewer myocardial defects and patients are improved symptomatically.¹,²

Scanning electron microscopy after coronary transluminal angioplasty in normal dog coronary arteries has been studied.³ However, the morphologic basis of angiographically successful transluminal angioplasty of a stenotic atherosclerotic artery is virtually unknown. Studies performed on human hearts at autopsy show that angioplasty may lead to plaque rupture and medial dissection.⁴ ⁵ However, at autopsy, the tissue is not viable and therefore is more susceptible to damage than vessels in vivo, and passage of shorter dilation catheters may have caused dissection if forced through fixed stenotic segments.
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