Regional Changes in Intracellular Potassium and Sodium Activity after Healing of Experimental Myocardial Infarction in Cats

Shinichi Kimura, Arthur L. Bassett, Marion S. Gaide, Patricia L. Kozlovskis, and Robert J. Myerburg

SUMMARY. After healing of experimental myocardial infarction in cat hearts, endocardial cells demonstrate persistent regional electrical changes. These include long action potential duration in surviving cells over the infarct scar, and short action potential duration and low membrane potential in border zone cells between the scar and normal tissue. We studied the basis for these electrophysiological changes by measuring intracellular potassium and sodium activity with ion-sensitive microelectrodes in normal, border, and infarct zone cells of the cat left ventricle 2-6 months after ligation of multiple distal tributaries of the left anterior descending and circumflex coronary arteries. In normal zone cells, intracellular potassium activity was 89.6 ± 12.3 mM (mean ± SD, n = 9), and sodium activity was 10.8 ± 2.2 mM (n = 5). Neither was significantly different from infarct zone cells (91.2 ± 15.0 and 10.5 ± 3.0 mM, respectively). In contrast, border zone cells had significantly reduced intracellular potassium activity (71.4 ± 5.1 mM, P < 0.01, compared to normal and infarct zone cells) and increased intracellular sodium activity (19.1 ± 5.9 mM, P < 0.05, compared to normal and infarct zone cells). The membrane potential in border zone cells was more positive to calculated potassium equilibrium potential, and less sensitive to the change in the extracellular potassium concentration in the range between 2 and 10 mM, compared to normal and infarct zone cells. Sixty minutes of exposure to 5 x 10⁻⁷ M ouabain reduced the membrane potential and intracellular potassium activity to a lesser degree in border zone cells than in normal and infarct zone cells (P < 0.05), but the percent changes from the initial values were not significantly different among the three zone cells. We conclude that action potential changes in infarct zone cells are not accompanied by changes in intracellular potassium and sodium activities, while border zone cell changes are accompanied by reduced intracellular potassium activity and increased intracellular sodium activity. Our data also suggest that border zone cells have altered potassium and/or sodium conductances. (Circ Res 58: 202-208, 1986)

EXPERIMENTAL myocardial ischemia produces both acute and long-term cellular electrophysiological abnormalities (Samson and Scher, 1960; Friedman et al., 1973; Downar et al., 1977; Myerburg et al., 1977; Lazzara et al., 1978). Among the earliest changes—beginning almost immediately after coronary ligation—are loss of resting membrane potential, decreased action potential amplitude, shortening of action potential duration, and decreased maximum upstroke velocity of phase 0 of the transmembrane action potential. Whereas these changes may abate in cells that do not progress to infarction (Friedman et al., 1975), our studies have demonstrated that cellular electrophysiological abnormalities persist long-term after healing from acute myocardial infarction (Myerburg et al., 1977; Wong et al., 1982). The action potentials recorded from endocardial cells overlying a discrete healed myocardial infarction scar show normal resting membrane potentials and upstroke velocity with prolonged action potential durations, whereas those recorded from the cells at the border between the infarct and surrounding normal tissue have low resting membrane potentials and short action potential durations.

The mechanisms responsible for the different types of action potential abnormalities in border and infarct zones have yet to be determined. The present study was designed to measure intracellular K⁺ and Na⁺ activity in surviving cells overlying the healed infarction scar, in cells at the border between scar and surrounding tissue, and in cells in normal areas, with ion-sensitive microelectrodes. In addition, we examined the effects of ouabain and extracellular K⁺ concentration on the intracellular K⁺ activity and membrane potential in an attempt to characterize the electrical properties of these different zones.

Methods

Preparations and Solutions

Conditioned adult domestic cats, weighing 2.4-3.5 kg, were anesthetized with sodium pentobarbital (30 mg/kg, ip). Acute myocardial infarction was created by single...
stage ligation of multiple distal tributaries of the left anterior descending and left circumflex coronary arteries. This procedure predictably produces an infarction of 5–15% of the left ventricular muscle at the base of the anterior papillary muscle and adjacent areas of the apex, apical free wall, and lower septum (Myerburg et al., 1977). The chest was closed after coronary ligation. Surgical mortality was less than 15%. The surviving cats were maintained in a colony for 2–6 months. On the day of terminal studies, the healed myocardial infarction cats were anesthetized with sodium pentobarbital (30 mg/kg, ip), and the heart was removed through a thoracotomy. The atria and right ventricle were excised, and the left ventricle was opened in cool, oxygenated Tyrode’s solution by an incision through the free wall between the posterior papillary muscle and posterior paraseptal free wall. The left ventricular preparations were placed with endocardial surface up in a Lucite tissue bath, and were superfused with warm (37°C) Tyrode’s solution equilibrated with 95% O₂ and 5% CO₂. The composition of Tyrode’s solution was (in mm): NaCl, 129; KCl, 4; NaHCO₃, 20; NaH₂PO₄, 1.8; MgCl₂, 0.5; CaCl₂, 2.7; dextrose, 5.5. The pH of the superfusate was 7.35.

### Electrical Stimulation and Recording

Driving stimuli at a cycle length of 800 msec were delivered to the left bundle branch through bipolar Teflon-coated silver wire electrodes. Pulse duration was 2 msec and current intensity was 2 times late diastolic threshold. Transmembrane action potentials were recorded by conventional microelectrode techniques. Glass microelectrodes, filled with 3 m KCl (resistance 10–30 MΩ), were connected through Ag-AgCl junctions to a high impedance electrometer (WPI, model FD-223). Ion-sensitive microelectrodes used for the measurements of intracellular K⁺ and Na⁺ activity also were connected to the input stage of the same electrometer. The amplifier output was displayed on dual beam oscilloscopes (Tektronix, 564 and 565) and photographed on Polaroid film and recorded on a polygraph (Grass, model 79).

### Fabrication and Calibration of K⁺ and Na⁺ Electrodes

K⁺-sensitive microelectrodes were made from filament-borosilicate glass. Micropipettes were exposed to the vapor of dimethylchlorosilane (Fluka), then were baked at 200°C for 1 hour. The potassium liquid ion exchanger (WPI, IE-190), which is 2% (wt/vol) potassium tetrakis (p-chlorophenyl) borate [(K(5-Cl)₄)₄B] in nitroxyolene, was introduced into the tip of the electrodes and the shaft was filled with 0.5 M KCl. Standard techniques were used to calibrate the K⁺ electrodes before and after use (Lee, 1981). The slope of the K⁺ electrode response ranged from 22–26 mV/e-fold increase in K⁺ activity (50–60 mV/10-fold increase). The selectivity coefficient (κ_{KNa}), as determined by the method using single-electrolyte solutions, ranged from 0.010–0.034. Since we studied in five additional preparations in which aK was not measured, for determining a_{Na}. Na⁺ electrodes were calibrated in mixtures of NaCl and KCl at a constant ionic strength (NaCl + KCl: 1 + 149, 3 + 147, 10 + 140, 30 + 120, 100 + 50 mm) and a_{Na} was determined from the calibration curve [unorthodox method (Thomas, 1978)].

Ion activity coefficients for calibrating single electrolyte solutions were taken from Shedlovsky (1950) and those for Tyrode’s solution were calculated using the equation of Pitzer and Mayorga (1973). The calculated K⁺ and Na⁺ activity coefficients of the Tyrode’s solution were 0.74 and 0.76, respectively.

### Experimental Protocol

After a 60-minute equilibration period, transmembrane action potentials were recorded from endocardial muscle cells of normal areas of the isolated left ventricle, and from the cells of overlying and bordering areas of healed myocardial infarction in the same heart. Grids were constructed to provide consistent sites for sampling from normal, border, and central infarct myocardial tissues; this sampling technique was used in detail elsewhere (Myerburg et al., 1977, 1982). Using conventional microelectrodes, specific regions of interest were identified that showed the characteristic action potentials of normal, border, and infarct zones as previously reported (Myerburg et al., 1977, 1982; Wong et al., 1982). After indentification of these three zones, we sampled cells within a small recording area (0.5 × 0.5 mm), where cells were distributed evenly, in each of the three zones for measurements of both membrane potentials (V_m) and K⁺ electrode potentials (V_k) or Na⁺ electrode potentials (V_na). Such small recording areas usually had cells with almost identical action potential configurations and resting potentials. V_k or V_na was obtained from the same recording area where V_m was recorded. We did not observe significant inhomogeneities of action potential configurations and resting potentials in any of the recording areas chosen for study. Even so, in order to reduce the error of potential measurements, we sampled 10 cells within the recording area for each measurement of V_m, V_k, or V_na, and the average potential obtained from the 10 cells was used to calculate a single value of a_k or a_{Na}. If the response time of the ion-sensitive electrode exceeded the cycle length, the stimulation was stopped to read the potentials (the potential reached a steady state within a second in most experiments). In a second series of experiments, we examined the change in a_{Na} after exposure to ouabain, in order to estimate Na⁺-K⁺ pump function of the cells in normal, border, and...
Figure 1. Representative recordings of transmembrane action potentials from normal, border, and infarct zones of healed myocardial infarction. RMP = resting membrane potential. APD
 = action potential duration measured at 90% repolarization. Note that the border zone cells have low resting membrane potential and short action potential duration, whereas cells overlying the infarct scar have long action potential duration.

infarct zones. After control measurements of Vm and V\textsubscript{K} in specific regions of different zones, the preparations were superfused with 5 \times 10^{-7} \text{M} ouabain (Sigma) for 60 minutes, and the measurements were then repeated.

Finally, we characterized the response of Vm to changes in the K\textsuperscript{+} concentration of the superfusate. Five preparations were exposed to a series of Tyrode's solutions with increasing K\textsuperscript{+} concentrations, ranging from 2-40 mM. Vm was recorded from cells in the normal, border, and infarct zones during superfusion with each K\textsuperscript{+} concentration in the series, after it had been determined that the potential had stabilized at its new level following a change in K\textsuperscript{+} concentration. The K\textsuperscript{+} concentration was raised by adding the required amount of K\textsuperscript{+} as 2.0 mM KCl directly to the Tyrode's reservoir without compensation for osmotic strength or Donnan conditions.

Statistical Analysis

All data are presented as mean ± SD, and were evaluated for statistical significance by analysis of variance with repeated measures, or unpaired t-test, where appropriate. Differences with P values < 0.05 were considered significant.

Results

Pale retracted scars were distinctly visible on the endocardial surface of the left ventricle in the preparations studied 2-6 months after coronary artery ligation. The infarcts involved the base of the anterior papillary muscle, plus variable involvement of the apex and the interpapillary portion of the free wall of the left ventricle. Histological characteristics have been described in previous reports from this laboratory (Myerburg et al., 1977; Wong et al., 1982).

Representative transmembrane action potentials recorded from normal, border, and infarct zones are shown in Figure 1. As previously reported (Myerburg et al., 1977; Wong et al., 1982), transmembrane action potentials recorded from border zone cells are characterized by short action potential durations and decreased membrane potentials. In contrast, surviving cells overlying the infarction scar showed long action potential durations and normal resting membrane potentials. These data are summarized in Table 1.

Intracellular K\textsuperscript{+} and Na\textsuperscript{+} Activity in Normal, Border, and Infarct Zone Cells

Representative recordings of Vm and V\textsubscript{K} obtained after identification of specific regions of normal, border, and infarct zone cells by their characteristic action potentials are shown in Figure 2A. The mean values of Vm and the calculated values of \(a_k\) and E\textsubscript{K} obtained from nine preparations are summarized in Table 2. \(a_k\) was 89.6 ± 12.3 mM (mean ± SD) in normal, 71.4 ± 5.1 mM in border, and 91.2 ± 15.0 mM in infarct zone cells. \(a_k\) was significantly lower in border zone cells than in normal and infarct zone cells (P < 0.01). The calculated values of E\textsubscript{K} were -90.4 ± 3.6 mV in normal, -84.7 ± 2.0 mV in border, and -90.9 ± 4.5 mV in infarct zone cells. Vm was more positive to E\textsubscript{K} in border zone cells than in normal and infarct zone cells (P < 0.05).

Table 1

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Border</th>
<th>Infarct</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMP (-mV)</td>
<td>79.3 ± 1.5</td>
<td>68.9 ± 2.2\textsuperscript{*}</td>
<td>79.2 ± 1.6</td>
</tr>
<tr>
<td>APA (mV)</td>
<td>107.9 ± 3.5</td>
<td>89.5 ± 6.3\textsuperscript{*}</td>
<td>108.2 ± 3.9</td>
</tr>
<tr>
<td>APD\textsubscript{50} (msec)</td>
<td>85.4 ± 10.6</td>
<td>54.5 ± 12.1\textsuperscript{*}</td>
<td>116.2 ± 11.6\textsuperscript{*}</td>
</tr>
<tr>
<td>APD\textsubscript{90} (msec)</td>
<td>130.1 ± 12.4</td>
<td>95.9 ± 14.7\textsuperscript{*}</td>
<td>169.0 ± 19.8\textsuperscript{*}</td>
</tr>
</tbody>
</table>

\(n = 11\). Data are presented as mean ± SD. RMP = resting membrane potential. APA = action potential amplitude. APD\textsubscript{50} and APD\textsubscript{90} = action potential duration measured at 50% and 90% repolarization, respectively.

\* Significant differences from the values in normal zone cells (P < 0.01).

\textsuperscript{*} Significant differences from the values in infarct zone cells (P < 0.01).
Figure 2. Panel A: representative recordings of membrane potential (Vm) and potential recorded by the K⁺-sensitive electrode (Vₖ) from normal, border, and infarct zones. Panel B: representative recordings of potential recorded by the Na⁺-sensitive electrode (Vₜₙ₉) from normal, border, and infarct zones. Vₙ₉ in normal, border, and infarct zone cells was -124 mV, -102 mV, and -126 mV, respectively, when the potential measured in 100 mM NaCl + 50 mM KCl was set as zero. Recordings in panels A and B were obtained from different preparations.

Table 2

<table>
<thead>
<tr>
<th>Vm (mV)</th>
<th>aK (mM)</th>
<th>Eₖ (mV)</th>
<th>Vm-Eₖ (mV)</th>
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</thead>
<tbody>
<tr>
<td>Normal</td>
<td>79.0 ± 0.9</td>
<td>89.6 ± 12.3</td>
<td>90.4 ± 3.6</td>
</tr>
<tr>
<td>Border</td>
<td>68.3 ± 0.9†</td>
<td>71.4 ± 5.1†</td>
<td>84.7 ± 2.0†</td>
</tr>
<tr>
<td>Infarct</td>
<td>79.2 ± 1.0</td>
<td>91.2 ± 15.0</td>
<td>90.9 ± 4.5</td>
</tr>
</tbody>
</table>

n = 9. Data are presented as mean ± sd. Vm = membrane potential, aK = intracellular potassium activity. Eₖ = calculated potassium equilibrium potential.

† Significant differences from the values in normal zone cells (P < 0.01).

*a Significant differences from the values in normal zone cells (P < 0.001).

†† Significant differences from the values in normal zone cells (P < 0.05).

Effects of Ouabain on aK in Normal, Border, and Infarct Zone Cells

We carried out a series of 12 experiments to determine the effects of ouabain on Vm and aK in normal, border, and infarct zone cells. In an initial series of three experiments, preparations were exposed to 10⁻⁶ ouabain for 60 minutes, using the method of Browning et al. (1981). However, all preparations became inexcitable within 60 minutes exposure to 10⁻⁶ M ouabain, making it difficult to measure aK. In a second series of nine experiments, we lowered the ouabain concentration to 5 × 10⁻⁷ M, and the measurements were made 60 minutes after superfusion with the drug. Since rapid sustained ventricular activity occurred in three of nine preparations, the analyzable data on the effect of ouabain were limited to six stable preparations.

As summarized in Table 4, 60 minutes of exposure to 5 × 10⁻⁷ M ouabain reduced Vm and aK in cells from all three zones. Reduction of absolute values of Vm and aK in border zone cells was significantly smaller than that in normal and infarct zone cells (P < 0.05, see Fig. 3). However, the percent change from the values obtained before exposure to ouabain was not significantly different among the three study sites. Percent changes of other action potential parameters by ouabain also were not significantly different among three zone cells (Fig. 4).

Effects of Extracellular K⁺ on Vm in Normal, Border, and Infarct Zone Cells

The results in the previous section demonstrated that Vm was more positive to EK in border zone cells than in normal and infarct zone cells. This finding suggests that altered steady state K⁺ and/or Na⁺ conductance, in addition to low aK, may underly the low membrane potential in border zone cells. To test this hypothesis, we studied the relationship between Vm and extracellular K⁺ concentration in cells from the three zones. In Figure 5, Vm was expressed by K⁺ activity rather than K⁺ concentration to estimate aK from the curves. Since infarct zone cells had K⁺...
Table 3

<table>
<thead>
<tr>
<th>Cell Type</th>
<th>Normal</th>
<th>Border</th>
<th>Infarct</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vm (mV)</td>
<td>64.3 ± 5.2</td>
<td>79.2 ± 1.0</td>
<td>60.7 ± 3.3</td>
</tr>
<tr>
<td>aiK (mM)</td>
<td>10.8 ± 2.2</td>
<td>19.1 ± 5.9</td>
<td>10.5 ± 3.0</td>
</tr>
</tbody>
</table>

n = 5. Data are presented as mean ± sd. aiK = intracellular sodium activity.

* Significant differences from the values in normal zone cells (P < 0.05).

† Significant differences from the values in infarct zone cells (P < 0.05).

Discussion

This study was designed to seek explanations for our previous findings that there are dissimilar types of long-term action potential abnormalities recorded from cells overlying infarct zones and border zones of healed myocardial infarctions in cats (Myerburg et al., 1977; Wong et al., 1982). The action potentials recorded from central infarct zone cells have normal resting membrane potentials and action potential amplitudes, but long action potential durations. In contrast, the action potentials recorded from the border between the infarct and surrounding normal tissue show low resting membrane potentials and short action potential durations.

Since the dissimilar types of action potential abnormalities may reflect different long-term electrophysiological consequences of ischemia and its healing process in the different areas, it is important to consider the mechanisms that may underlie these changes. The results of the present study demonstrated that aiK was indeed different in the infarct and border zone cells. The value of aiK in ventricular muscle cells from normal zones was similar to that previously reported for normal cat papillary muscle cells (Browning et al., 1981). In addition, aiK in infarct zone cells was similar to that in normal zone cells. However, aiK was significantly lower in border zone cells than in normal and infarct zone cells. Thus, the depolarization in border zone cells is partially explained by a less than normal aiK. aiK indirectly estimated from the relation between Vm and extracellular K+ activity (Fig. 5) was also different for normal and border zone cells, although aiK measured with the K+ microelectrodes was slightly smaller than extrapolated aiK. This difference might stem from experimental errors.
The relationship between Vm and EK in border zone cells was also different from that in normal zone cells. Vm was more positive to EK in border zone cells than in normal zone cells. This finding suggests that a decreased K+ conductance, an increased Na+ conductance, or both, is also involved in the depolarization in border zone cells. This conclusion is supported by the finding that border zone cells did not function well as a K+ electrode at extracellular K+ activity of 1.5–7.3 mM (2–10 mM [K+]), as shown in Figure 5.

Reduction of the Na+-K+ pump activity results in an increase in aK, and a decrease in aNa, and thus membrane potential becomes less negative. It has been reported that [Na+] is increased and [K+] is decreased during prolonged hypoxia (8 hours) in guinea pig papillary muscles (McDonald and MacLeod, 1973; Baumgarten et al., 1981). We found that, in addition to low aK, aNa was increased in border zone cells, suggesting that their Na+-K+ pump activity is depressed. However, our data on the effect of ouabain suggest that Na+-K+ pump activity is, to some extent, still present in border zone cells. An increased Na+ leak also may explain the increase in aNa in border zone cells.

Border zone cells have short action potential durations. Under hypoxic conditions, shortening of action potential duration has been explained by the increased background K+ current (Vleugels et al., 1980). The mechanism underlying shortening of action potential duration in the border zone cells of the healed infarct preparations is unclear. However, an increased aNa may produce intracellular Ca++ accumulation via the Na+-Ca++ exchange system, as in the case of cardiac glycosides. There is evidence which suggests that an increase in intracellular Ca++ concentration reduces the slow inward current by decreasing Ca++ gradient at the plateau phase (Reuter, 1973; Gibbons and Fozzard, 1975) and activates K+ outward current at the repolarization phase (Ise-enberg, 1975, 1977). Thus, a change in intracellular Ca++ concentration, if one occurs, may explain why border zone cells have short action potential durations.

In infarct zone cells, aK and aNa were the same as in normal zone cells. In addition, the experiments with ouabain and changes in extracellular K+ concentration, designed to differentiate Na+-K+ pump activity level and electrical properties, demonstrated no difference in the responses between normal and infarct zone cells. This finding indicates that infarct...
zone cells have normal function of Na⁺-K⁺ pump and normal steady state K⁺ conductance. This is not surprising, since infarct zone cells had normal resting membrane potentials. However, the prolongation of action potential duration in infarct zone cells cannot be explained by this approach. The mechanisms involved in this phenomenon may be quite different.

As previously mentioned, it has been demonstrated that the change in intracellular Ca²⁺ concentration affects the action potential duration (Isenberg, 1975, 1977). Although we did not measure intracellular Ca²⁺ activity in this study, the infarct zone cells could have Ca²⁺ concentrations that are different from those of normal zone cells.

A more likely possibility is electrical uncoupling. Increased internal resistance and electrical uncoupling of myocardial cells have been demonstrated during hypoxia and acute ischemia (Wojcik, 1979; Childers et al., 1979). The elevation of free Na⁺ and Ca²⁺ in the cytosol increases junctional resistance and causes cell-to-cell uncoupling (De Mello, 1975, 1976). This might be the case in the border zone cells that overlie the scar, and thus infarct zone cells may be electrically isolated from both the surrounding and deeper layer cells. Our previous studies (Myerburg et al., 1977; Wong et al., 1982) have shown histologically that there is interdigitation of surviving muscle cells and infarct scars, which lead us to suggest that a complex pattern of electrical uncoupling may exist in the healed infarct preparations. Altered electronic interaction (Mendez et al., 1969) may be involved in the prolongation of the action potential duration in infarct zone cells. However, the experiments in this study did not directly address the question of cell-to-cell uncoupling.

In conclusion, cellular electrophysiological abnormalities in the border zone between healed myocardial infarction and normal tissue are, at least in part, caused by low ac, high aK, and altered steady state K⁺ and/or Na⁺ conductances. The infarct zone cells do not share these specific abnormalities, and their cellular electrophysiological abnormalities must therefore be explained by other mechanisms.

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