体 重 0.42 ± 0.09 kg 小 兔 8 只。将 兔 置 于 带 有 放 射 性 标 记 的 供 氧 环 境 中， 连 续 记 录 小 兔 血 压 变 化 和 心 率 变 化。心 电 图 采 用 普 通 电 置 记 录 仪 记 录。血 压 使 用 水 压 计 直 接 测 定。结 果 表 明，进 行 体 外 芳 酸 转 移 激 活 可 以 有 效 地 减 少 小 兔 的 血 压 波 动。
至AP复极至80%所用时间（APD_{80}）AP幅度（AP）2。部分性缺血致心肌梗死，观察Nic，Ver，Iso对AP的影响，测定5个参数AP_{max}，APD_{max}，APD_{min}，APD_{r1}，APD_{r3}，正常心肌缺血组：观察Nic对AP的AP_{max}，APD_{max}，APD_{min}的影响，用药前恢复和在一周内随机取样进行，经放大后校验，数据处理采用配对t检验。

实验结果：Nic北京制药厂；Iso北京制药厂；Ver苏联ORI厂。

结果
Nic，Iso及Ver对窦房结细胞AP的影响
1. 标本以Nic 33 mmol/L灌流15 min后，与用药前相比，AP_{max}增加33%（P<0.001），APD_{max}缩短5%（P<0.01），APD_{min}缩短15%（P<0.001），V_{max}及APA无显著变化（图2，表1）。
2. 标本以Iso 0.15 mmol/L灌流5 min后，与用药前相比，AP_{max}增加55%（P<0.001），APD_{max}缩短29%（P<0.05），APD_{min}缩短41%（P<0.05），V_{max}增加141%（P<0.05），APA无显著变化（图2，表1）。
3. 标本以Ver 0.22 mmol/L灌流10 min后，与用药前相比，AP_{max}增加43%（P<0.001），APD_{max}缩短35%（P<0.001），APD_{min}缩短33%（P<0.05），V_{max}下降80%（P<0.05），APA下降15%（P<0.05）（图2，表1）。

Tab 1. Effects of nicotineamide, isoprenaline and verapamil on slope of phase 4, sinus cycle length, action potential duration at 90% repolarization, action potential amplitude, and V_{max} of phase 5 of action potential of SA node cells, x̄±SD, *P<0.05, **P<0.01

<table>
<thead>
<tr>
<th></th>
<th>Nic</th>
<th>Iso</th>
<th>Ver</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>15</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>SCL (ms)</td>
<td>54±17</td>
<td>46±12</td>
<td>48±11</td>
</tr>
<tr>
<td>APD_{max} (ms)</td>
<td>173±9</td>
<td>63±8</td>
<td>62±3</td>
</tr>
<tr>
<td>APA (V)</td>
<td>8±5</td>
<td>8±8</td>
<td>8±3</td>
</tr>
<tr>
<td>V_{max} (V/ms)</td>
<td>2</td>
<td>3</td>
<td>2</td>
</tr>
</tbody>
</table>

图2. Effect of nicotineamide, isoprenaline and verapamil on action potential of SA node cells, a) control, b) after drugs.

Nic，Iso，Ver对部分性缺血致心肌梗死的影响
1. 标本以Nic 33 mmol/L灌流30 min后，与用药前相比，AP_{max}下降21%（P<0.01），APD_{max}延长3%（P<0.05），APD_{min}延长4%（P<0.05），V_{max}及APA无显著变化（图3，表2）。
2. 标本以Iso 0.15 mmol/L灌流15 min后，与用药前相比，AP_{max}增加82%（P<0.01），APD_{max}延长16%（P<0.01），APD_{min}延长15%（P<0.05），APA增加11%（P<0.01），V_{max}无显著变化（图3，表2）。
3. 标本以Ver 0.22 mmol/L灌流30 min后，与用药前相比，AP_{max}下降23%（P<0.01），APD_{max}缩短7%（P<0.01），APD_{min}缩短6%（P<0.01），APA无显著变化（图3，表2）。

图3. Effects of nicotineamide, isoprenaline and verapamil on action potential of SA node cells, a) control, b) after drugs.
Table 2: Effects of nicotinamide, isoprenaline and verapamil in \( V_{max} \) of fast phase and slow phase

<table>
<thead>
<tr>
<th>( n )</th>
<th>( V_{max} ) (V/m)</th>
<th>( V_{max} ) (V/m)</th>
<th>APDmax (ms)</th>
<th>APDmax (ms)</th>
<th>APA (mV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nic</td>
<td>control</td>
<td>70 ± 22</td>
<td>19 ± 6</td>
<td>158 ± 17</td>
<td>172 ± 14</td>
</tr>
<tr>
<td></td>
<td>30 min</td>
<td>55 ± 18**</td>
<td>19 ± 5</td>
<td>165 ± 12**</td>
<td>179 ± 11</td>
</tr>
<tr>
<td>Isop</td>
<td>control</td>
<td>54 ± 11</td>
<td>22 ± 4</td>
<td>160 ± 14</td>
<td>170 ± 10</td>
</tr>
<tr>
<td></td>
<td>15 min</td>
<td>61 ± 11*</td>
<td>40 ± 7***</td>
<td>180 ± 25**</td>
<td>180 ± 25</td>
</tr>
<tr>
<td>Verap</td>
<td>control</td>
<td>72 ± 13</td>
<td>22 ± 6</td>
<td>163 ± 18</td>
<td>176 ± 18</td>
</tr>
<tr>
<td></td>
<td>30 min</td>
<td>74 ± 14*</td>
<td>37 ± 4**</td>
<td>152 ± 16***</td>
<td>164 ± 19</td>
</tr>
</tbody>
</table>

Fig 5: Effects of nicotinamide, verapamil and isoprenaline on action potentials of guinea pig ventricular papillary muscle. Each point is the mean of 3 experiments.

Fig 4: Effects of nicotinamide on action potentials of normal guinea pig papillary muscle cell. a) control, b) 15 min after nicotinamide.

Nicotinamide 11% (p<0.01), APDmax 10% (p<0.01), APA 

Discussion

Electric field stimulation reveals, in isolated guinea pig (L) atrial myocardium, that the action potential of a single atrial cell is determined by the height of the atrial muscle. In our experiments, the atrial muscle is stimulated with a stepwise increase in stimulus intensity (SP). At a stimulus intensity of 100%, the APDmax and the effect on the action potential of the atrial muscle are maintained. The application of nicotinamide (Nic) decreases the APDmax by 10% (p<0.01), APA by 10% (p<0.01). The effects on the APDmax and APA of the atrial muscle are not significant different.

In the presence of 3 x 10^-6 M nicotinamide (Nic), the APDmax is decreased by 10% (p<0.01), APA by 10% (p<0.01) compared to the control. The effects of nicotinamide on the APDmax and APA of the atrial muscle are not significant different.

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Effects of nicotineamide on action potentials of sinusoidal node cell and depolarized papillary muscle cell of guinea pig

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ABSTRACT The effects of nicotineamide (Nic) on action potentials (AP) of guinea pig SA node cell, normal papillary muscle cell and papillary muscle cell depolarized by high K+ were studied. Nic 33 mmol/L increased the slope of phase 4 of AP of SA node cell by 43 %; APD90 of normal papillary muscle cell was shortened by 10 %; Vmax of phase 0 of AP of depolarized papillary muscle cell was not much changed. Vmax of phase 0 of AP of depolarized papillary muscle cell was decreased by 21 %.
excitability of the depolarized papillary muscle was inhibited. Verapamil 0.22 μmol/L decreased the slope of phase 4 and $V_{\text{m}}$ of AP of SA node cell respectively by 44% and 88%. $V_{\text{m}}$ of AP of the depolarized papillary muscle cell was decreased by 25%; $V_{\text{m}}$ was not significantly changed.

The results suggest: 1) There may be a difference between $I_{\text{Na}}$ in the phase 4 of SA node AP and $I_{\text{Na}}$ in the phase 0 of SA node AP and $V_{\text{m}}$, of AP of the partially depolarized papillary muscle cell. 2) The inhibition of Nc on excitability of myocardium may be related to the effect on Na+ channel.

KEY WORDS: nicotinamide, isoproterenol, verapamil, sinoatrial node, papillary muscles, action potentials