speeds using limb leads.

Reperfusion was carried out in 28 cases. Three cases died of ventricular fibrillation (VF) during coronary occlusion. Twenty seven of the 28 cases developed reperfusion arrhythmia (sustained ventricular tachycardia (VT) 15, non-sustained VT 5, VF 2, and frequent ventricular premature contraction (VPC) 5).

Coronary reocclusion was performed within 3 minutes after reperfusion (early coronary reocclusion) in 10 cases. Sustained VT in 8 cases and frequent VPC in 2 cases all disappeared after reocclusion in 1 to 75 seconds.

Coronary reocclusion was performed in 9 cases between 20 and 30 minutes (late coronary reocclusion). Sustained VT after reperfusion in 4 cases which were temporarily suppressed by lidocaine bolus injection and reappeared in 15 to 20 minutes all disappeared in 18 to 100 seconds after reocclusion. Non-sustained VT in 4 cases all disappeared after reocclusion.

These results demonstrate that coronary reocclusion has an interrupting effect on reperfusion arrhythmia.

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A STUDY OF THE ARRHYTHMOGENIC MECHANISM IN HYPOKALEMIA

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The arrhythmogenic mechanism in dogs with hypokalemia was investigated in relation to the effect of α -1 blockade. Hypokalemia was induced by insertion of ion-exchange resin into colon. After the premedication of 60 min, 10 μ g/kg of epinephrine was injected intravenously. Heart mitochondria (Mt) was prepared 5 min after the epinephrine injection and Mt Ca^{2+} content was measured. The severity of arrhythmias was expressed by the arrhythmic ratio (the percentage of ventricular ectopic beats divided by the total heart rate) during 5 min after the epinephrine injection. In the hypokalemia group, dogs with the arrhythmic ratio of over 10% were observed 9 out of 17. However, in the control group ($n=13$) which was maintained normal serum K^+ level, only one dog showed the arrhythmic ratio of over 10%. Furthermore, dogs with the lower serum K^+ levels showed the higher arrhythmic ratio. Ca^{2+} content of heart Mt, which is considered to be the reflection of intracellular Ca^{2+} concentration, was 24.4 ± 5.7 nmoles/mg protein in the control group 5 min after the epinephrine injection, while that from the hypokalemia group was increased to 35.0 ± 13.4 . A good reciprocal correlation between the concentration of serum K^+ just before the epinephrine injection and Mt Ca^{2+} content was observed in the hypokalemia group ($r=-0.79$), while in the control group, no correlation was observed. Clear correlation between Mt Ca^{2+} content and the arrhythmic ratio was also observed in the hypokalemia group. Pretreatment of bunazosin, an α -1 blockade, prevented effectively the increase in Mt Ca^{2+} content, and reduced the arrhythmic ratio. These results indicate that, in hypokalemia, a relatively small dosis of epinephrine, which scarcely effects the dogs maintained with normal serum K^+ level, could induce the severe ventricular arrhythmias probably due to the Ca^{2+} influx to myocardium through α -1 adrenergic responsiveness.

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171-176 M. Arita, *Oita*
177-181 J. Toyama, *Nagoya*
182-186 M. Hiraoka, *Tokyo*
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DESENSITIZATION OF ACh-INDUCED K^+ CURRENT IN ATRIAL CELLS OF THE GUINEA-PIG HEART

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Acetylcholine (ACh) increases K^+ current (I_{ACh}) in atrial tissues. Although it has been generally described that muscarinic increase of K^+ current does not desensitize during continuous application of ACh in cardiac tissues, we found that I_{ACh} does desensitize. Here, we characterize the desensitization phenomenon of I_{ACh} in single atrial cells of the guinea-pig. The tight-seal whole cell clamp technique was used. The pipette solution contained 100 μ M GTP and 1 mM MgCl_2 . Various concentrations of ACh were applied in the bath solution. ACh 10^{-8} - 10^{-4} M increased I_{ACh} dose-dependently. I_{ACh} evoked by ACh more than 10^{-6} M decreased with time after initial activation. The time-course of the decay could be divided into two exponential components. The time-constants of both components were not affected by the membrane potential but depended on the concentrations of ACh. The I_{ACh} in the steady state was 67% (10^{-8} M ACh), 53% (10^{-5} M ACh) & 43% (10^{-4} M) of the peak current, respectively. When 10^{-5} M ACh was applied after perfusion of 10^{-6} M ACh, the peak current evoked by 10^{-5} M ACh decreased to 68% of the control, which was coincident with the decrease of I_{ACh} during perfusion of 10^{-6} M ACh. We interpret this phenomenon as representing the desensitization of muscarinic receptors by ACh.

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ACETYLCHOLINE AND ADENOSINE RECEPTORS LINK WITH A K^+ CHANNEL VIA GTP-BINDING PROTEINS IN CARDIAC ATRIAL CELL MEMBRANE

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Both acetylcholine (ACh) and adenosine (Ado) reduce the heart rate and atrio-ventricular conduction by increasing a K^+ conductance of cardiac cell membrane. To clarify the molecular mechanisms underlying the effects of ACh and Ado, we examined the mode of regulation of K^+ channel