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DECREMENTAL MODE OF ELECTROTONIC POTENTIAL IN THE BEATING RABBIT SINUS NODE STRIP AS MEASURES OF ELECTRICAL COUPLING

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Sinus node is known to consists of many weakly coupled pacemaker cells whose automaticity could be easily affected by subthreshold stimuli.

To study the relationship between electrical coupling and pacemaker synchronization, we have used the sucrose gap technique for current pulse application to thin (0.5 x 5 mm), spontaneously beating rabbit sinus node strips recording the transmembrane potentials with microelectrodes. In six preparations in which the intra sinus node phase differences were brief (<20 msec), electrotonic potential induced by 50 msec pulse applied at various times during the diastole declined with distance from the gap, as would be expected for a linear cable, but increased gradually during the depolarization. Consequently, space constant increased from $469 \pm 133 \mu\text{m}$ at the maximum diastolic potential, to $746 \pm 197 \mu\text{m}$ just before threshold was reached ($p < 0.02$). These pulses induced phase-related changes in pacemaker cycle length whose magnitude was the same at proximal and distal sites of the strip. There were no pacemaker shifts. In 2 other strips, phase differences in the strips were relatively long (>20 msec), electrotonic potentials declined sharply along the limited area where the phase delay was marked. This suggests the presence of decreased electrical coupling along those areas. Under these conditions, current pulses induced phase related changes that were much smaller in distal portion than proximal portion. As a result, alteration of the dominant pacemaker was observed.

Conclusion: Gradual increment of the space constant during the diastole reflects the increase of the membrane resistance. Response of the sinus node strip to the external perturbation is a good indicator of the electrical coupling and pacemaker shift may be induced due to the differential sensitivity to the perturbation in the inhomogeneous strip.

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THE ELECTRICAL EFFECTS OF ACTIVATION COLLISION IN HEART MUSCLE

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We studied the electrical effects of action potential (AP) collision in isolated cardiac canine Purkinje strands (PS; n=7) and cat papillary muscles (PM; n=9). The effects of collision were obtained by comparing APs during collision and during one-way conduction including quantitative changes in the measured indexes of AP depolarization and repolarization. In normal tyrode solution ($[K^+]_o = 4.4 \text{ mM}$), APs during collision showed significantly increased maximum rate of rise of upstroke (V_{max}) and significantly decreased AP duration (APD) as follows;

	PS	PML	PMT
ΔV_{max} v/s	152±40	93±30	95±42
% change	20.5	61.0	49.4
ΔAPD , msec	-0.9±0.3	-2.3±0.9	-1.9±1.3
% change	-0.33	-1.47	-1.27

Δ ; collision - one-way, %; Δ /one-way, L; longitudinal, T; transverse
The spatial extent of the collision which was confined by mapping was narrow region at and on each side of collision site within 1 mm (PS>PML>PMT), however, in PML and PMT there was no

difference in the peak magnitude of the changes. In $[K^+]_o = 11.0 \text{ mM}$, the extent of % change of V_{max} and APD increased significantly in both preparations more than normal tyrode solution in accordance to the slowing of conduction velocity. These findings revealed that repolarization was affected by electrotonic effects by changing activation sequence. AP collision is a common occurrence in heart. Under certain circumstances, the resulting collision-induced changes may significantly contribute to the electrical inhomogeneities which are thought to be involved in the formation of cardiac arrhythmias.

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PROTECTIVE EFFECT OF TAURINE AGAINST DECLINE OF CARDIAC SLOW ACTION POTENTIALS DURING HYPOXIA

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Taurine (2-aminoethanesulphonic acid) is found in high amounts in mammalian hearts, constituting about 50% of the free amino acid pool of that organ. It is generally accepted that taurine is lost from the heart subjected to hypoxic or ischemic insult, although the mechanism of this phenomenon has not been established. The effect of taurine on cardiac slow action potentials (APs) during hypoxic superfusion was studied in isolated guinea-pig papillary muscles. Transmembrane potentials were recorded with conventional microelectrodes filled with 3M KCl. Ca^{2+} -dependent slow APs were induced by isoproterenol (10^{-6} M) in preparations which were voltage-inactivated by high (25mM) K^+ . Hypoxia was produced by switching to a solution gassed with a mixture of 95% N_2 - 5% CO_2 . The isoproterenol-induced slow APs were depressed or abolished during hypoxia, and hypoxic superfusion for 30 min resulted in the significant decrease of myocardial taurine content. Taurine (10mM) treatment exerted a significant protective effect against the decline in all parameters of the slow APs (amplitude, duration at 50% depolarization, and the maximum rate of rise) during hypoxic superfusion. On the other hand, taurine had no effect on the slow AP parameters during normoxia. Taurine also restored slow APs that had been previously abolished by hypoxia. These results suggest that taurine may have an important physiological role in maintaining the cardiac cell function under hypoxic condition.

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THE FACILITATORY EFFECT OF SODIUM SALICYLATE ON QUINIDINE-INDUCED FAST Na^+ CHANNEL BLOCK IN GUINEA-PIG VEN- TRICULAR MUSCLE

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The facilitatory effect of sodium salicylate on quinidine-induced rate-dependent, time-independent