Effects of tryptamine on mechanical and electrical activities of sino-atrial frog auricular trabeculae

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The effects of tryptamine on ionic currents were studied in frog atrial fibres by means of a double sucrose gap technique associated with a transducer RCA 5734 for the recording of mechanical tension.

Under current clamp conditions, tryptamine (5×10^{-6} g/ml), without modifying the membrane resistance, caused a significant increase in the slow phase of depolarization and an increase in plateau duration. The rate of rise of the action potential was not affected, but tryptamine produced a marked increase in contraction strength. When rapid inward current was blocked by tetrodotoxin and external calcium was removed, the inotropic effect was still observed. In sodium-free medium, it disappeared.

Under voltage clamp conditions, calcium inward current and the phasic component of the contraction were not modified. In calcium-free medium, the increase in contraction amplitude may be related to the important rise of slow sodium inward current. Unlike the description by Horackova & Vassort (1974), our observations show that variation of the contraction strength responds, beat to beat, to the variation of slow inward sodium current.

These results suggest that the inotropic property of tryptamine is mainly due to an increase of the slow inward current and that sodium induces calcium release.

Reference

HORACKOVA, M. & VASSORT, G. (1974). Excitation contraction coupling in frog heart—Effect of Veratrine. *Pflügers Archiv. für Physiol.*, **352**, 292–302.

Autonomous control of the heart, myocardial blood flow and S-T segment in canine ischaemic myocardium

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The effects of cardiac parasympathetic stimulation and of bilateral stellectomy on regional myocardial blood flow (measured by tracer microspheres) and the S-T segment were investigated in normal and ischaemic regions on a reproducible model of temporary coronary occlusion in the dog.

The control coronary occlusion did not modify heart rate but induced in the ischaemic area an increase in S-T segment and a decrease in endo- (−71%) and epicardial (−49%) blood flows, resulting in a diminished endo/epi ratio (ranging from 0.40 ± 0.04 to 0.56 ± 0.08 as compared with 0.97 ± 0.02 to 1.04 ± 0.04 in the non-ischaemic region).

Bilateral stellectomy decreased heart rate (−33%), lowered S-T segment elevation (−62%) and further reduced endo- (−35%) and epicardial (−30%) blood flows in the ischaemic area without inducing redistribution (endo/epi ratio $= 0.52 \pm 0.05$ as compared with 0.56 ± 0.08). Endo- and epicardial blood flows were also reduced (−40%) in non-ischaemic areas and endo/epi ratio was not modified.

Vagal stimulation (30 Hz, 2 ms, 3 V) decreased heart rate (−16%) and S-T segment elevation (−30%) and increased slightly but non-significantly endo- and epicardial blood flows (+11%) and endo/epi ratio (+5%) in ischaemic areas. However, at 70 Hz, vagal stimulation still reduced heart rate (−28%) and S-T segment elevation (−44%) but significantly increased endo (+108%) and epicardial (+92%) blood flows resulting in an endo/epicardial blood flow redistribution (endo/epi ratio: 0.56 ± 0.03 as compared with 0.40 ± 0.04, $P < 0.01$). These effects of vagal stimulation at high frequencies were maintained under electrosystolic pacing of the heart but abolished under combined electrosystolic pacing and atropine administration.

It is concluded that (1) there is no correlation between regional blood flows and endo/epi ratio evolution in ischaemic areas on the one hand and bradycardia on the other hand, although there is correlation between decrease in heart rate and lowering of S-T segment, (2) the redistribution phenomenon observed after vagal stimulation at high frequencies is probably related to a muscarinic effect of acetylcholine.