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of the left anterior descending and right coronary arteries, were excised from 23 autopsied cadavers within 8 hours after death, and were sectioned into 2mm ring segments. They were preserved in physiological saline solution saturated with a mixture of 95% O₂ and 5% CO₂ (37±0.5°C). The developed tension was recorded isometrically. Y-19638 and atropine showed an antagonistic action against acetylcholine-induced contractions and the pA₂ value was 6.64 and 9.20, respectively (P<0.001). Nitrendipine did not show an anticholinergic action. Y-19638 as well as nitrendipine inhibited 3X10⁻⁷M prostaglandin F₆ and 30μM potassium-induced contractions. Estimated 50% inhibitory concentration against potassium contraction was 6.9x10⁻⁷M for Y-19638 and 1.6x10⁻⁴M for nitrendipine. Both spontaneous and prostaglandin F₆-induced, periodic contractions were disappeared by the application of Y-19638 and nitrendipine. Atropine did not inhibit periodic contractions. In conclusion, Y-19638 is a novel compound which has both effects of anticholinergic and calcium blocking actions on the isolated human coronary artery.

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EFFECTS OF PARATHYROID HORMONE ON THE CARDIOVASCULAR SYSTEM

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It has been well accepted that the bone and the kidney are the principal organs of parathyroid hormone (PTH) actions, but there has been little work on the cardiovascular system. We evaluated the effects of PTH on the cardiovascular system of rats. In thiobutabarbital anesthesitized rats, synthetic bovine parathyroid hormone, containing the amino acid (bPTH 1-34) in dose of 0.1-10 μg/kg iv, caused dose related decrease in mean arterial blood pressure (MAP), left ventricular pressure (LVP), left ventricular end diastolic pressure (LVEDP) and LV dp/dt max, increase in heart rate (HR) and LV dp/dt/p. After the administration of 10 μg/kg, PTH decreased the MAP from 104.3 to 55.5 mmHg, LVP from 122.1 to 96.4 mmHg, LVEDP from 6.70 to 6.37 mmHg and LV dp/dt max from 5684 to 4735 mmHg/sec. The HR and LV dp/dt/p increase from 399.7 to 410 bpm, 95.5 to 108.4/sec, respectively. Propranolol, phentolamine, atropine and promethazene did not affect the these actions of PTH. On the basis of this findings, we conclude that PTH has the directory vasodepressive action and augmentation of the contractile force on the cardiovascular system.

Chairman

131-136 M. Iizuka, Tokyo
137-142 Y. Hirota, Takatsuki

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A STUDY ON FRANK STARLING CURVES CONTRUCTED IN HUMANS

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We constructed Frank Starling curves (stroke work index vs end diastolic volume index) by changing preload using the lower body positive and negative pressure method and investigated if these curves were justified. Subjects are seven patients with heart disease. We changed lower body pressure (Px mmHg) to P5 = 20, P10 = 10, P3 = 0, P2 = -20, P1 = -40 mmHg and made hemodynamic measurements at the end of each step. End diastolic volume index and stroke work index increased by 29% and 88% from P1 to P5, respectively. Heart rate and systemic vascular resistance increased at P1 and at P2 compared with those at P3, but changes in serum norepinephrine concentration were not significant. End systolic pressure volume ratio decreased significantly only at P5.

We concluded that we obtained Frank Starling curves at almost constant contractility using the lower body positive and negative pressure method.

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THE SECOND COMPONENT OF LEFT VENTRICULAR SYSTOLIC PRESSURE

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Arterial and left ventricular systolic pressures are divided into two components by the anacrotic notch. The first component is mainly caused by left ventricular ejection and the second component by the peripheral reflection wave. This study is based on the hypothesis that the second component of left ventricular systolic pressure is the result of an excessive ventricular afterload. Thirty five patients, 15 with myocardial infarction, 10 with angina pectoris and 10 others, took part in this study. The pressure in the left ventricle and at the base of the ascending aorta were measured by means of micromanometer-tipped catheter in the subjects' normal condition and after an intravenous injection of Angiotensin and, in 14 cases, a sublingual administration of Nitroglycerin. Aortic reflection wave ratio(AoWR)/(late peak systolic pressure - pulse pressure) X 100 (%). Left ventricular reflection wave ratio(LVWR)=(late peak systolic left ventricular pressure - late ventricular pressure at anacrotic notch)/(late peak systolic left ventricular pressure) X 100 (%). Mean AoWR was 32.4 ± 12.7 % (± SD) and LVWR 11.4 ± 7.8 %. AoWR(X) and LVWR(Y) correlated well (Y = 0.36X - 0.34, r = 0.76, p<0.001), and the drug load and degree of change were both directly proportional. LVWR was higher in the ischemic heart disease patients than in the others. LVWR was greater in myocardial infarction patients given Angiotensin. In the patients given Nitroglycerin, the decrease of second component was largely responsible for the decrease in LVWR.

The ratio of the second component to the increase in left ventricular pressure, is considered to be an useful index of excessive left ventricular afterload.