will be required to adjust the contraction of long axis direction when compared at end systolic and diastolic phase and some fixed points such as bifurcations and stenosed points of coronary arteries will be useful as a natural land marks. As a conclusion, this new technique will be useful for evaluating a cardiac hemodynamics in spite of existing some limitations.

Chairman

160-165 K. Hiejima, *Tokyo*

166-170 H. Mashiba, *Yonago*

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EFFECTS OF ISCHEMIA ON THE ENDOCARDIAL ACTIVATION SEQUENCE OF CANINE LEFT VENTRICLE

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We have measured conduction time in Purkinje fibers (PCT) and endocardial muscles (MCT) of left ventricles by using isolated canine hearts with coronary artery perfusion and endocardial superfusion. A PCT was defined as the time interval between the Purkinje fiber activation times at the base of left bundle branch and at the Purkinje-ventricular muscle junction (PMJ), and a MCT as the time interval between the endocardial muscle activation times at the PMJ and the apex of anterior papillary muscle. The prolongation of MCT (234±99 % of the control) was more than that of PCT (123±15 % of the control) at a 30-min cessation of coronary artery perfusion. Coronary artery perfusion with "ischemia-like solution" (PO₂ 20-40 mmHg, pH 6.8 and K⁺ 10 mM) produced the similar conduction delay to that when the coronary artery perfusion was ceased. On the contrary to the previous cases where endocardium was superfused with the normal solution, endocardial superfusion with "ischemia-like solution" with cessation of coronary artery perfusion prolonged the PCT as well as the MCT. These results suggest that (1) in the acute phase of myocardial ischemia, excitation conduction of Purkinje fibers is preserved if endocardial superfusion with intracavitary blood is maintained, despite the deterioration of endocardial muscle activation sequence, and that (2) a combination of hypoxia, acidosis and hyperkalemia may play a major role on the conduction delay of endocardial muscle.

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ROLE OF THE CENTRAL SYMPATHETIC SYSTEM IN THE DEVELOPMENT OF TORSADE DE POINTES

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The purpose of this study was to investigate whether the central sympathetic system that originates in the ventromedial hypothalamic nuclei (VMN) was related to the occurrence of torsade de

pointes. Forty-eight male Wistar rats, 21-27 w of age (270-395g), were used in this study. Animals were classified into 3 groups: 27 control rats, 11 VMN-lesioned rats and 10 sham-operated rats. Torsade de pointes was induced by the administration of ouabain. The following doses of ouabain were given i.p. at 30 min intervals; 5, 10, 15, 20, 25, 30, 35, 40, 45, 50 and 55 mg/kg. Bilateral VMN were lesioned passing DC current of 3mA for 30 sec under urethane anesthesia. Repetitive episodes of torsade de pointes were observed following marked intra-ventricular conduction defects (IVCDs) and frequent multiform ventricular arrhythmias in the doses of ouabain from 10 to 45 mg/kg in 25 of the 27 control rats, while in the VMN-lesioned rats, the IVCDs and ventricular arrhythmias were suppressed significantly and torsade de pointes episodes appeared in only 3 of the 11 rats. Ouabain-induced prolongation of the RR and QT intervals was not suppressed, but enhanced in the VMN-lesioned rats. These results suggest that the central sympathetic system is strongly related to the occurrence of torsade de pointes, and that the underlying electrophysiologic mechanism is altered automaticity with several arrhythmic foci and/or the presence of multiple reentrant circuits in accordance with abnormal local conduction blocks.

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INTRAMYOCARDIAL ACTIVATION PATTERNS OF VENTRICULAR FIBRILLATION INDUCED IN THE LATE MYOCARDIAL INFARCTION

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We studied epicardial and intramural isochronal maps of ventricular activation during ventricular fibrillation (Vf) by programmed premature stimulation in 25 mongrel dogs that weighed 10-14 kg, alive for 3-5 days after acute myocardial infarction. In 13 dogs epicardial potentials were recorded simultaneously from 48 electrodes and in 12 dogs intramural potentials were recorded simultaneously from 8 transmural needles consisted of 7 electrodes. These were analyzed using a computerized multiplexing technique.

During epicardial mapping, 6 pleomorphic ventricular tachycardia (PVT) and 10 Vf were induced. During intramural mapping 5 PVT and 9 Vf were induced. Epicardial isochronal maps from PVT which rapidly degenerated into Vf showed multiple irregular arcs of functional conduction block, and which revealed the marked changes in the arcs of block and in the circulating wavefronts at the border of infarcted zone. On the other hand, intramural isochronal maps from PVT which rapidly developed into Vf followed by slow conduction and functional conduction block of activation wavefronts, and which were observed, at first, in the epicardial site and then observed in the endocardial site.

This study suggests that Vf may occur when antecedent PVT accompanied by the marked changes in the geometry of functional conduction block and the circulating wavefronts.