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Programmed electrical stimulation and epicardial mapping were performed to study electrophysiological mechanisms for induction of nonsustained polymorphic ventricular tachycardia (PVT) in 30 dogs with 7-day old myocardial infarction. Nine dogs had 15 episodes of reproducible monomorphic (M) VT. Seven episodes of PVT were induced in 6 dogs, 3 of which degenerated into ventricular fibrillation. Epicardial conduction delay during premature stimulation at the time of VT induction was more marked in MVT than in PVT (183 ± 43 vs 123 ± 31 msec, $p < 0.01$). Induction of MVT was preceded by the development of functional conduction block in 13 of 15, while in 3 of 7 PVT. During PVT, epicardial activation was radial from the earliest activation site, similar to that observed during left ventricular endocardial pacing. The QRS polymorphism was associated with a beat-to-beat variation of the earliest epicardial activation site. Recordings of endocardial electrograms revealed local delay or block and the early endocardial activation preceding epicardial activation during PVT.

In conclusion, inducibility of PVT depends less on the epicardial activation compared to MVT. Intramyocardial reentry involving the Purkinje network is suggested as a mechanism of PVT.

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SIMULTANEOUS EPICARDIAL AND ENDOCARDIAL MAPPING DURING INDUCED VENTRICULAR TACHYCARDIA

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Reproducible ventricular tachycardia (VT) was induced by a programmed double or triple premature stimulation technique in 5 dogs with 7-day-old myocardial infarction (%MI: $1.0 - 17.3$, 10.8 ± 6.3). Epicardial mapping using a 4×5 cm patch electrode containing 48 bipolar electrodes failed to show a continuous epicardial circus movement in 7 of these VT. A total of 48 bipolar plunge electrodes were impaled subendocardially (ENDO) and subepicardially (EPI) for simultaneous mapping.

Results: Temporal relationship between the earliest and latest activation in ENDO and EPI, and the presence of functional conduction block enabled to locate reentry circuit in 2 of 7 VT. During a premature stimulus preceding one VT, epicardial functional block developed and a slow epicardial circuitous excitation wave activated the central infarct area late in diastole, while the earliest activation during the first induced beat was located in the subendocardium immediately underneath the maximal epicardial delay. Thus, a reentry circuit was suggested to involve both deep intramyocardium and EPI. In another VT, location of functional block and delay and the earliest activation changed between EPI and ENDO in an alternate beat. In other 5 VT, both EPI and ENDO activations were radial from the earliest activation sites and reentrant circuits could not be identified, while one of these was suggested to be due to microreentry in ENDO since one

ENDO electrode located closely to the earliest ENDO activity revealed continuous fragmented activity during VT.

In conclusion, a simultaneous EPI and ENDO mapping was useful to understand mechanisms of some of the induced VT in a infarction model.

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SIGNAL-AVERAGED ELECTROCARDIOGRAMS IN PATIENTS WITH VENTRICULAR TACHYCARDIA -FREQUENCY ANALYSIS OF QRS COMPLEX BY REVERSED-FOURIER TRANSFORM-

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Frequency analysis of QRS complex was studied in 9 patients either with sustained ventricular tachycardia (s-VT) or with ventricular fibrillation (VF, group I) and in 11 patients without them (group II). Signal averaged surface electrograms during sinus rhythm were analysed by Fourier transform and by reversed-Fourier transform with several frequency ranges. In each frequency range, ratio of the area (S), which was compared to that of original QRS complex, and peak time (T) in reversed-Fourier transformed QRS complex were compared between two groups. In the range of 40-80Hz, S in group I (0.095 ± 0.045) was significantly larger than that in group II (0.050 ± 0.034 , $P < 0.05$). And the frequency components of this range were more contained within early two-thirds of QRS complex in group I than in group II. There was no significant difference in T between two groups.

It was suggested that the difference in frequency components in the range of 40-80Hz may have relation to fragmented electrograms, which were observed in patients with s-VT using endo- or epicardial mapping. In conclusion, the frequency components of 40-80Hz in patients with s-VT or VF were larger than that in patients without them. Frequency analysis of signal averaged electrogram was useful to evaluate VT not only about the terminal portion of QRS complex but also about total QRS complex.

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USEFULNESS OF ELECTROPHYSIOLOGIC-PHARMACOLOGIC TESTING TO PREDICT LONG-TERM EFFICACY OF ANTIARRHYTHMICS FOR PREVENTION OF RECURRENT SUSTAINED VT

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Usefulness of electrophysiologic-pharmacologic testing (EPT) to predict long-term efficacy of antiarrhythmics for prevention of recurrent sustained ventricular tachycardia (RSVT) was investigated with clinical follow-up.

Study populations consisted of 26 RSVT patients (pts.) in whom clinical VT was inducible by programmed ventricular stimu-

lation (PVS). Underlying diseases were coronary artery disease (17), mitral valve disease (2), dilated cardiomyopathy (1), ARVD (1), and unknown (5).

Base-line electrophysiologic study was performed with PVS using 1-3 (maximum RVRs ≤ 5) was defined as effective drug identified.

According to EPT, 26 pts were classified into 14 (54%) with effective drug identified (EPS-S) and 12 unidentified (EPS-U). There were no differences between 2 groups in clinical, hemodynamic and electrophysiologic backgrounds. The mean number of ineffective drugs given were 2.0 in EPS-S and 3.8 in EPS-U. During a mean follow-up of 14 (6-38) months, in EPS-S sudden cardiac death (SCD) or SVT occurred in 5 pts (36%) including 1 within 6 months. In EPS-U, SCD or SVT occurred in all the 4 pts except 8 pts with amiodarone given, within 2 months in 2 pts. Positive and negative predictive value and predictive accuracy of EPT were 86%, 50% and 78% within 6 months and 64%, 100% and 72% over 6 month.

EPT appears to have a limited value for predicting long-term drug efficacy, however to be useful for confirming drug unefficacy.

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PROGNOSIS OF VENTRICULAR TACHYCARDIA

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We examined the clinical features and prognostic implications of 80 patients with ventricular tachycardia (VT: $\geq$ triplets) detected mainly with 24 hour ambulatory electrocardiograms with a mean follow up of 27 ± 40 months.

The underlying heart disease was ischemic heart disease in 29 patients (36%); cardiomyopathy in 17 patients (21%); valvular heart disease in 2 patients (3%); other heart disease in 3 patients (4%); and apparently normal heart in 29 patients (36%).

There were 16 deaths (20%) during the follow-up period, including 2 noncardiac deaths. Eight of the 14 cardiac deaths were sudden and the remaining 6 patients died of uncontrollable heart failure. The mortality rates (cardiac death) in the groups of diseased heart and normal heart were 28% (14/50) and 0%, respectively ($p < 0.01$). The value of left ventricular dimension at end-diastole and ejection fraction on echocardiograms of the nonsurvivors (cardiac death) and the survivors were 64 ± 11 mm and 54 ± 10 mm ($p < 0.005$), $34 \pm 11\%$ and $55 \pm 9.9\%$ ($p < 0.005$), respectively. Ten of 24 patients with sustained VT (≥ 30 seconds) suffered cardiac death compared with 4 of 56 patients with non-sustained VT ($p < 0.01$). No other characteristics of VT including episode per day, longest run of VT, rate, morphologic characteristics (bundle branch block pattern, monomorphic or polymorphic), or premature ventricular contraction per day was important prognostically. Further, administration of effective drugs on the basis of 24 hour ambulatory electrocardiograms was not a good predictor of survival ($P : NS$).

Thus, underlying heart disease, left ventricular dysfunction, dilated left ventricle, or sustained VT were the predictors of mortality in patients with VT.

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PREVENTIVE EFFECT OF PROPAFENONE ON VENTRICULAR TACHYCARDIA IN A CANINE MODEL OF MYOCARDIAL INFARCTION -A COMPARATIVE STUDY WITH LIDOCAINE-

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Using programmed electrical stimulation and simultaneous epicardial mapping, we examined electrophysiologic effects of propafenone and lidocaine in dogs with 7-day-old myocardial infarction. Induction of ventricular tachycardia (VT) was prevented by intravenous propafenone (2 mg/kg) in 6 of 8 dogs, while in one of 6 dogs by lidocaine of a same dose ($p < 0.05$). Epicardial conduction velocity within the infarction zone (IZ) during basic drive and the second extra-stimulus beats was reduced by both drugs to the same extents. Effective refractory period in the IZ was lengthened by propafenone ($14 \pm 10\%$, $n=9$, $p < 0.01$), while not by lidocaine ($2 \pm 5\%$, $n=7$, NS). Propafenone interrupted the activation wave fronts of reentry circuit that developed in response to programmed stimulation and was located in the surviving epicardial layer within the IZ by abolishing the re-excitation of the proximal site of the arc of functional unidirectional block. On the other hand, lidocaine could not interrupt the circling wave fronts of reentry circuit and failed to prevent the initiation of VT.

In summary, our findings suggest that higher effectiveness of propafenone than lidocaine in prevention of VT induction is attributed to its more potent effect on refractoriness of the myocardium within the IZ.

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STUDIES TO DEVELOP IMPLANTABLE "CCU"; EFFECTIVENESS AND SAFETY FOR DETECTING OF VENTRICULAR TACHYARRHYTHMIA BY CARDIOMOVIOGRAM

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We have attempted to develop the implantable CCU for prevention of sudden cardiac death due to ventricular tachyarrhythmia. In this study, We evaluated effectiveness and safety for detecting of ventricular tachyarrhythmia by Cardiomovio-gram.

Methods: We recorded intracardiac Cardiomovio-gram (CMG) of variable experimental ventricular arrhythmia by handmade Cardiomovio-gram. We evaluated changes of CMG induced by coronary artery occlusion, and β -blocker agent, and change of recording site.

Results: i) In Ventricular fibrillation (Vf), the amplitude of CMG decreased and regularity of CMG was lost. Within 30 seconds after onset of Vf, amplitude of CMG decreased to less than 50%. While 60 seconds after onset of Vf, we could terminate Vf by handmade endocardial defibrillator. ii) In Ventricular tachycardia