have a close relation with the thickness and position of FTs, and be due to mechanical stretching of FTs or Purkinje fibers contained within them.

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PATHOLOGICAL SUBSTRATUM FOR MYOCARDIAL FATTY INFILTRATION AND SUSTAINED VENTRICULAR TACHYCARDIA

Keisuke Fukuda, Kazuyuki Takada, Kikuo Arakawa

Second Internal Medicine, Fukuoka University School of Medicine

To clarify myocardial fatty infiltration associated with sustained ventricular tachycardia (s-VT), 9 cases of dilated cardiomyopathy (DCM) and each one case of right ventricular dysplasia (RVD) and muscular dystrophy were examined pathologically. The materials with fatty infiltration were divided into 2 groups; group A (6 cases) recorded s-VT repeatedly and group B (5 cases), including one case of nonsustained VT, not recorded s-VT. The following results were obtained. Fatty infiltration layered characteristically and was present severely in a subepicardial area. (1) In group A, the layered fatty infiltration was transmural and extended in at least one ventricle in 5 cases. That is, 2 cases with the lesions in right ventricle (RV) recorded LBBB on ECG during VT, 1 of 3 cases with those in both ventricles revealed RBBB, 1 case presented LBBB and 1 case recorded undetermined type. The remaining one case showed mild fatty infiltration and characteristic localized transmural fibrosis in left ventricle (LV), and recorded RBBB during VT. (2) In group B, non-transmural fatty infiltration with moderate to severe fibrosis was characteristically present in myocardium and existed in LV in 3 cases and in both ventricles in 2 cases.

It was noticeable that transmural and extensive fatty infiltration was observed in cases with s-VT and the lesion was found in not only RV of RVD but also LV of DCM, some of which showed RBBB on ECG during VT.

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ELECTROPHYSIOLOGICAL CHARACTERISTICS AND PHARMACOLOGICAL RESPONSE OF EXERCISE INDUCED IDIOPATHIC VENTRICULAR TACHYCARDIA

Harumizu Sakurada, Ken-ichi Namba, Naokata Sumitomo, Masahiko Sakamoto, Nariaki Ejiri, Yoshiki Tokuyasu, Kouji Watanabe, Takeshi Motomiya, Masaya Sugiura, Masayasu Hiraoka*
Department of Cardiology, Tokyo Metropolitan Hiroo Hospital, Tokyo. *Department of Cardiovascular Institute, Tokyo Medical and Dental University.

To evaluate the electrophysiologic mechanism and pharmacological response of exercise induced idiopathic ventricular tachycardia, we performed programmed stimulation and isoproterenol infusion before and after intravenous administration of disopyramide, lidocaine, propranolol, verapamil in 6 patients with exercise reproducibly induced idiopathic ventricular tachycardia. Ventricular tachycardia was initiated by programmed stimulation in 2 out of 6 patients and isoproterenol infusion reproducibly provoked ventricular tachycardia in all 6 patients. Intracardiac mapping during ventricular tachycardia revealed that right ventricular outflow tract was the earliest activation site. At initiation of ventricular tachycardia with ventricular pacing, direct

relationship between the last pacing interval and the interval of the last paced beat to the first QRS of the ventricular tachycardia was observed. Propranolol effectively terminated and prevented isoproterenol provoked ventricular tachycardia in all 6 patients. Verapamil terminated and prevented isoproterenol provoked ventricular tachycardia in all 6 patients and prevented pacing induced ventricular tachycardia in all 2 patients. Lidocaine was effective in 5 patients and ineffective in 1 patient. Disopyramide was effective in 2 patients and ineffective in 2 patients.

These findings suggest that catecholamine sensitive abnormal automaticity including triggered activity was the mechanism of exercise induced idiopathic ventricular tachycardia. Propranolol and verapamil seemed to be clinically useful agents to prevent exercise induced idiopathic ventricular tachycardia.

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VENTRICULAR TACHYCARDIA (VT) IN PATIENTS WITH IDIOPATHIC CARDIOMYOPATHY -HEMODYNAMIC FINDINGS, LATE POTENTIALS AND PROGNOSIS-

Shimizu A., Nagata S., Ohe T., Kohnoe A., Matsuhisa M., Kamakura S., Sato I., Nakajima K., Shimomura K.

National Cardiovascular Center, Division of Cardiology

163 patients with idiopathic cardiomyopathy (ICM) were divided into 3 groups (sustained VT; group S, nonsustained VT; group NS, no VT; group N). Hemodynamic findings, late potentials and prognosis were studied. Nonsustained VT was defined as having ≥ 3 consecutive VPBs but lasted < 30 seconds, and sustained VT as lasting ≥ 30 seconds.

No differences among 3 groups (group S n=17, group NS n=31, group N n=33) were observed in regard to CTR, hemodynamic measurements including C.I., LVEDP, and RVEF, but LVEF in group S was significantly higher than that in group NS (35.8 ± 9.4 vs $28.9 \pm 7.9\%$, $p \leq 0.025$) in 81 pts with dilated cardiomyopathy (DCM). Follow-up observation from 1 to 108 months (33 ± 27) showed: sudden death (SD) 5, congestive heart failure (CHF) 1 in group S, SD 3 and CHF 5 in group NS, SD 0 and CHF 2 in group N. LPs were recognized in 9/9 pts (group S), 3/14 (group NS), and 0/10 (group N). Sustained VT and LPs were not recognized in 65 pts with HCM. 2 pts with sustained VT were observed, and LP was recognized in 1/1 (group S), in 17 pts with dilated HCM. It is concluded that (1) there is no correlation between VT and hemodynamic findings in DCM, (2) LPs are useful for predicting sustained VT in ICM, and (3) prognosis is poor in group S and NS compared with group N, and sudden death is the most frequent cause of cardiac death in DCM.

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EPICARDIAL MAPPING OF INDUCED NON-SUSTAINED POLYMORPHIC VENTRICULAR TACHYCARDIA IN CANINE MYOCARDIAL INFARCTION

SATOSHI OGAWA, TOSHIHISA MIYAZAKI,
TAKASHI SAKAI, MICHIO HOSOKAWA, KENJI
SAKURAI, YOSHIRO NAKAMURA

CARDIO-PULMONARY DIVISION, DEPART-
MENT OF MEDICINE, KEIO UNIVERSITY SCHOOL
OF MEDICINE

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

Takashi Sakai, Satoshi Ogawa, Toshihisa Miyazaki,
Michio Hosokawa, Kenji Sakurai, Yoshiro Nakamura

Cardiopulmonary Division, Department of Medicine,
Keio University School of Medicine

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: $1.0 - 17.3\%$, $10.8 \pm 6.3\%$). 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-

Naoto Yoneda, Katsuya Kobayashi,
Shigeki Itoh, Shinichi Kaneda,
Tomoo Inoue, Hisashi Fukuzaki

The First Department of Internal Medicine,
Kobe University School of Medicine

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

Yoshito Iesaka*, Nobuhiro Shiroyama*,
Hiroshi Amemiya*, Hideomi Fujiwara*,
Kazutaka Aonuma**

*Division of Cardiology, Tsuchiura
Kyohdoh General Hospital,
**Musashino Redcross Hospital

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-