The Clinical Evaluation of the Late Potentials in Patients with Ventricular Arrhythmias

YUKIO OZAWA, M.D., SHUJI YAKUBO, M.D., NAOSHI TANIGAWA, M.D.
MASAKI NAGASAWA, M.D., RYUSUKE KOJIMA, M.D., KAZUHIKO JINNO, M.D.
KAZUHIRA HIBIYA, M.D., ICHIRO WATANABE, M.D., TOMOAKI SAITO, M.D.
SATOSHI SAITO, M.D., AND MICHINOBU HATANO, M.D.

We investigated the recognition of late potentials in patients with and without organic heart diseases and spontaneous ventricular arrhythmias. None of the normal subjects had late potentials and patients with ventricular arrhythmias but no organic heart diseases, also had no late potentials as well as patients with idiopathic ventricular tachycardias. Late potentials in patients with idiopathic cardiomyopathy were noted more frequently in the dilated type than in the hypertrophic type, especially in those with high grades of ventricular arrhythmias. Patients with old myocardial infarctions had a higher rate of late potentials recognition in cases of sudden death or ventricular tachycardias. On the other hand, we observed lower rates in patients during early stage of acute myocardial infarction in spite of the evidence of a higher rate of ventricular electrical instability.

There was no association between ejection fractions, wall motion scores and late potentials. However, a higher recognition of late potentials was found in patients with inferior or posterior myocardial infarction and ventricular aneurysm.

We concluded that the late potential must be evaluated in each of the different groups of organic heart diseases in order to estimate the clinical value of ventricular arrhythmias.

THE approach for the clinical evaluation of ventricular arrhythmias has been studied by long term electrocardiographic recording or programmed electrical ventricular stimulation.

Recent studies¹⁻⁷ have shown that the late potentials may predict the development of reentrant ventricular arrhythmias and sudden cardiac electrical death. The late potentials are represented as delayed activation potentials

of diseased myocardial zone⁷ and have been recorded from the ischemic myocardium in the animal experiments and in humans, by endocardial or epicardial ECG⁸⁻¹²

The direct recording of late potentials is usually performed non invasively from the body surface by means of signal averaging^{1,12-14} or low noise and high resolution electrocardiography¹⁴⁻¹⁶. This signal may be a new independent marker to evaluate ventricular arrhythmias in comparison with long term electrocardiography or electrical stimulation methods.

At the present time, we have studied the recognition of late potentials for evaluating spontaneous ventricular arrhythmias in patients

Key words:

Late potential
Ventricular arrhythmia
Sudden death
Cardiomyopathy
Myocardial infarction

The Second Department of Internal Medicine, School of Medicine, Nihon University, Tokyo, Japan

This study was supported in part by Research Grant in 1985 from Nihon University.

Mailing address: Yukio Ozawa, M.D., The Second Department of Internal Medicine, School of Medicine, Nihon University 30-1, Ohyaguchikamimachi, Itabashi-ku, Tokyo 173, Japan

TABLE 1 PATIENTS POPULATION AND THE RECOGNITION OF LATE POTENTIALS

	Cases (Sex)	Age Mean $\pm$ SD (Range)	LP (%)
1) Normal subjects	60 (M : 36, F : 24)	41.2 $\pm$ 15.6 (15–68)	0
2) PVCs without organic heart disease	45 (M : 20, F : 25)	43.4 $\pm$ 16.4 (12–68)	0
3) PVCs with organic heart disease	64 (M : 38, F : 26)	52.9 $\pm$ 14.4 (16–77)	7.8
4) Idiopathic VT	9 (M : 5, F : 4)	44.4 $\pm$ 15.0 (23–67)	0
5) Idiopathic cardiomyopathy			
DCM	15 (M : 10, F : 5)	47.8 $\pm$ 14.1 (24–68)	46.7
HCM	8 (M : 5, F : 3)	44.4 $\pm$ 16.2 (23–66)	12.5
6) Myocardial infarction			
Acute (within 4 weeks)	65 (M : 53, F : 12)	60.0 $\pm$ 10.1 (32–81)	39.3
Old (over 4 weeks)	152 (M : 115, F : 37)	61.7 $\pm$ 11.2 (35–83)	42.1

LP; Late Potential, PVCs; Premature ventricular contractions, VT; Ventricular tachycardia, DCM; Dilated cardiomyopathy, HCM; Hypertrophic cardiomyopathy

Fig. 1. VA in 45 patients without organic heart disease. No LP can be recognized in these patients. VA = ventricular arrhythmia; LP = late potential

with and without organic heart disease.

MATERIALS AND METHODS

We studied 418 cases who were referred to the second department of internal medicine at Nihon University Itabashi Hospital.

Study populations were classified into 6 groups (Table I). Group 1 comprised 60 cases of normal subjects who had no symptoms and no

abnormal findings from electrocardiography and X-ray examination. Group 2 had 45 cases with premature ventricular contractions (PVCs) but without organic heart disease. Group 3 had 64 cases with PVCs and organic heart disease excluding idiopathic cardiomyopathy and myocardial infarction. Group 4 had 9 cases with idiopathic ventricular tachycardia. Group 5 had 23 cases with idiopathic cardiomyopathy 15 of which were of the dilated type and 8 were of the hypertrophic type. Group 6 had 65 cases with acute myocardial infarction (within 4 weeks) and 152 cases with old myocardial infarction (more than 4 weeks). All of PVCs have been observed by standard electrocardiography (ECG), long term ECG and/or ECG monitoring in coronary care unit (CCU) and post CCU. The evaluation of PVCs was done by using a modification of Lown's classification¹⁷ which is as follows; Grade 1 is within 1440 PVCs/day in long term ECG, Grade II is more than 1440 PVCs/day in long term ECG, Grade III is multiple PVCs in standard ECG and monitoring or long term ECG, Grade IVa is coupled PVCs and Grade IVb is continuous repetitive PVCs less than 6 beats, and Grade V is R on T. We also classified ventricular tachycardia (VT) as being continuously more than 6 beats. Long term ECGs recorded 1–5 times (mean 3.2) in each of cases except in normal subjects. If a patient had many grades of PVCs in the classifications mentioned above, we filled and evaluated each of III, IVa, IVb, V grades and VT. On the other hand, the highest classification grade among 0, I and II in the same patients was

Fig.2. VA and LP in 64 patients with organic heart disease excluded idiopathic cardiomyopathy and myocardial infarction.

VA = ventricular arrhythmia; LP = late potential

Fig.3. VA and LP in 15 patients with DCM.

VA = ventricular arrhythmia; LP = late potential; DCM = dilated cardiomyopathy

selected and filled from several long term ECGs recordings.

Signal averaging ECGs were recorded by means of a high resolution MAC-1 unit (Marquette Electronics) in order to estimate late potentials non-invasively. The MAC-1 unit uses three electrodes from which six leads are generated. For recording late potentials the electrodes are normally placed in V₁, V₅ and V_{6R}, or in V₂, low V₄ (6 cm below V₄) and high V₅ (6 cm above V₅)². The frequency response/output is the upper limit of 300 Hz and the lower limit of

0, 25, 50, 100, 200, or 0, 20, 40, 80 Hz. Number of beats averaged is usually 1024 beats and recording speed is 100 and 200 mm/sec. Signal sensitivities are 400 cm/mV in Channel 1, 20 cm/mV in Channel 2 and 1 cm/mV in Channel 3. Important filter features are zero phase shift, non-ringing and finite impulse response¹⁸. We evaluated late potentials usually at a portion of band pass filter setting from 100 (or 80), 200 to 300 Hz. We diagnosed late potentials, as signals outlasting the standard QRS complex by more than 20 msec in at least three or more leads,

Fig.4. The appearance of LP at one week after AMI can be seen in this patient.
LP = late potential; AMI = acute myocardial infarction

and exceeding twice the amplitude of base line noise. In some studies,^{16,19} the criteria of LP duration was shown by more than 10 msec from the end of QRS.

We also investigated the relationship between the late potentials and left ventricular function, and wall motion abnormalities, in 10 patients with dilated cardiomyopathy and in 43 patients with myocardial infarction. These cases received left ventricular cineangiography. Wall motion abnormalities were estimated by a score index calculated from the number of segments and degrees of wall motion.²⁰ Hypokinesia, akinesia, dyskinesia and aneurysmal were classified by 1, 2, 3, and 4 point respectively in each 7 segment of left ventricular wall. Total number of points in each segment was added as wall motion scores.

RESULT

1) Normal subjects

No late potentials were recorded in 60 normal subjects (Male: 36, Female: 24), age 41.2 ± 15.6 years old (range 15–68).

2) PVCs without organic heart disease

In 45 patients (M: 20, F: 25), who have PVCs but no heart disease, age 43.4 ± 16.4 yrs. (range 12–68), distributions of PVCs in this group are shown in Fig. 1. The rate of recognition of PVCs

in each grade decreased gradually in higher grades of the classification. Late potentials have not been observed in this group.

3) PVCs with organic heart disease (excluding myocardial infarction and idiopathic cardiomyopathy)

This group (64 cases, M: 38, F: 26), age 52.9 ± 14.4 yrs. (range 16–77), has 22 cases of effort angina pectoris, 4 of variant form of angina pectoris, 10 of hypertensive heart disease, 11 of mitral valve prolapse, 6 of rheumatic valvular disease, 5 of congenital heart disease (VSD: 3, ASD: 2), 2 of mitral valve replacement, 1 of Marfan syndrome, 1 of cor. pulmonale, 1 of Limb-girdle muscular dystrophy and 1 of sick sinus syndrome.

The contribution of PVCs in this group are shown in Fig. 2. Late potentials were recorded for 7.8% of this group. In 4 (80%) of the 5 patients with late potentials, PVCs of a higher grade than IVa were observed.

4) Idiopathic ventricular tachycardia

We studied 9 cases of patients with idiopathic ventricular tachycardia who were estimated by electrical stimulation and long term ECG. In these 9 patients (M: 5, F: 4), age 44.4 ± 15.0 yrs. (range 23–67), VT were induced in 2 cases, not induced in 5 cases by means of electrical programmed stimulation. 2 cases who had sustained ventricular tachycardia in long term

ECG had not received programmed stimulation. None of the 9 cases showed standard electrocardiographic and echocardiographic abnormali-

Changes of LP in AMI

(Ventricular Aneurysm with VT)

Fig. 5. Changes of amplitude and duration of LP during AMI.
LP = late potential; AMI = acute myocardial infarction

ties. Late potentials were not seen in this group.

5) Late potentials and ventricular arrhythmias in patients with idiopathic cardiomyopathy

23 patients of this group were divided into idiopathic dilated cardiomyopathy and hypertrophic cardiomyopathy. In the former group of 15 patients (M: 10, F: 5), age 47.8 ± 14.1 yrs. (range 24–68), late potentials were observed in 7 cases (46.7%). One (12.5%) of 8 cases (M: 5, F: 3) in the latter group showed, late potentials and high grade (II, III, IVa and IVb) of PVCs.

Recognition of both ventricular arrhythmias and late potentials in patients with dilated cardiomyopathy are shown in Fig. 3. Late potentials in each grade of PVCs showed a higher rate (100%) in IVb and VT. On the other hand, late potentials were not recorded in 72.7% of patients without VT, IVa and IVb.

6) Late potentials and PVCs in patients with acute myocardial infarction

In this group, 65 cases (M: 53, F: 12) of acute myocardial infarction, age 60.0 ± 10.1 yrs. (range 32–81), were investigated. Late potentials were recognized in 24 (39.3%) of 61 patients within 7 days, in 27 (42.2%) of 64 patients at second and third week, and in 28 (43.1%) of 65 patients at the fourth week from onset. We also recognized five new appearances and one disappearance of late potentials during acute phase of myocardial infarction. One case of late potentials appearance is shown as Fig. 4. The reproducibility of late potentials in acute myocardial infarction has been changed in some cases as shown by Fig. 5. We have one case of sudden

Fig. 6. VA and LP in 61 patients with AMI.
VA = ventricular arrhythmia; LP = late potential; AMI = acute myocardial infarction

Fig.7. VA and LP in 152 patients with OMI.

VA = ventricular arrhythmia; LP = late potential; OMI = old myocardial infarction

Fig.8. EF and LP in patients with MI or DCM.

EF = ejection fraction; LP = late potential; MI = myocardial infarction; DCM = dilated cardiomyopathy

death resulting from left ventricular rupture in our series during acute phase of myocardial infarction. This patient had not shown ventricular tachycardia and late potentials before sudden death. The recognition of late potentials for ventricular arrhythmias in patients with acute myocardial infarction is shown as Fig. 6.

In patients with higher grade of PVCs (IVb, V and VT), late potentials were more frequently recorded. On the other hand, we also observed late potentials in low grade of PVCs, because our investigation of late potentials in this series was

the evaluation in each grade of PVCs and patients may have 2 or more grades in this classification, for example, a patient may have both grade II and grade IVb.

7) Late potentials and PVCs in patients with old myocardial infarction

152 patients (M: 115, F: 37) with old myocardial infarction were studied. The age of this group was 61.7 ± 11.2 yrs. (Range 35–83). Late potentials were recorded in 64 (42.1%) of 152 patients, and found in all 4 cases of sudden cardiac death in this group. The recognition of

PVCs and the evaluation of late potentials for each grade of PVCs in this group were shown as Fig. 7. In all of 14 patients with VT, ventricular

Fig.9. Wall motion score and LP in patients with MI.
LP = late potential; MI = myocardial infarction

fibrillation or sudden cardiac death, late potentials were recognized. We also observed gradual late potentials more frequently in higher grades of PVCs in the classification. On the other hand, no late potentials were recognized in 88 (63.8%) of 138 patients without VT, Vf and sudden cardiac death, and in 66 (78.6%) of 84 patients without PVCs of higher grade than IVa.

The reproducibility of late potentials in old myocardial infarction was excellent, because no patients showed new appearance or disappearance of it during the stage of old myocardial infarction, and the shape or the duration of it was similar to each other in same patients.

8) The relationship between late potentials and ejection fraction in patients with dilated cardiomyopathy and myocardial infarction (Fig. 8).

Ejection fractions of 10 patients with dilated cardiomyopathy and 43 patients with myocardial infarction were evaluated by means of left ventricular angiography. In patients with dilated cardiomyopathy, ejection fractions were 40.8 ± 10.7 and 47.4 ± 18.2 in cases with and without late potentials respectively. There were no significant differences between both groups.

In patients with myocardial infarction, ejec-

T. S. 56y Male OMI (Ventricular aneurysm, VT)

Fig.10. Disappearance of LP and VT after LV aneurysmectomy in this patients with MI and VT.
LP = late potential; VT = ventricular tachycardia; LV = left ventricular; MI = myocardial infarction

tion fractions were 50.7 ± 9.6 and 55.7 ± 13.9 in cases with and without late potentials respectively. No relationship between the two groups was found statistically.

9) The relationship between late potentials and abnormal wall motion in patients with myocardial infarction

We estimated the late potentials and wall motion score measured by left ventricular angiography in patients with myocardial infarction. Wall motion scores were 6.7 ± 2.8 and 5.7 ± 2.5 in these cases with and without late potentials respectively as shown by Fig. 9. No difference between the scores in the cases with and without late potentials were observed statistically, in spite of slightly higher scores in patients with late potentials.

10) Late potentials in the location of myocardial infarction with ventricular aneurysm

We studied late potentials in 23 patients with anterior (antero septal and/or lateral) and inferior (inferior and/or posterior) myocardial infarction, and with ventricular aneurysm. Late potentials were found in 5 (35.7%) of 14 patients with anterior myocardial infarction and ventricular aneurysm. On the other hand, we recognized late potentials in 8 (88.9%) of 9 patients with inferior myocardial infarction and ventricular aneurysm.

11) The effects of aneurysmectomy for late potentials in patients with VT and ventricular aneurysm

In 2 patients with ventricular aneurysm and VT, late potentials were found before aneurysmectomy. After operation, late potentials and VT had disappeared in these cases with old myocardial infarction. One of cases is shown by Fig. 10.

DISCUSSION

The evaluation of prognosis in patients with ventricular arrhythmias has been usually estimated by standard ECG, long term ECG and programmed electrical stimulation. Recent studies have shown that the direct recording of late potentials from body surface may predict a prognosis of ventricular electrical instability. Usually, late potentials were recognized as representation of delayed potentials in damaged myocardium which were initially described from the ischemic zone of the experimental dogs^{9,23,24}. Also, these delayed potentials were recorded from the epicardium and endocardium of a diseased human heart by ECG mapping.^{10,25} In

order to record these small delayed activation potentials as late potentials on body surface, a signal averaging method^{1,11-13} or low-noise high resolution ECG¹⁴⁻¹⁶ was applied.

In our study, we recorded late potentials from bodysurface by means of high resolution signal averaging method.

No late potentials have been observed in our normal 60 cases, and terminal portions of QRS shown by averaging of filtered signals of high amplitude were relatively clear in this group, even in older age. Same results have been reported in other studies. Normal subjects, however, may have some kind of arrhythmias such as ventricular premature contractions in long term ECG. Indeed, our study of the patients without heart disease showed occasional PVCs in long-term ECGs. However, no late potentials have been recognized in this group, in spite of the fact that some PVCs may result from the mechanism of re-entry. Also, we observed no late potentials in patients with idiopathic ventricular tachycardias, even in patients who had VT induced by programmed electrical stimulations. Their standard 12 leads ECGs and echocardiographic findings have shown no abnormalities. In this group, the mechanism of ventricular arrhythmias may not be re-entry in some patients and, may be micro-reentry through purkinje fibers. If the circuits of re-entry is microscopic, late potentials may not be observed on body surface. Late potentials were recognized in 7.8% of patients with heart disease showed by figure 2, excluding myocardial infarction and idiopathic cardiomyopathy. Three of 5 patients with late potentials suggested evidence of diseased myocardium which was shown by left ventricular dysfunction or secondary myocardial disease such as muscular dystrophy. All of them had shown high grade of PVCs (over IVa) several times in their long term ECGs. On the other hand, 2 patients with mitral valve prolapse had late potentials without any evidence of severe diseased myocardium. One of them showed Grade IVb and the other one showed Grade II of PVCs. 80% of patients with late potentials in this group have shown high grade (over IVa) of ventricular arrhythmias.

Recent studies also suggested that late potentials may predict a high risk of ventricular electrical instability in patients with heart disease excluding ischemic heart disease but including dilated cardiomyopathy^{26,27} arrhythmogenic right ventricular dysplasia²⁸ hypertrophic cardio-

myopathy²⁷ and post-operative tetralogy of Fallot.²⁹

In our studies, 46.7% of the patients with dilated cardiomyopathy showed late potentials with a high incidence of late potentials in grade IVb of PVCs and VT. 72.7% of the patients in this group with PVCs of grade I, II and III had no late potentials. These figures suggest that late potentials may be useful to identify a high risk of ventricular electrical instability in patients with idiopathic dilated cardiomyopathy.

On the other hand, the patients with idiopathic hypertrophic cardiomyopathy showed a low rate of late potentials. One patient with idiopathic hypertrophic cardiomyopathy, however, showed late potential and multi-type of PVCs (II, III, IVa and IVb), together with congestive heart failure and localized thin myocardium of left ventricular posterior wall. The other typical hypertrophic cardiomyopathy patients didn't show late potentials.

The late potential in the patients with ischemic heart disease may be most useful to predict sudden cardiac death and ventricular electrical instability. Some studies indicate that in experimental animals with myocardial infarction, slow conduction and fragmented electrical activation related to the occurrence of re-entrant ventricular tachyarrhythmias can be observed.^{9,23,24,30} In human studies, fragmented electrical activity were recorded directly on endocardial or epicardial mapping during open heart surgery,^{31,33} or on endocardial mapping^{23,25,34} by means of electrode catheters, in patients with ventricular tachycardia after myocardial infarction. El-Sherif et al²⁴ have reported recently that conduction delays and reentrant excitation usually occur in the surviving, thin epicardial layer overlying the myocardial infarction after coronary ligation in the dog. This delayed activation can be recorded as late potentials from body surface by means of the signal averaging method or the low noise high resolution ECG in the experimental dog and in the patient with myocardial infarction. Recent studies^{1-3,19-21} have suggested that the late potential may be promising for the identification of patients at risk of ventricular tachyarrhythmias and of patients with myocardial infarction at risk of electrical sudden death.

In our study, we investigated the correlation between late potentials and spontaneous ventricular arrhythmias in patients with acute myocardial infarction (within 4 weeks) and old myocardial

infarction (over 4 weeks). Spontaneous PVCs were evaluated by combinations of several times (mean 3.2) of long term ECG, standard 12 leads ECG, and ECG monitoring in CCU and post CCU.

In the study of patients with acute myocardial infarction, late potentials were recorded in 39.3% within 7 days, 42.2% at second and third weeks, and 43.1% at fourth week after onset. These figures suggest that late potentials were increasing gradually week by week during the acute phase. We recognized many grades of PVCs without late potentials in the initial 7 days. This finding may suggest that the mechanism of ventricular arrhythmias in early stages is not simple, and may involve not only reentry but also triggered activity, high automaticity and others. Another reason may be the unstable or incomplete circuits of reentry in initial stage of acute myocardial infarction. Indeed, late potentials in some patients, such as Fig. 5, showed unstable changes in duration, shapes and amplitude during acute stage. Some patients had sustained VT, the first day of this disease but they recorded no late potentials during sinus rhythm during same day.

During acute myocardial infarction, patients with high grade of PVCs (Grade IVb, and VT) have shown higher rate of late potentials than other PVCs group. Grade V (R on T) had 100% of late potentials in this group, however, the analysis of R on T might be incomplete in long term ECG, as the numbers of R on T cases was too small to evaluate. A recent study³⁵ showed that QT interval and QTc did not correlate with the QRS duration and late potential duration.

Patients with old myocardial infarction (different group over 4 weeks from onset) have shown late potentials by 42.1%. In this group, we recognized a gradual increase of late potentials in higher grade of ventricular arrhythmias excluding Grade V which is incomplete to evaluate by long-term ECG. In particular, late potentials were recognized in 100% of patients with sudden cardiac death or VT and VF. Recent studies³⁶⁻³⁷ have suggested that the late potential may disappear in some patients with old myocardial infarction. However, our study has shown a good reproducibility of the recognition of late potentials during old myocardial infarction, in spite of changes in its form or duration.

Programmed electrical stimulation may be useful for the prediction of electrical sudden death in patients with coronary artery disease.

Breithardt et al.³⁸ studied the ventricular late potentials and inducible ventricular tachyarrhythmias as a marker for ventricular tachycardia after myocardial infarction. They concluded the signal averaging for the detection of late ventricular potentials and programmed ventricular stimulation seem to be promising new techniques for the identification of patients at risk of ventricular tachyarrhythmias. A recent report³⁹ on the prediction of ventricular tachycardia at programmed electrical stimulation by the signal averaging has also shown that the averaging had a greater predictive accuracy for both sustained and non-sustained VT in patients with coronary artery disease than in non-coronary artery disease.

On the other hand, long-term ECG may be also useful to predict ventricular electrical instability; however, reproducibility of this method is not without problems.

In some studies⁴⁰ of the signal averaging and long-term ECG monitoring, patients with both late potentials and a peak ventricular arrhythmia rate greater than 100/hr had a 91% probability of developing ventricular tachycardia, whereas patients with neither of these had only a 13% probability of developing ventricular tachycardia. In another report³⁷ results of the signal averaging and long-term ECG were discordant.

In our study, we investigated the recognition of the late potential in classification of spontaneous ventricular arrhythmias by long-term ECG, 12 leads ECG and ECG monitoring. We evaluated ventricular arrhythmias as often as possible by these methods, in order to reduce the problem of reproducibility. Our study showed that the late potential can be seen more frequently in patients with both old myocardial infarction and high grade of ventricular arrhythmias than in patients with both acute myocardial infarction and high grade of ventricular arrhythmias.

Several studies^{5,21,23} have suggested that there is an association between late potentials and ventricular dysfunction, particularly ventricular wall motion abnormalities.

In our study, the ejection fraction and the left ventricular wall motion score by left ventricular cineangiography showed statistically no difference between patients with and without late potentials. However, we recognized high rate of late potentials in patients with inferior or posterior myocardial infarction and left ventricular aneurysm. This discrepancy may originate from the wall motion score index which is

calculated by not only degrees, but also ranges of wall motion abnormalities. Sometimes, we recognized no late potentials in patients with wide range of myocardial infarction and large ventricular aneurysm. Also, for the recognition of late potentials in terminal portion of QRS, the location of delayed potentials of myocardium may be important.

Pollak et al.⁴² have also found no association between late potentials and left ventricular dysfunction, measured either as ejection fraction or wall motion score. An explanation for the differences in their results and those of others is that the differences in methods of recording and criteria of late potentials are likely to be most important. Indeed, there are many criteria for the measurement of late potentials at the present time, and recording techniques are also different from each other. Our results may suggest that the convenient combinations of both the degree and ranges of damaged myocardium are important for recognition of late potentials. In some reports,³⁶ late potentials disappeared after a re-attack of myocardial infarction.

The evaluation of late potentials for the treatment of ventricular dysarrhythmias is usually distinguished in patients with post operative left ventricular aneurysmectomy. Their late potentials and ventricular tachyarrhythmias had disappeared after aneurysmectomy^{2,43} as well as our study. One of our patients with ventricular aneurysm and ventricular tachycardias is shown in Fig. 10.

Our results suggest that late potentials may change its duration or amplitudes by antiarrhythmic drugs, such as disopyramide and high doses of lidocaine. However, the evaluation of late potentials for drug therapy remains obscure. Some studies⁴⁴ have also shown that antiarrhythmic drugs had no consistent effects on presence or timing of late potentials on the signal averaged ECG.

CONCLUSION

We studied the recognition of late potentials by body surface signal averaging ECG in patients with spontaneous ventricular arrhythmias.

1) Late potentials have not been recorded in normal subjects, in patients with PVCs only and in patients with idiopathic ventricular tachycardias.

2) Patients with PVCs and organic heart disease showed the low rate of the recognition of

late potentials.

3) In patients with idiopathic cardiomyopathy, the recognition of late potentials were more frequent in cases with high grade of PVCs and dilated cardiomyopathy than those in hypertrophic cardiomyopathy.

4) In spite of the evidence of the high grade of ventricular arrhythmias, the recognition of late potentials were at the lower rate in acute stage of myocardial infarction than in old myocardial infarction.

5) The patients with old myocardial infarction have shown high rate of recognition of late potentials in cases with sudden cardiac death or ventricular tachycardias.

6) The ejection fraction and the left ventricular wall motion score index in patients with myocardial infarction are likely to have no association with the recognition of late potentials, however, the high rate of late potentials are recognized in patients with inferior or posterior myocardial infarction and ventricular aneurysm.

7) The evaluation of changes of late potentials for treatment of ventricular arrhythmias remains obscure. However, the effects of aneurysmectomy for the ventricular electrical instability can be estimated by the disappearance of late potentials after operation.

REFERENCE

- BREITHARDT G, BECKER R, SEIPEL L, ABENDROTH RR, OSTERMEYER J: Noninvasive detection of late potentials in man, a new marker for ventricular tachycardia. *Eur Heart J* 2: 1, 1981
- ROZANSKI JJ, MORTARA D, MYERBURG RJ, CASTELLANOS A: Body surface detection of delayed depolarization in patients with recurrent ventricular tachycardia and left ventricular aneurysm. *Circulation* 63: 1172, 1981
- SIMSON MB: Use of signals in the terminal QRS complex to identify patients with ventricular tachycardia after myocardial infarction. *Circulation* 64: 235, 1981
- HOMBACH V, BRAUN V, HOPP HW, GIL-SANCHEZ D, SCHOLL H, BEHRENBECCK DW, TAUCHERT M, HILGER HH: The application of signal averaging technique in clinical cardiology. *Clin Cardiol* 5: 107, 1982
- BREITHARDT G, BORGGREFE M, KARBENN V, ABENDROTH R, YE H, SEIPEL L: Prevalence of late potentials in patients with and without ventricular tachycardia: Correlation with angiographic findings. *Am J Cardiol* 49: 1932, 1982
- DENES P, SANTARELLI P, HAUSER RG, URETZ EF: Quantitative analysis of the high-frequency component of the terminal portion of the body surface QRS in normal subjects and in patients with ventricular tachycardia. *Circulation* 67: 1129, 1983
- EL-SHERIF N, GOMES JAC, RESTIVO M, MEHRA R: Late potentials and Arrhythmogenesis. *Pace* 8: 440, 1985
- DURRER D, VAN LIER A, BULLER J: Epicardial and intramural excitation in chronic myocardial infarction. *Am Heart J* 68: 765, 1964
- BOINEAU JP, COX JL: Slow ventricular activation in acute myocardial infarction. A source of reentrant premature ventricular contractions. *Circulation* 48: 702, 1973
- WAXMAN HL, SUNG RJ: Significance of fragmented ventricular electrograms observed using intracardiac recording techniques in man. *Circulation* 62: 1349, 1980
- JOSEPHSON ME, HOROWITZ LN, FARSHIDI A: Continuous local activity. A mechanism of recurrent ventricular tachycardia. *Circulation* 57: 659, 1978
- FONTAINE J, PIERFITTE M, TONET JL, FILLETE F, FRANK R, GROSGOGGAT Y: Interpretation of afterpotentials from epicardium, endocardium and bodysurface in patients with chronic ventricular tachycardia. ed by HOMBACH V, HILGER HH, Signal averaging technique in clinical cardiology, FK Schattauer Verlag, Stuttgart New York, 1981, p 177
- SIMSON MB, SPIELMAN SC, HOROWITZ LN, HARKEN AH, JOSEPHSON ME, KASTOR JA: After potentials in man and cardiac arrhythmias. ed by HOMBACH V, HILGER HH, Signal averaging technique in clinical cardiology, FK Schattauer Verlag, Stuttgart-New York, 1981, p 253
- FLOWERS NC, SHVARSTMAN V, HORAN LG, PALAKURTHY P, SOHI SS, SRIDHARAN MR: Analysis of PR subintervals in normal subjects and early studies in patients with abnormalities of the conduction system using surface His bundle recordings. *J Am Coll Cardiol* 2: 939, 1983
- EL-SHERIF N, MEHRA R, GOMES JAC, KELEN G: Appraisal of a low noise electrocardiogram. *J Am Coll Cardiol* 1: 456, 1983
- HOMBACH V, KEBBEL U, HOPP HW, WINTER U, HIRCHE H: Noninvasive beat by beat registration of ventricular late potentials using high resolution electrocardiography. *Int J Cardiol* 6: 167, 1984
- LOWN B, WOLF M: Approaches to sudden death from coronary heart disease. *Circulation* 44: 130, 1971
- MARQUETTE ELECTRONICS INC.: Guide book of High-Resolution Amplifier Module for MAC Electrocardiography. Appendix 10, Technical Specifications. Milwaukee.
- JOSEPHSON ME, SIMSON MB, HARKEN AH, HOROWITZ LN, FALCONE RA: The incidence and clinical significance of epicardial late potentials in patients with recurrent sustained ventricular tachycardia and coronary artery disease. *Circulation* 66: 1199, 1982
- BREITHARDT G, SCHWARZMAIER J, BORGGREFE M, HAERTEN K, SEIPEL L: Prognostic significance of late ventricular potentials after acute myocardial infarction. *Eur Heart J* 4: 487, 1983

21. ZIMMERMANN M, ADAMEC R, SIMONIN P, RICHEZ J: Prognostic significance of ventricular late potentials in coronary artery disease. *Am Heart J* 109: 725, 1985
22. SIMSON MB, UNTEREKER WJ, SPIELMAN SR, HOROWITZ LN, MARCUS NH, FALCONE NH, JARKEN AH, JOSEPHSON ME: Relation between late potentials on the body surface and directly recorded fragmented electrocardiograms in patients with ventricular tachycardia. *Am J Cardiol* 51: 105, 1983
23. WALDO AL, KAISER GA: A study of ventricular arrhythmias associated with myocardial infarction in the canine heart. *Circulation* 47: 1222, 1973
24. EL-SHERIF N, SMITH RA, EVANS K: Ventricular arrhythmias in the late myocardial infarction period in the dog. 8. Epicardial mapping of reentrant circuits. *Circ Res* 49: 255, 1981
25. SPIELMAN SR, UNTEREKER WJ, HOROWITZ LN: Fragmented electrical activity-relationship to ventricular tachycardia. *Am J Cardiol* 47: 448, 1981
26. POLL DS, MARCHLINSKI FE, FALCONE RA: Abnormal signal-averaged ECG in non-ischemic congestive cardiomyopathy: Relationship to sustained ventricular tachyarrhythmias. *Circulation* 70: II-253, 1984 (Abst.)
27. ISHIDA K, ITO S, YOKOTA Y, KOBAYASHI K, IMAMURA H, FUKUZAKI H: Assessment of ventricular late potentials for predicting ventricular tachycardia in hypertrophic cardiomyopathy and dilated cardiomyopathy. *Circulation* 70: II-370, 1984 (Abst.)
28. EDVARDSSON N, HIRSCH I, LINDBLAD A, OLSSON SB: Properties of late potentials in arrhythmogenic right ventricular dysplasia. *Circulation* 70: II-373, 1984 (Abst.)
29. DANFORD DA, GARSON A Jr: Abnormal conduction related to ventricular dysrhythmias by signal averaged electrocardiography in post operative tetralogy of Fallot. *Circulation* 70: II-207, 1984 (Abst.)
30. BERBARI EJ, SCHERLAG BJ, HOPE RR, LAZZARA R: Recording from the body surface of arrhythmogenic ventricular activity during the ST segment. *Am J Cardiol* 41: 697, 1978
31. FONTAINE G, GUIRAUDON G, FRANK R, VEDEL J, GROSGOGGAT Y, CABROL C, FACQUET J: Stimulation studies and epicardial mapping in ventricular tachycardia: study of mechanisms and selection for surgery. Kulbertus HE, ed Re-entrant arrhythmias mechanisms and treatment. Lancaster. MTP Press: 333, 1977
32. OSTERMEYER J, BREITHARDT G, KOLVENBACH R, KORFER R, SEIPEL L, SCHULTE HD, BIRCKS W: Intraoperative electrophysiologic mapping during cardiac surgery. *Thorac Cardiovasc Surg* 27: 260, 1979
33. KLEIN H, KARP RB, KOUCHOUKOS NT, JAMES TN, WALLO AL: Ventricular mapping of abnormal myocardium in patients with and without arrhythmias. *Circulation* 60: II-24, 1979 (abst.)
34. WILLIAMS DO, SCHERLAG BJ, HOPE RR, EL-SHERIF N, LAZZARA R: The pathophysiology of malignant ventricular arrhythmias during acute myocardial ischemia. *Circulation* 50: 1163, 1974
35. MARINCHAK RA, KLINE RA, ENGEL TR: Relationship of delayed depolarization to the QT interval after acute myocardial infarction. *Am Heart J* 110: 742, 1985
36. KERTES P, POLLAK SJ, OLSEN A, WALTER P: Evolutionary changes in the signal averaged electrocardiogram after myocardial infarction. *Circulation* 72: III-212, 1985 (abst.)
37. DUCHAR D, THORBURN C, SAMMEL N: Natural history and clinical significance of late potentials after myocardial infarction. 72: III-477, 1985 (abst.)
38. BREITHARDT G, BORGGREFE M, HERTEN K: Ventricular late potentials and inducible ventricular tachyarrhythmias as a marker for ventricular tachycardia after myocardial infarction. *European Heart J* 7: 127, 1986
39. KERTES PJ, POLLAK SJ, WALTER PF: Programmed stimulation for ventricular tachycardia: Responses predicted by signal averaging in patients with and without coronary disease. *Circulation* 72: III-433, 1985 (abst.)
40. KANOVSKY M, FALCONE RA, DRESDEN CA, JOSEPHSON ME, SIMSON MB: Identification of patients with ventricular tachycardia after myocardial infarction: Signal-averaged electrocardiogram, Holter monitoring, and cardiac catheterization. *Circulation* 70: 264, 1984
41. BREITHARDT G, BORGGREFE M, QUANTIUS B, KARBENN U, SEIPEL L: Ventricular vulnerability assessed by programmed ventricular stimulation in patients with and without late potentials. *Circulation* 68: 275, 1983
42. POLLAK SJ, KERTES PJ, BREDLAW CE, WALTER PF: Influence of left ventricular function on signal averaged late potentials in patients with coronary artery disease with and without ventricular tachycardia. *Am Heart J* 110: 747, 1985
43. BREITHARDT G, SEIPEL L, OSTERMEYER J: Prognostic significance of late potentials after acute myocardial infarction. *Eur Heart J* 4: 487, 1983
44. DENNISS AR, ROSS DL, RICHARDS DA, CODY DV, RUDELL PA, YOUNG AA, UTHUR JB: Effect of antiarrhythmic therapy on delayed potentials detected by the signal-averaged electrocardiogram in patients with ventricular tachycardia after myocardial infarction. *Am J Cardiol* 58: 261, 1986