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Diastolic Oscillatory Waves (DOWs) and Primary Ventricular Fibrillation (PVF)

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Ventricular tachycardia (VT) and primary ventricular fibrillation (PVF) are the leading causes in cardiac sudden desth (CSD). Some electrocardiographic changes which occurred before the onset of ventricular arrhythmias may reveal the electrical instability of the heart.

To recognize and correct these abnormalities may be helpful for prevention of CSD.

Electrocardiographic changes before and during onset of PVF monitoring in coronary care unit (CCU) or by Holter, have been studied retrospectively in the hospital. They were composed of 4 male and 12 female, aged 21–76 years (average 48.25 years), and associated with one of the following disorders, such as ischemic heart disease, hypertension, mitral valve stenosis, myocarditis, hypokalemia, primary or secondary QT prolongation etc. As a mater of fact that VT or PVF often preceded by a lot of abnormalities of ECG in the group. In addition to prolonged QT interval, ventricular premature beats (VPBs) showed multiple origin or in pairs and R on T phenomenon, we have found a bizarre wave at the end of the T waves over 6 cases in the group, and the name of diastolic oscillatory waves (DOWs) was given. It became more prominent after VPBs in pairs or a short run of VT. The wave often changed its amplitude from time to time, it appeared wax and wane even at the same lead. As a rule, when its aplitude became higher, VPB, VT or PVF should ensue.

Case 1. —A 41 year old woman was admitted on Aug 28, 1982. She got a seizure before her admission, and showed a prolonged QT interval at ECG monitoring, and finally resulted in a frequent VPBs, VT and VF. She had several attacks of syncope recently, but none of her family members did so. On admission, her

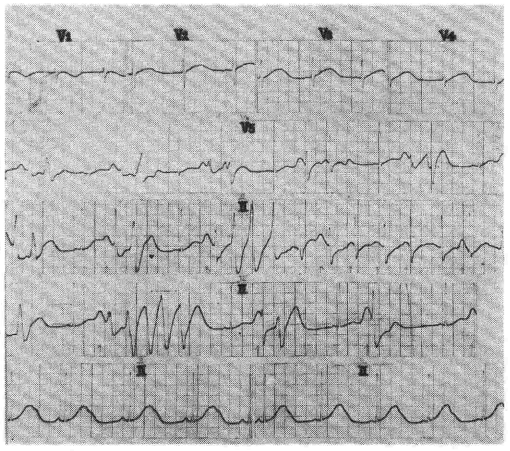

Fig. 1:
Strip 1—QT prolonged up to 0.06 sec before ventricular tachycardia;
2—VPBs of multiple origin and DOWs could be seen. After VPBs the amplitude of DOWs became higher;
3—Amplitude of DOWs was enhanced from beat to beat then the short run of VT ensued;
4—Frequent DOWs followed by short run of VT;
5—DOWs disappeared.

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HR was 62 beats per min, and BP 100/60 mmHg. In addition to extrasystole, her heart sound was normal. Laboratory findings showed a serum potassium level of 3.3–4.6 mEq/l, and her chest film and UCG were within normal limits.

Case 2. —A 21 year old young man was admitted on Sept 3, 1982 for a burning accident. He became edematous and unconsious when he was administered by a great deal of glucose and saline solution (6.000 ml per day). When he was getting better after transfusions and diuresis, he was received a skin grafting repeatedly, therefore he got some trouble from the infections, and produce severe hypokalemia (3.5 to 2.1 mEq/l). Afterward he showed a frequent attack of Adams-Stokes syndrome and moved into CCU. His monitoring ECG showed the close correlation between the ventricular arrhythmias and the DOWs.

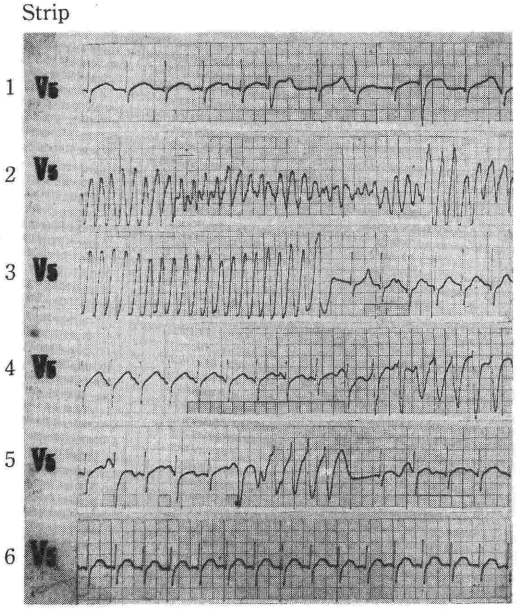

Fig. 2:
Strip 1—Before VT and VF the DOWs could be clearly seen;
2—VT and VF developed;
3—The peak of DOWs shifted ahead after ventricular flutter;
4—The DOWs got higher and higher and VT ensued;
5—The DOWs were significant before VT and VF and waning after VT and VF;
6—DOWs disappeared after administration of large dose potassium chloride.

Discussion

Diastolic waves (DWs) as a prodrome leading to ventricular arrhythmias has been described by Orinius and Eivinsson since 1979 and 1980.[1-2] They suggested that DWs could be refered as a prodrome of ventricular tachycardia. In strict sense, these waves differ from the U waves.

Based on the electrophysiologic studies[3], many factors, such as hypoxemia, low k^+, low Cl^-, low Ca^{++}, hypoosmolar solution, mechanic trauma, and some drugs may result in the early afterdepolarization in the Purkinje fibers and myocardium, and cause the low membrane potential oscillations (−30 to −40 mV). In addition, these factors could also produce lower membrane potential oscillations (−70 to −85 mV). It was known as delayed afterdepolarizations which may occur either by pacing or by premature stimulations. When a susceptible fiber is paced progressively and rapidly, the delayed afterdepolarization increases in amplitude and peaks earlier until it reaches the threshold and produces a premature action potential, it can also produce tachyarrhythmia. In some cases of the group, the additional waves arise from the end of the T waves often enhance its amplitude after premature beats particularly in pairs, and the peak of the waves also may shift ahead after the short run of VT. Lazzara[4] have reported a case of long QT syndrome associated with a seizure of Adams-Stokes syndrome. Using bipolar electrogram, he found many slow potential waves in various sites at the endomyocardial surface of both ventricle, arising from the end of the T waves. When epinephrine 0.02 ug/kg/min was administered, the ventricular extrasystole could be found at the site of extrapotential recorded.

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The findings were compatible with the hypothesis that abnormally large afterpotentials generated by ventricular myocardial cells are enhanced by β-adrenergic stimulation to reach threshold for excitation and produce automatic firing.

Based on the electrophysiological point of view as mentioned above, we suppose that the DOWs showed in ECG of the group may reflect the oscillatory potential arising from the delayed after depolarization under some pathologic conditions. It is reasonable to consider the DOWs as electrical instability of the heart, and as a prodrome of ventricular arrhythmias. It seems that DOWs is more apt to occur in the presence of QT prolongation. According to the characteristic findings of DOWs, it is obviously differed from U waves. The electrocardiographic change of U waves O- is more stable and often associate with ST-T changes. It is rarely seen that a U wave changes its amplitude from beat to beat, and it is never regarded as a prodrome of VT and VF.

Reference

1) Orinius E: Acta Med Scand **206** : 127-129, 1979.
2) Ejvinsson G et al.: Acta Med Scand **208** : 445, 1980.
3) Braunwald E: Heart Disease—A Textbook of Medicine Saunders Company Philadelphia, London Toronto 1980, Vol 1, p.645-646.
4) Lazzar R et al.: Am J Cardiol **45** : 472, 1980.

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