NEGATIVE T WAVES AFTER VENTRICULAR PREMATURE BEATS

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Postextrasystolic T wave change has been described since 1915, and a number of mechanisms have been proposed to explain it, including an abnormal pathway of repolarisation, prolonged diastolic filling time and an abrupt change, in the cycle length (1, 2, 6, 7, 13).

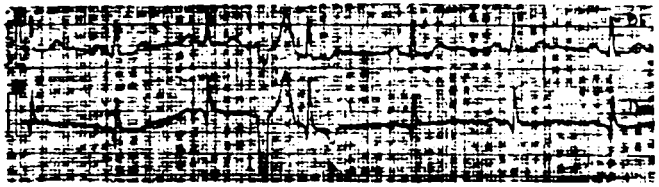
A negative T wave configuration occurring in patients with coronary heart disease, only in the postextrasystolic beat has been found in two situations: after a compensatory postextrasystolic pause, and after an interpolated ventricular beat.

Our intention is to present this electrocardiographic observation, and to try to explain the underlying mechanism.

Material

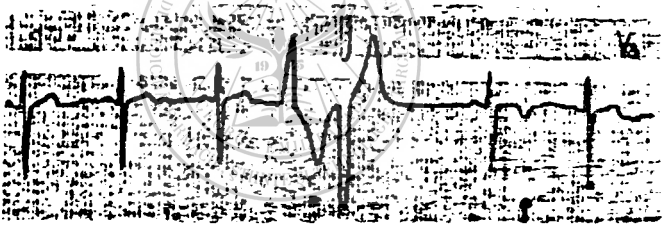
A negative T wave was found in nine patients with clinically and electrocardiographically documented coronary artery disease, in five after a compensatory postextrasystolic beat, the premature beats being in all of ventricular origin.

These two situations are presented in the following figures:



In Figure 1, after an interpolated ventricular premature beat, a negative T wave in the sinusal complex is shown (arrow).

In Figure 2, after ventricular beats with compensatory pauses, negative T waves are shown (arrows).



In Figure 3 two coupled premature ventricular beats are followed after a compensatory pause by a negative T wave (arrow).

Discussions

A premature depolarisation results in a reduced mechanical contraction directly proportional to the degree of prematurity; the ensuring contraction is more forceful than the normal ones (2, 4).

During a compensatory pause, the ventricular end-diastolic volume is augmented, and this increased preload along with a greater contractility increases the stroke volume, the ventricular contraction being more forceful than normal, - phenomenon termed postextrasystolic potentiation (1, 5, 9, 13). A ventricular extrasystole augments contractility, although to a

decreasing extent, for several cardiac cycles. This effect in hearts with depressed function is more prominent (1, 3, 5, 8).

In interpolated ventricular premature beat the following complex being placed immediately after electric refractory period. results in a small, but increased contraction velocity and a short duration of contraction, - the so-called force frequency relation, predisposing to aberrancy, phenomenon which does not occur in the normal heart, but only when conductivity is depressed (2, 7, 10, 11, 12).

The action potential duration of the endocardium is longer than that of the epicardium, and the ventricular repolarisation takes place from epicardium to endocardium. in the presence of myocardial ischemia, the action potential duration is shorter, creating electrical gradients, which results in ST segment depression and negative T wave, depending on the surface ECG leads. Increased myocardial oxygen demand, with failure to increase, or an actual decrease in regional coronary blood flow, will usually cause this phenomenon (5, 8). The ventricular loading from beat to beat using left ventricular monophasic action potentials, showed a significant change in the timing of depolarisation, the peak systolic pressure and the QT interval having a significant correlation. Elevation of end-diastolic pressure lengthened and the evaluation of peak systolic pressure shortened the monophasyc action potential duration (9).

Since the myocardial oxygen contracttility is an important determinant of myocardial oxygen deprivation in an underlying myocardial disease, alongside with inadequate removal of metabolites following reduced perfusion, it may produce a myocardial ischemia, manifested as an ischemic T wave pattern (2, 5, 11). The inotropic stimulation induced by postextrasystolic pause and the increased contraction velocity after interpolated premature beat tend to increase the nonuniformity of contraction and relaxation of the left ventricle, already present in coronary artery disease patients, where there may be scattered areas of ischemia with different rates of recovery of excitability in neighboring fibres (2, 5, 10, 12), ischemia being an important determinant of heterogeneity of repolarisation and conduction (12).

Ventricular premature beats are frequent in patients with coronary heart disease, but the postextrasystolic negative T waves were observed in a few cases only, and after a careful follow-up of the ECG recordings. A more complex investigation of this phenomenon should be an interesting work.

We believe that the negative T wave appearing in patients with coronary artery disease only in the postextrasystolic beats after compensatory pause or interpolated ventricular extrasystoles, may be a sign of silent myocardial ischemia.

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Postextrasystolic changes in ventricular depolarisation manifested as a negative T wave were found in two situations in patients with coronary artery disease: a) after a compensatory postextrasystolic pause and, b) after an interpolated ventricular beat. The explanation and the underlying conditions of this phenomenon are discussed. In our opinion the postextrasystolic change may be a silent myocardial ischaemia.

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