Right ventricular monophasic action potentials in patients with long QT syndrome

Sir,

Gavrilesco and Luca in their recent paper (*British Heart Journal*, 1978, 40, 1014-1018) have reported different patterns of ventricular monophasic action potential recorded in patients with long QT syndrome. On the basis of their observations they conclude that ventricular arrhythmias in those patients may be triggered by critical differences of refractoriness between various ventricular areas resulting from uneven and delayed repolarisation of ventricular myocardium. A close analysis of their right ventricular monophasic action potential tracings suggests the following.

(1) The different morphological configuration of

the right ventricular monophasic action potential shown in Fig. 5 (A) and Fig. 5 (B) suggests that the 2 recordings were obtained from different types of ventricular fibres. The former (A) with an obvious plateau, from Purkinje fibres; the latter (B) from contractile myocardium. This should, at least in part, account for the wide difference in monophasic action potential duration (Fenici *et al.*, 1978b).

(2) I agree that the so-called 'second monophasic action potential deflections' (0_1 —Fig. 4 (C) and Fig. 5 (C)) are not artefacts but I think that they could be interpreted as afterpotentials rather than as delayed repolarisations, especially as their amplitude

Fig. Examples of 'after-potentials' recorded: at atrial level (X), a patient with paroxysmal atrial fibrillation; and at ventricular level (Y), patient with massive digoxin intoxication.

increased with decreasing cycle length (Crane-field, 1977). This might suggest 'triggered activity' rather than re-entry as the most reasonable mechanism of the onset of ventricular arrhythmias in that patient. Similar diastolic phenomena have been recorded by me in a patient with paroxysmal atrial fibrillation (Fenici *et al.*, 1978a) and another with severe digoxin intoxication (Fenici *et al.*, 1978c). In both cases arrhythmias appeared to be triggered, either spontaneously or electrically, from the ascending phase of the 'afterpotentials' (see Fig.).

(3) Monophasic action potential recording in case 3 shows also that QT intervals of the same duration may be explained either by the duration of the action potential of 'plateau' fibres (Fig. 5 (A)), or by the occurrence of 'early' afterpotentials (Fig. 5 (C)). This suggests that further investigations by monophasic action potential recording technique could prove to be useful in clarifying the electrophysiological mechanism underlying the genesis of the U wave which is at present still controversial (Hoffman and Crane-field, 1960).

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References

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This letter was shown to Drs Gavrilescu and Luca who reply as follows.

Sir,

We are grateful to Dr Fenici for his pertinent observations. However, we must reiterate our belief that the method of monophasic action potential recording in the intact heart has many limitations so that conclusions drawn by using this method alone are often speculative. It is true that different morphological right ventricular monophasic action potential configurations can be the result of recording from various anatomical structures, but in our experience with normal and pathological hearts both in man and animals we have never encountered these differences, with the exception of the long QT syndrome. On the other hand, such a configuration can change, as in case 1, simultaneously with the regression of the prolonged QT interval. We can not exclude the explanation forwarded by Dr Fenici concerning the configuration of monophasic action potential recordings shown in Fig. 4 (C) and Fig. 5 (C). However, we believe that the prolonged refractoriness found with the help of pacing is more likely to be the result of prolonged repolarisation. We agree that some modifications of the T wave and of the U wave may be clarified by using monophasic action potential recordings.

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