

Ventricular Tachyarrhythmias*

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The mechanisms of ventricular tachyarrhythmias fall into two broad categories of increased automaticity and reentry. It is usually difficult to differentiate clinically between the two mechanisms; however, I plan to discuss certain approaches that may be helpful in this regard.

Experimental evidence of increased automaticity in the isolated Purkinje fibers can be obtained by placing cardiac tissue in a bath and adding epinephrine, isoproterenol, digitalis, or lowering the potassium concentration in the bath. This suggests that ventricular tachycardias precipitated by infusion of isoproterenol, excessive doses of digitalis, or hypokalemia may be ascribed to increased automaticity. On the other hand, quinidine or hyperkalemia suppresses automaticity in isolated preparations, and therefore ventricular arrhythmias occurring in patients treated with excessive doses of quinidine or potassium are probably due to reentry rather than to increased automaticity. This type of extrapolation from an experimental setup to a clinical situation does not prove the mechanism but the data may be used in support of a reasonably sound working hypothesis.

The experimental evidence for reentry presented by Drs. Hoffman, Cranefield, Moe and others in the isolated preparation appears soundly documented. Similar conclusions can be drawn from experimental studies in the entire heart following production of myocardial infarction in dogs (fig. 1). Figure 1 shows the result of an experiment in which electrograms are recorded from right atrium (AD), right ventricle (VD), and left ventricle (VS) after experimental occlusion of the left coronary artery in a dog. In the infarcted area (VS) the conduction is slow as evidenced by delayed onset and long

duration of the QRS complex. This QRS complex is inscribed at the time when the recovery of the noninfarcted area (QT interval in the right ventricle) is completed. Therefore, the extrasystole (Ex) can be ascribed with reasonable certainty to the reentry of the slowly propagating impulse from the infarcted into the noninfarcted area.

Since most of our patients have closed chests, we have no access to local electrograms, and we must limit our analysis to the surface electrocardiogram. This may occasionally provide information concerning the mechanism of ectopic beats which initiate ventricular tachycardias. There are two types of ectopic beats—dependent and independent. The dependent requires the presence of a preceding dominant pacemaker. Features that support a dependent mechanism are: 1) fixed coupling; that is, the beats are coupled in a fixed way to the preceding sinus beat, and 2) predictable response to change in the rate of the sinus pacemaker. For instance, when the cardiac rate slows, the ectopic beats become less frequent in response to carotid sinus stimulation (fig. 2), but when the sinus rhythm accelerates, the number of ectopic beats increases. Figure 3 illustrates some of the possible explanations for the dependent mechanism. The propagated impulse disturbs a quiescent Purkinje fiber and induces diastolic depolarization (3B). Such a fiber would acquire pacemaker properties, and by firing before the next sinus impulse, would produce a ventricular extrasystole with a fixed coupling interval due to repetitive firing. Another possibility of a repetitive firing is shown in figure 3C. In this case the fiber has a short refractory period as evidenced by short duration of action potential. This fiber can be depolarized again by a current generated during repolarization. In this case reentry preceding a closely coupled extrasystole is ascribed to non-

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Fig. 1—Electrograms of a dog with experimental occlusion of left coronary artery. (Reproduced by permission of L. Baschieri, L. Palagi, and M. Puletti. "Experimental Study of the Pathogenesis of Ventricular Extrasystolia." *Cardiologia*, 50:371, 1967. Explanation in text.)

homogeneous refractoriness. The third mechanism which supports the dependent type, and which is most commonly illustrated in textbooks, is the mechanism whereby a particular fiber is bypassed on the way to the ventricle (3D). In such a case of a so-called unidirectional block, the bypassed fiber does not fire during normal propagation of an impulse but becomes depolarized when the impulse returns from another side and causes an ex-

trasystole. These are the three possibilities whose occurrence may produce a ventricular extrasystole of a dependent type.

An example of an independent mechanism is a parasystole. How does one diagnose parasystole? Obviously, we look for evidence of variable coupling, fusion beats, and a common denominator indicating the assumed rate of firing of the independent, or "protected" pacemaker. When the parasystolic impulses do not appear at the expected time, we postulate an exit block, or intermittence due to temporary loss of protection. When application of carotid sinus stimulation suppresses the dominant pacemaker, the so-called parasystolic focus will emerge undisturbed and fire at the same rate as prior to the carotid sinus stimulation (fig. 4).

In clinical practice, automaticity and reentry can be occasionally distinguished on the basis of response to treatment. For instance, treatment of digitalis induced ectopic atrial tachycardia with potassium demonstrates that the rate of the ectopic pacemaker gradually decreases. This would suggest that the rate of diastolic depolarization is slowed and that this type of arrhythmia is based upon automaticity rather than reentry. We believe that reentry would be more likely to cease rather abruptly and not by gradual slowing of the rate. This is shown in figure 5. In this patient severe hyperkalemia produced ventricular flutter which did not

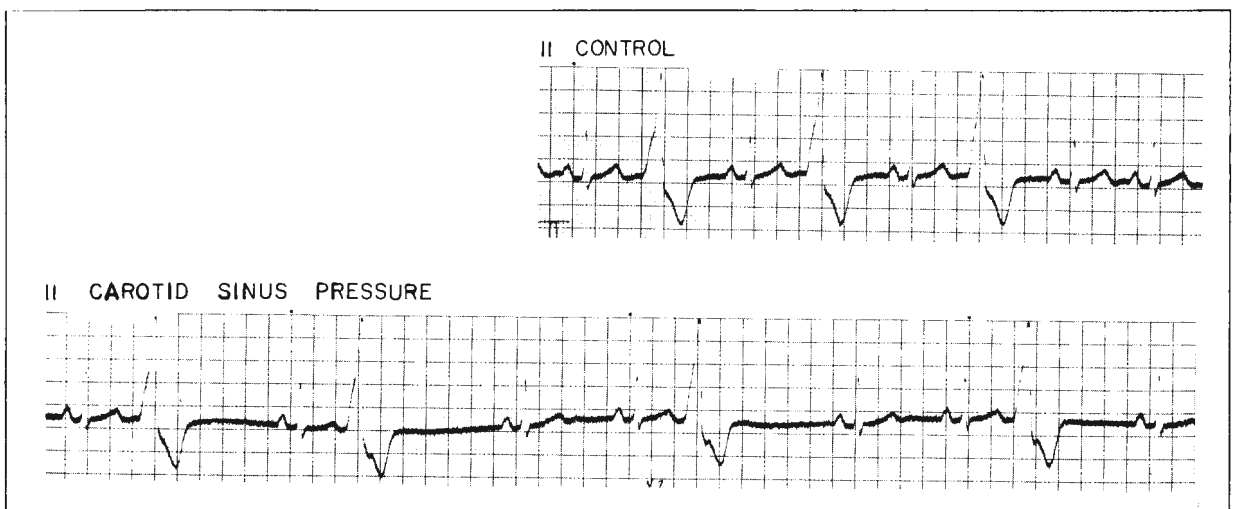


Fig. 2—Continuous tracing of lead II of a 48-year-old man with rheumatic heart disease. Ventricular ectopic beats with fixed coupling interval. After carotid sinus stimulation (lower tracing), the sinus rate is slower but the coupling interval remains unchanged and no ventricular ectopic beats appear during long P-P intervals. (Reproduced by permission of B. Surawicz and M. G. MacDonald and the *American Journal of Cardiology*, 13:199, 1964.)

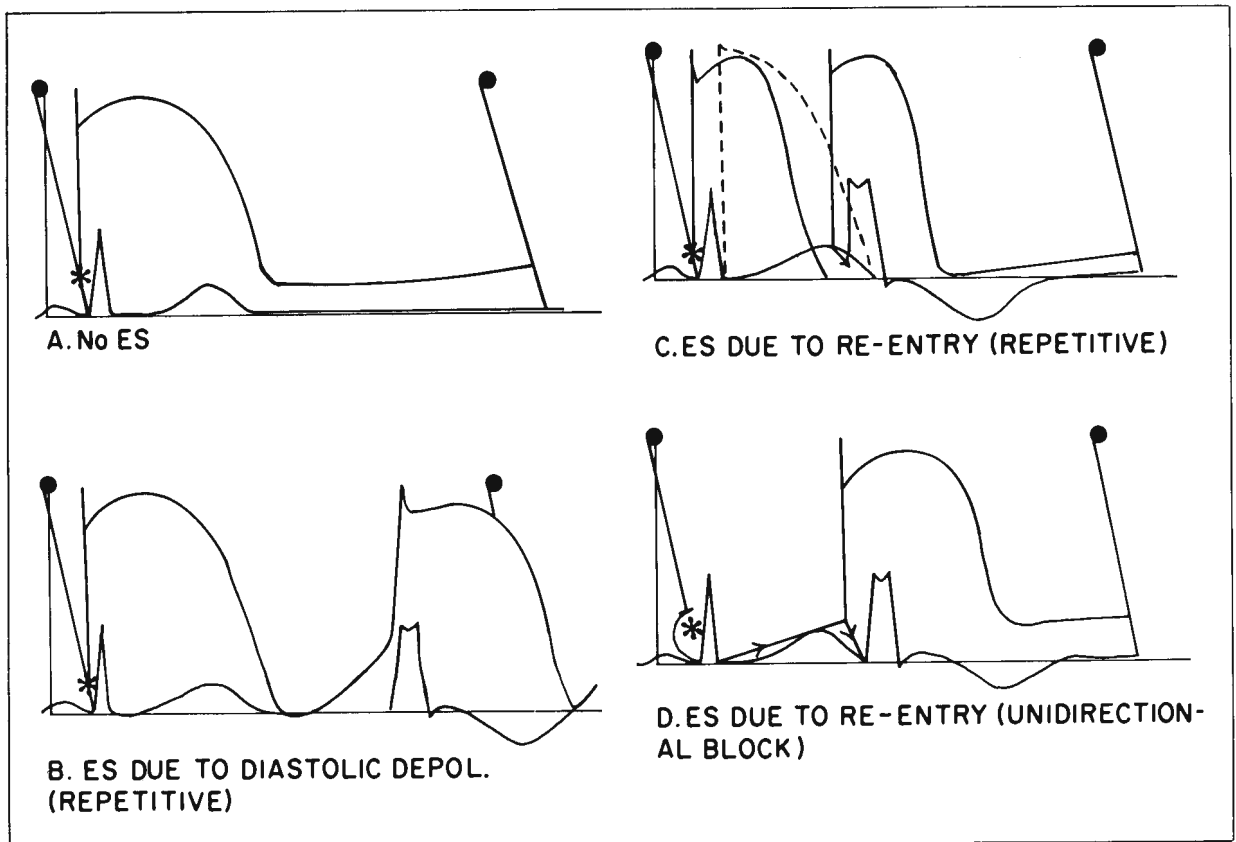


Fig. 3—Diagram representing 3 different mechanisms of dependent ventricular extrasystole originating in a Purkinje fiber marked by asterisk. In A, the fiber is depolarized by the impulse spreading from the sinus node (black dot) and no extrasystole ensues. In B, the sinus impulse enhances diastolic depolarization which causes repetitive firing. In C, the refractory period of the depolarized Purkinje fibers is short, and the repolarization is completed during ventricular repolarization (dashed action potential). In this case, reentry may be due to potential difference between the ventricular fiber and the prematurely repolarized Purkinje fiber if these two fibers are in close proximity to each other. In D, reentry is due to unidirectional block (see text).

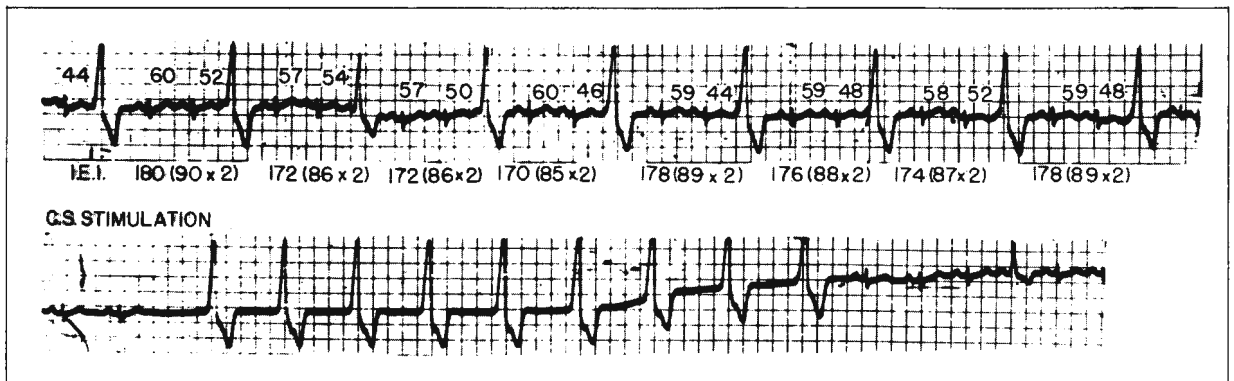


Fig. 4—Lead II of a 64-year-old laborer admitted to the hospital after an episode of syncope suffered on the street. Continuous strip of a tracing recorded on admission. The range of R-R intervals in the entire tracing was 0.52 to 0.68 sec and of coupling intervals, 0.42 to 0.56 sec. The interectopic intervals are multiplicants of an interval ranging from 0.82 to 0.98 sec. After carotid sinus stimulation (lower tracing), the sinus pacemaker is inhibited and the first and second ventricular ectopic beats appear before the first atrial deflection is recorded. The intervals between ventricular ectopic beats measure 0.98 sec and are constant. The P waves are apparent on the descending limb of the T wave from the 2nd to the 8th ventricular ectopic beats but the corresponding atrial impulses are not conducted to the ventricles. Note a fusion beat at the end of the lower strip. (Reproduced by permission of B. Surawicz and M. G. MacDonald and the *American Journal of Cardiology*, 13:199, 1964.)

respond to conventional treatment of hyperkalemia. The administration of lidocaine terminated the flutter abruptly, and this would suggest that the origin of arrhythmias is on the basis of reentry. Another interesting clinical situation was that of a 42-year-old man with ventricular tachycardia complicated by syncope and requiring repeated electrical defibrillation. This patient's particular tachyarrhythmia was resistant to all forms of therapy with the exception of fairly large amounts of procainamide. Thus, it was decided to introduce a temporary ventricular pacemaker catheter in order to convert his arrhythmia. When the patient developed ventricular tachycardia, the first pacemaker stimulus fell upon the refractory period, and the second one fell upon the T wave and produced a fusion beat that resulted in prompt reversion to sinus rhythm (fig. 6). It is interesting to observe that the pacemaker rate was slower than the rate of the ventricular tachycardia. This type of response suggests a reentry mechanism rather than one based upon automaticity.

There have been other methods for managing refractory ventricular tachycardia, such as aorto-coronary bypass and resection of a ventricular aneurysm. It is reasonable to conclude that the removal of dead myocardium with resultant disappearance of the arrhythmia suggests a reentry mechanism rather than increased automaticity. Finally, it is conceivable that certain tachyarrhythmias may be due to combinations of increased automaticity and reentry, for example, repetitive reentry into a partially, or intermittently protected parasystolic focus.

In summary, there is well documented experimental evidence of both increased automaticity and reentry in the experimental preparations. However, in the clinical setup, the distinction between these two mechanisms of ventricular tachyarrhythmia is usually very difficult to determine. Certain diagnostic clues can be obtained from the observations of the dependence of the ectopic beats upon the preceding dominant impulses and from the responses to therapeutic interventions.

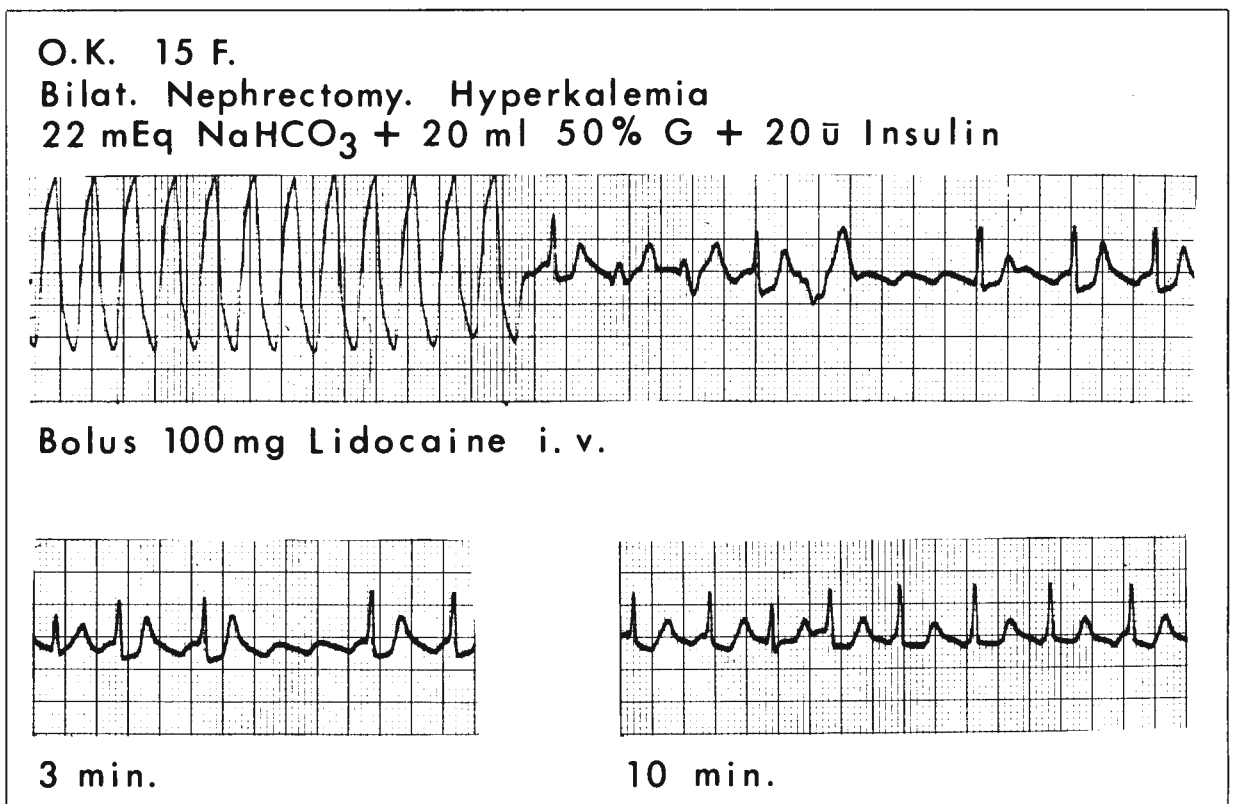


Fig. 5—Abrupt termination of ventricular flutter after administration of lidocaine in a 15-year-old patient with hyperkalemia and atrial flutter. Note the characteristic peaked T wave after termination of ventricular tachyarrhythmia. Lidocaine was administered after the failure of conventional therapy with sodium bicarbonate, glucose and insulin.

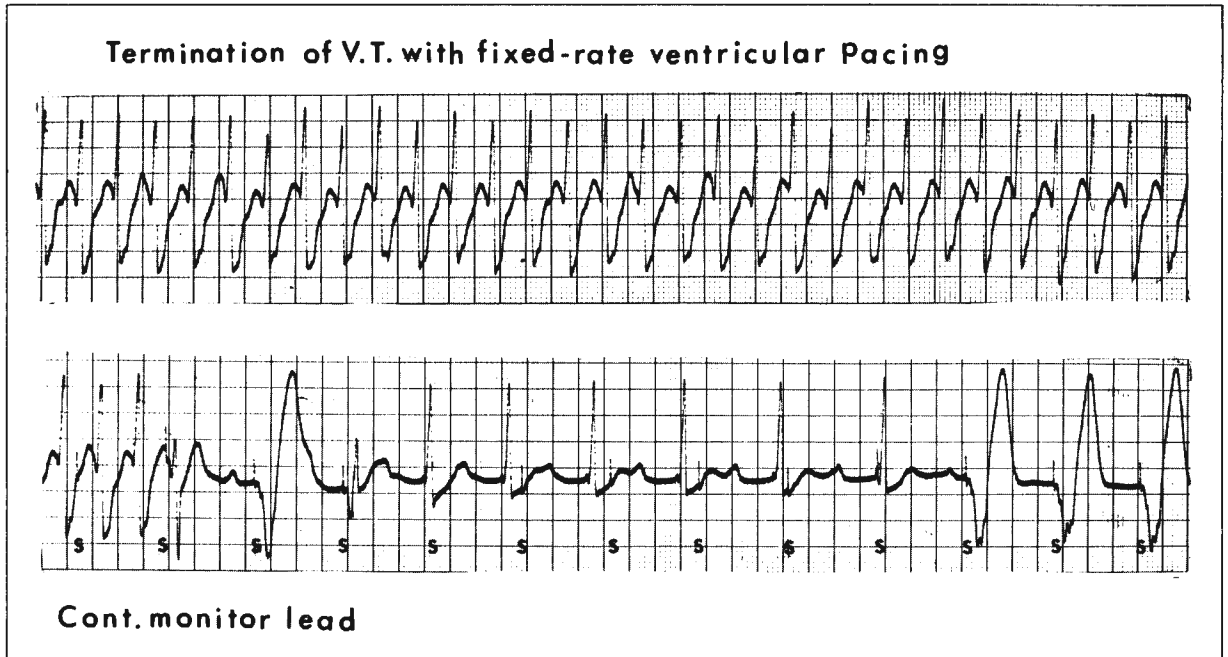


Fig. 6—Monitor lead of a 42-year-old man with ventricular tachycardia terminated by transvenous right ventricular pacing (see text).