the A-V block occurs at the level of the bundle branches the deficit in blood supply must be in both the anterior and posterior circulations of the heart since branches to this area are supplied from both anterior and posterior perforating arteries. Thus involvement of both bundles necessarily implies extensive myocardial damage.1-4

In our experience, patients with A-V block having a widened QRS all developed cardiac failure or shock even if these complications were not present at the moment of the onset of the block. Regardless of the electrocardiographic location of the infarction, the mortality rate despite pacing is about 90%.

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References

The author replies:
I feel that this view is too simplified to be of practical importance. It is generally known that the QRS duration is important in prognosis. However, it is certainly not our experience that all patients with wide QRS develop failure or shock, or that the related mortality is “about 90%.” Our studies regarding experiences in this area have been reported in a subsequent issue of Circulation. Any effective subgrouping will require use of more than just one descriptor. It is for this reason that we included location of infarction, degree of heart failure experienced by the patient, and site of A-V block as criteria for inclusion within the subgroup that we reported. It may be necessary, in fact, for a fourth or fifth descriptor to be added on a larger number of patients to be able to accurately choose the best management for the individual patient.

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References

Bretylium and Ventricular Arrhythmias

To the Editor:
The paper “Deleterious Effects of Bretylium in Cats with Digitalis-induced Ventricular Tachycardia” by Gillis, Clancy and Anderson (Circulation 47: 974, 1973) documents the worsening of ventricular arrhythmias which bretylium can induce in the anesthetized cat intoxicated with deslanoside. The purposes of this letter are threefold: (1) to support the authors’ conclusion that the catecholamine releasing action is related to the ventricular arrhythmias bretylium produces, (2) to speculate on how bretylium aggravates ventricular arrhythmias, and (3) to set these observations in the cat into clinical perspective.

The communication by Gillis and coworkers shows deleterious effects of bretylium on ventricular tachycardia caused by excess digitalis. A number of mechanisms can produce ventricular tachycardia in digitalis toxicity or otherwise. These include: (1) enhanced normal automaticity, (2) abnormal automaticity in Purkinje fibers and (3) simple or complex forms of reentry in Purkinje fibers and/or ventricular muscle.1,2 The data presented by Gillis, Clancy and Anderson suggest that bretylium may aggravate changes in automaticity induced by bretylium. Two previous studies evaluated the effects of bretylium on normal automaticity in isolated preparations of mammalian cardiac Purkinje fibers and ventricular muscle cells.3,4 These studies showed that bretylium induces or increases the rate of spontaneous firing in these preparations for 10 to 15 minutes, after which it has a variable effect on rate.5,4 Low concentrations of quinidine were shown to have the opposite effect, i.e., to reduce automaticity in the same preparations.4 The ability of bretylium to enhance or evoke automaticity in cardiac Purkinje fibers is completely nullified by pretreatment with propranolol or reserpine;3,4 this finding suggests that the augmentation of automaticity bretylium is due to release of norepinephrine (NE) from the postganglionic sympathetic nerve terminals in the isolated preparations.

How bretylium-induced NE release from sympathetic nerve endings in ventricular tissues might aggravate ventricular arrhythmias is not certain. Several effects of catecholamines on mammalian ventricular tissues are almost certainly relevant; the catecholamines can: (1) enhance Purkinje fiber automaticity by causing potassium current (IK) to decline more rapidly during diastole,5 (2) hyperpolarize depolarized cardiac tissues, either by increasing IK or by increasing electrogenic Na+ pumping, particularly when depolarization is produced by stretch,8 (3) induce slowly propagating active potentials in depolarized tissues (active responses which are qualitatively different from the normal action potential)7 and (4) augment the diastolic oscillations (“abnormal automaticity”) seen in digitalis toxicity.8 Any one or combination of these effects could under appropriate conditions result in ventricular tachycardia. Which of these mechanisms leads to the aggravation of digitalis-induced arrhythmias reported by Gillis, Clancy and Anderson remains to be elucidated.

Circulation, Volume XLVIII, November 1973
Duration of QRS Complex: The author replies:
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Circulation. 1973;48:1158
doi: 10.1161/01.CIR.48.5.1158

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