Normal Intraventricular Conduction following Post-Extrasystolic Pause in Bundle Branch Block; with Critical Rate Phenomenon

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The patient, a 47-year-old white woman, had been treated in the hospital seven years ago because of thyrotoxicosis and thyrotoxic heart disease, and received radioactive iodine. She was readmitted January 12, 1962 because of palpitations. Examination revealed mild exophthalmus, blood pressure 120/70 to 150/100; heart sounds were not remarkable, rate 115; no evidence of right or left sided heart failure. X-rays showed cardiac enlargement.

Electrocardiograms: (1) April 19, 1955. Sinus tachycardia, rate 120, otherwise normal (not illustrated).
(2) January 9, 1962 (Fig. 1). Sinus tachycardia, rate 111, left bundle branch block, frequent ventricular premature beats. A fully compensatory pause, 0.70 second, is present, but this is not long enough for the block to disappear in the post extrasystolic beat.
(3) January 12, 1962 (Fig. 2). Sinus rhythm, rate 77. P in V₁ is diphasic +1.0, −0.8 mm.; P-R, 0.14 second; S in V₃ is 36 mm., R in V₉=9 mm., QRS duration 0.12 second; intrinsicoid deflection time (to R' peak) in V₆=0.095 second. Frequent premature ventricular beats from right ventricle with fixed coupling at 0.39 second and full compensatory pause 1.18 seconds. Of special note is the critical rate phenomenon in ventricular conduction. A beat with normal intraventricular conduction occurs only after the long post-extra-systolic pause with R-R of 1.18 seconds, and not after the post-extra-systolic pause of 0.70 second of the previous tracing (Fig. 1), or regular R-R of 0.78 second of present tracing. The critical cardiac rate is between 51 and 77 per minute. The sequence of extrasystole, compensatory pause, and then a normally conducted beat occurred 16 times in the 12 lead record.

Also of note in the tracing are: (1) ventriculo-phasic sinus arrhythmia, P-P between regular bundle branch block beats=0.77 second; P-P containing a bundle branch block beat plus extrasystole=0.71 second; P-P of post extrasystolic pause, with no ventricular complexes, 0.88 second.

Figure 2
(2) The post-extrasystolic normal QRS complex is followed by an inverted T in leads V₂, V₃, and V₄ suggesting myocardial ischemia—not shown in the 1955 tracing, or by bundle branch block alone except possibly for the upright T with small upright and wide QRS in V₅.

(3) The amplitude of the post-extrasystolic QRS (V₂) is only 20 mm. compared to 36 mm. for the regular aberrant QRS. If the latter represents left ventricular hypertrophy this suggests a component of bundle branch block to explain some of the aberration of the ventricular complex in left ventricular hypertrophy.

GLYCOSEURIA AND DIABETES MELLITUS FOLLOWING INH THERAPY

In certain cases, isoniazid can apparently inhibit the release of endogenous insulin which is again forthcoming when the drug is withdrawn. In other cases, the supply of endogenous insulin will return only when stimulated by tolbutamide. The possibility of glycosuria occurring during isoniazid administration and the potential danger of giving this drug to diabetics must always be considered. Three cases are reported.


FAMILIAL CARDIOMYOPATHY

A group of families, the descendents of one couple through four generations, is described. In these families, nine cases of familial cardiomyopathy were discovered, and six other probable cases. Five of these 15 have died suddenly, while two lived one day and a third was stillborn. The remaining seven are still alive, and four have no symptoms. The environment of these families is discussed by the authors as well as the clinical, radiologic, electrocardiographic and pathologic findings. The mode of inheritance in these families appears to be due to a Mendelian dominant gene with incomplete penetrance. The possibility of a recessive gene being responsible has been eliminated as far as possible. A metabolic defect in cardiac muscle has been demonstrated in two of the cases coming to necropsy. This consisted of the finding of a non-metachromatic neutral polysaccharide in the cardiac muscle fibers. It is suggested that the cause of this condition is an inherited metabolic defect in cardiac muscle.


ARRHYTHMIA IN CHRONIC COR PULMONALE

The clinical records and electrocardiograms of 80 patients suffering from chronic cor pulmonale are reviewed from the standpoint of incidence of arrhythmias. Some type of rhythm disturbance was found in 42.2 per cent of the cases, indicating that arrhythmias are a common finding in this condition. The possible determining factors are considered, stressing the frequency of digitalis-induced arrhythmias which calls for caution when using the drug in these patients.