narrow complex exceeded those preceding a wide QRS complex. One of these three atypical cycles is illustrated in the upper panel of Figure 1 (sixth beat), whereas the other cycles of Figure 1 illustrate the rate-dependent widening of the QRS complex, as observed in the other 197 cycles. The mean lengths of the cycles preceding the two forms of the QRS complex were significantly different from each other (P < 0.001).

**Discussion**

Delays in conduction following acute myocardial infarction may be due to any of several pathogeneses. The specific conducting bundles may be rendered ischemic or necrotic as a direct correlate of myocardial necrosis, or the delay may result from circuitous activation of surviving cells within and around the scarred zone. Both types have been labeled "peri-infarction block," the former by Grant and the latter by Wilson et al, First et al, and others.

The present case may represent an uncommon combination of these two mechanisms of block. Fixed left axis deviation is generally considered to reflect aberrations in the proximal conducting bundles, either in the left anterior superior fascicle or within the main left bundle branch itself. Excessive widening of the QRS complex, here observed intermittently as a function of the length of the cycle, mandates the coexistence of a second abnormality of conduction. In the absence of complete bundle-branch block, a focal and peripheral form of block is likely. This may occur in the peripheral Purkinje network or within the intramural myocardial fiber bundles. Data presented by Durrer et al support the latter mechanism as the cause of aberrations in the QRS complex after myocardial infarction, as no delay was detected in the activation of endocardial Purkinje fibers.

Several notions support the diagnosis of a focal peripheral form of postinfarctional block as the second abnormality. First, a prior myocardial infarction with scarring, as occurred in this case, is the prototypical model for abnormal intramural conduction. Secondly, the widening of the QRS complex with short lengths of the preceding cycles was related exclusively to prolongation of the terminal components of the wave-form. This absence of changes in the initial force has been considered to be a diagnostic criterion for focal block by both Rosenbaum et al and Castellanos et al. Thirdly, the constancy of the mean electrical axis in the two types of QRS complexes is consistent with a focal block, the lesion being too localized to significantly divert the mean depolarization vector.

The mechanism for the tachycardia-dependent nature of this focal block is likely to be similar to that invoked for other forms of rate-dependent aberrancy. Depolarization of incompletely repolarized tissue generates a transmembrane potential with reduced amplitude and upstroke velocity, both of which have been associated with slowed conduction of the resultant wavefront. Again, the setting of possible chronic ischemia in the area of the block favors this interpretation; for example, Mandel and associates have documented prolongation of refractory periods in such zones. Thus, an impulse need be less premature to be conducted aberrantly under these conditions than if the refractory periods were of shorter duration. Myocardial damage resulting from chronic disease of the aortic valve may well contribute to these physiologic abnormalities.

**References**


**Idiopathic Atrial Flutter, High Grade Atrioventricular Block and Sino-atrial Dysrhythmia in a Young Man**

**Effects of Exercise Testing**

Raymond J. Aronson, M.B., B.Ch.

A 25-year-old man with idiopathic atrial flutter and high grade atrioventricular (AV) block is described. Postcardioversion, sino-atrial (SA) and AV nodal dysrhythm-
Idiopathic atrial flutter is a rare arrhythmia. In a survey of 122,043 routine electrocardiograms on asymptomatic subjects at an Air Force base, Hiss and Lamb found only five cases of atrial fibrillation and flutter. The association of idiopathic atrial flutter with high grade atrioventricular (AV) block must be an even less frequent occurrence. This report describes a young man with this combination who had evidence of sick sinus syndrome after cardioversion. The amelioratory effects of the treadmill exercise on the arrhythmias both before and after cardioversion are described. A hypothesis is presented attempting to explain these arrhythmias on a neurogenic basis.

**Case Report**

A 25-year-old factory worker who had been well all his life presented with a chief complaint of fatigueability and depression. He had served in the Navy without any medical disability. There was no history of chest pain, dyspnea on exertion, syncope or palpitations. He acknowledged a state of emotional turmoil aggravated by divorce proceedings which he had recently instituted against his wife.

Physical examination revealed a thin, nervous man. Blood pressure was 120/80 mm Hg. The pulse rate was irregular, varying from 40 to 60/min. Flutter waves could be discerned in the neck. The heart was unremarkable except for an irregular apical rate. There was no clinical evidence of thyrotoxicosis. The balance of the examination was unremarkable.

Chest x-ray film findings, urinalysis, complete blood count and serum creatinine, electrolytes and CT4 were normal. Electrocardiogram (Fig 1) demonstrated atrial flutter with an atrial rate of 260/min, AV block varying from 4:1 to 11:1, and RR pauses of up to 2.42 sec. Average ventricular rate was 55/min. The ECG was otherwise unremarkable, except for increased voltages and prominent right precordial R waves, both of which were felt to be normal for his age and build. A 24-hour Holter tape showed the same rhythm throughout. An echocardiogram demonstrated flutter of the left atrium and mitral valve leaflets but otherwise was normal. Specifically, there was no suggestion of mitral valve prolapse.

A graded treadmill exercise test was undertaken. As the exercise.
Throughout qum (Fig 3)* PP marked were exemis block. Four months later demonstrated the sinus was in amduction interval changes. The wrist was repeated. This young man was found to have atrial flutter with action potential 15 (SA) and AV add~on. These were noted by flutter and noted transient 1:1 AV conductimins six

The Treadmill

This young man is unclear. He had no evidence of systemic disease. The lack of chest pain and the patient's treadmill performance seem to rule out coronary artery disease quite convincingly. There was no clinical or echocardiographic evidence of cardiomypathy or valvular disease. Similar cases reported in the literature seem to be of family origin, usually with autosomal dominant inheritance. However, the case reported here appears to be an isolated one: no arrhythmia or AV block was found in the numerous family members studied.

Amat-y-Leon et al§ did pathologic and intracellular action potential studies on atrial tissue obtained from a

FIGURE 2:  oversi on treadmill streas

FIGURE 3. Representative tracings from 24-hour Holter tape post-cardioversion. Upper strip demonstrates marked sinus arrhythmia with SA arrests of up to 1.5 secs. Lower tracing also shows sinus arrhythmia, but with concomitant PR elongation. (It is possible but not certain that a F wave is "buried" in the T wave preceding the longest RR interval, suggesting that this may be an example of Wenckebach block).
lar fibrillation which seemed to be unequivocally psy-
etic. All described a patient with recurrent ventricu-
susyndrome. Moss and McDonald described such a case
where the rhythm at 124/min with normal PR. Lower strip (taken at submaximal exercise) shows further increase in sinus rate to 165/min.

patient with familial atrial arrhythmia and AV block. These demonstrated partially depolarized cells with decreased intra-cellular action potential amplitude, phase 4 diastolic depolarization with spontaneous firing, diminished excitability and responsiveness, and decremental conduction with local block and re-entry. Histologic findings revealed vacuolar degeneration, hypertrophy, and early necrosis of atrial cells.

Although the present case is not familial, it does bear a clinical resemblance to that described by Amat-y-Leon et al. However, the parallel nature of the SA and AV nodal dysfunction, and especially the marked improvement in SA and AV nodal function with exercise, seem to suggest a neurogenic mechanism rather than intrinsic atrial disease. The role of vagal stimulation in inducing atrial flutter in experimental animals is well known, and there have been two clinical reports of atrial flutter induced by carotid sinus pressure. Thus, excessive vagal activity while at rest could explain both the SA and AV brady-arrhythmias, as well as the occurrence of atrial flutter, with the exercise-induced improvement in SA and AV nodal function being due to an increase in sympathetic tone. There are isolated references in the literature to possibly similar situations. In 1952, Benedict and Evans described Wenckebach phenomenon in an apparently healthy medical student in whom the arrhythmia seemed to be a direct concomitant of acute anxiety attacks. The frequency of Wenckebach type block in top performance athletes (Meytes et al) is also thought to be due to a neurogenic mechanism. There is recent convincing evidence that autonomic dysfunction can cause potentially lethal ventricular arrhythmias, especially those related to the prolonged QT syndrome. Moss and McDonald described such a case where left stellate ganglion section was curative. Lown et al have described a patient with recurrent ventricular fibrillation which seemed to be unequivocably psychogenic in origin.

The patient under discussion in this report was clearly in a state of emotional turmoil during the period he was being studied. Although no case of "psychogenic atrial flutter" has been postulated previously, it seems reasonable to relate this man's unusual arrhythmias to his labile emotional state. The mechanism of the arrhythmia may be of prognostic and therapeutic importance: the similar (albeit familial) patient described by Amat-y-Leon et al, in whom unequivocal intrinsic atrial disease was demonstrated, had a complicated course culminating in permanent pacemaker placement. Presumably, a patient with "vagogenic" supraventricular arrhythmias which are ameliorated by exercise should have a benign prognosis. The long-term follow-up of this patient should help to resolve this question.

The usefulness of stress testing in the evaluation of arrhythmias is emphasized by this case. This patient's management and the decision not to undertake more complex hemodynamic studies hinged upon the knowledge that his cardiac performance under conditions of stress was more than satisfactory. The noninvasive and physiologic aspects of such an evaluation commend its wider application.

REFERENCES