Atrial Flutter with Exit Block

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SUMMARY The mechanism of atrial flutter is controversial. A 76-year-old woman with rheumatic heart disease was referred to our clinic with an unusual rhythm disturbance which initially appeared to be classic atrial flutter at a rate of 300 beats/min. Later tracings, however, demonstrated a rate exactly one-half that of the earlier ECGs, with an identical p-wave morphology and vector. This latter rhythm also behaved in a manner expected for a flutter mechanism in that both spontaneously and with carotid pressure high-degree atrioventricular block occurred without alteration of the underlying atrial mechanism. Finally, the two rates interchanged spontaneously over several days without any significant interval changes in medical therapy.

These findings were initially explained as probable digoxin toxicity. The underlying mechanism, however, was more likely atrial flutter with exit block and in this patient may have represented another facet of her sick sinus syndrome. This unusual phenomenon is discussed in terms of previous reports and possible implications for the mechanism of atrial flutter.

Atrial flutter is an enigmatic rhythm disturbance incompletely understood and often refractory to a variety of therapies. As pointed out by Marriott,1 no clear-cut definition distinguishes flutter from other forms of atrial tachycardia. Moreover, the development of a rapid supraventricular tachycardia with atrioventricular (AV) block in a patient taking digoxin leads to additional problems. The question often arises as to whether this represents paroxysmal atrial tachycardia (PAT) with block2 or more rarely, atrial flutter as a manifestation of digoxin toxicity.3,4

Recently an elderly patient presented to us with a recurrent supraventricular tachycardia (SVT) with several unusual features. The initial rhythm appeared to be classic atrial flutter at a rate of 290–300 beats/min with 2:1 AV block. Subsequent tracings, however, showed a rate of exactly half that of the initial rhythm, with an identical p-wave morphology and vector. Although this latter rhythm was initially interpreted to be digoxin-related PAT with block, it probably represented atrial flutter with a 2:1 exit block from the flutter focus. The development of exit block in atrial tachysystole simulating flutter has been clearly described in one patient,5 and atrial tachycardia with exit block has been reported rarely.6,10 This case is an example of a phenomenon only rarely reported previously. It presented an intriguing differential diagnosis in terms of possible digoxin-related PAT with block.

Case Report

The patient was a 76-year-old white female treated at Massachusetts General Hospital for several years. For longer than 10 years, she had had documented mild aortic regurgitation and mitral stenosis and has been in atrial flutter or fibrillation throughout this period. In the past 6 months, several emergency ward visits have been precipitated by either severe dizziness, a frank syncopal episode, or more frequently by marked dyspnea. Each time, a rapid SVT intervened, typically atrial flutter at a rate of 290 beats/min with 2:1 AV block (fig. 1). However, in September 1977, an SVT at a rate of 145 beats/min with 2:1 AV block was noted (fig. 2). Distinct p waves were present with a constant PR interval. Initially, PAT with block was diagnosed and we suspected digoxin toxicity. However, the patient's digoxin level was 0.8 and 0.6 ng/ml (normal 1.2 ± 0.4 ng/ml) on two measurements and unchanged from previous levels when the rhythm had been in atrial flutter. No interval change in medications had been made. The serum potassium level was stable at 4.0–4.5 mEq/l. During the next few weeks, on several occasions, the rhythm spontaneously reverted from atrial flutter at a rate of 290–300 beats/min with 2:1 AV block to SVT with 2:1 AV block and at times 1:1 conduction. Each time at the slower atrial rate of 145 beats/min, p-wave vector and morphology were identical to that noted with atrial flutter at a rate of 290. In addition, on at least two occasions, the atrial rate was exactly half that of the flutter rate recorded before or just after its occurrence. Moreover, high-grade AV block occurred spontaneously, as well as with carotid sinus pressure, both during clear-cut atrial flutter at a rate of 290 beats/min (fig. 3) and with the SVT at a rate of 145 beats/min (fig. 4). The rhythm strip in figure 3 shows the more classic "sawtooth" pattern characteristic of flutter.

A low total and free T4 and a high thyrotropin were documented during this same period, but the patient was not treated initially because she had no symptoms of hypothyroidism. In addition, considering her age and underlying heart disease, we feared that more refractory episodes of tachycardia would occur. Eventually we realized that these episodes of SVT did not represent excessive digoxin therapy, and the digoxin dose was increased from 0.25 mg/day to 0.375 mg/day. The patient then developed higher degrees of AV block with a ventricular response as slow as 30–40.

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Therefore, a permanent ventricular pacemaker was implanted and the patient was maintained on digoxin 0.375 mg/day, resulting in a serum level of 1.0–1.2 ng/ml. Her rhythm is now predominantly v-paced, with associated atrial flutter or SVT with high-degree AV block.

The series of rhythm disturbances presented here illustrates at least three unusual features of atrial flutter. First, the spontaneous development of apparent flutter with 2:1 exit block from the flutter focus was observed. That this was the mechanism underlying the observed tachycardia at a rate of 145 beats/min is supported by these observations. First, this rate is one-half that of the underlying flutter rate of 290 beats/min, with an identical p-wave morphology and apparent vector. Second, each of the rhythms (the typical flutter at a rate of 290 beats/min and with exit block at a rate of 145 beats/min) occurred spontaneously with progression from one to the other over several days with repeated documentation and without any significant interval change in medical therapy. Third, both spontaneously and with carotid pressure at the atrial rate of 145 beats/min, high-degree AV block occurred without alteration of the underlying atrial mechanism. This behavior is that predicted for a flutter mechanism. It is particularly interesting to compare elements of this with the report of Dressler et al., in which they describe the association of PAT with exit block. In their patient, an intrinsic atrial rate of 300 beats/min was documented with the spontaneous development of 2:1 exit block in a fashion similar to our case. Furthermore, their rhythm strips show a p-wave morphology with peaked p waves in V1 identical to those of our patient, suggesting a similar site for an ectopic focus. They also point out the difficulty in determining whether the underlying rhythm in their patient was flutter or PAT. At times, the baseline in their patient’s ECG was isoelectric and other times, it was clearly “sawtooth.” We noted a similar pattern in our patient (compare figs. 1 and 3).

The second interesting feature of this case is the confusion introduced into the patient’s management by the repeated occurrence of the flutter mechanism with exit block. Since control of her ventricular response was critically important in her management, digitalis had a major therapeutic role. Consequently,
mechanism for atrial flutter, the apparent development of exit block in atrial flutter lends support to the hypothesis that an automatic pacemaker is an alternative mechanism in some patients. In addition to older published work supporting this etiology (including vector analysis, simulated pacing studies, and atrial mapping), Waldo et al. have recently demonstrated that it is possible to depolarize portions of the atrium with surgically implanted wires during spontaneous flutter. This supports the existence of either an automatic focus or, alternatively, a small reentrant loop that does not travel throughout the atrium. The finding of flutter with exit block in this patient is most consistent with either a discrete automatic focus or possibly a small circumscribed reentrant loop. This latter mechanism has been suggested by the work of Allessie et al., in which a small localized reentry process was demonstrated to mimic a process of rapid impulse formation. Support for an ectopic focus in our patient stems from the finding that the p-wave vector and in particular its morphology in V1 is quite similar to that reported by Mirowski and Alkan to represent an underlying left atrial automatic focus.

Our patient is well-controlled on higher-dose digoxin therapy with concomitant ventricular pacing. Both our patient and that of Javier et al. eventually required v-demand pacing secondary to the development of high-degree AV block with slow ventricular responses. This unusual syndrome of atrial tachysystole with exit block may represent yet another facet of the more general sick sinus syndrome. The role that hypothyroidism might have played in leading to this unusual form of exit block is difficult to evaluate.

References

Figure 3. High-degree atrioventricular block recorded during an episode of typical flutter with same p-wave morphology. Atrial rate again is 290-300 beats/min.

With the rhythm disturbance noted above, PAT with block reflecting possible digoxin toxicity was repeatedly diagnosed. Although atrial tachysystole with exit block has been associated with possible digoxin toxicity, simultaneous digoxin levels in our patient were relatively low (0.4-0.8 ng/ml on multiple determinations) with normal electrolytes and, in particular, normal potassium levels. It was not until we discovered the underlying mechanism for this rhythm that a more appropriate approach to digoxin therapy was initiated. In fact, control of the patient’s rhythm was finally achieved with ventricular pacing and higher-dose digoxin therapy (0.375 mg/day), which resulted in stable high-degree AV block. Previous attempts to induce and maintain either sinus rhythm or stable atrial fibrillation had been unsuccessful.

Although circus movement using specialized atrial conduction pathways is thought to be the primary

Figure 4. High-degree atrioventricular block during episode of supraventricular tachycardia with same p-wave morphology noted above with atrial rate of 145-150 beats/min using a chest monitoring lead similar to lead V1.
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