Atrial Tachysystole (Flutter?)
with Apparent Exit Block

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SUMMARY
A case of atrial tachysystole with apparent exit block is reported in which the basic cycle length of atrial activity is 260 msec. Simultaneous right intra-atrial electrograms show cycle lengths of 510 to 780 msec which are roughly multiples of 260 msec establishing the diagnosis of exit block. Rapid atrial stimulation (1,200/min) to overdrive the rapid ectopic atrial pacemaker was only transiently successful in converting the arrhythmia to atrial fibrillation with a slower ventricular response. Two days later the rhythm converted to normal sinus rhythm (cycle length, 600 msec) with periods of sino-atrial block measuring 2,400 and 1,240 msec. Atrial rate further slowed so that the patient ultimately required a permanent demand pacemaker.

The case supports the theory of repetitive discharge from an ectopic atrial pacemaker to explain the mechanism producing atrial tachysystoles that simulate atrial flutter.

Additional Indexing Word:
Atrial tachycardia

The concept of exit block, as originally described by Kaufmann and Rothberger in 1920,1 constitutes one of the basic tenets in the explanation of the abnormal unidirectional conduction of an ectopic pacemaker in parasystole. The occurrence of exit block in paroxysmal atrial tachyarrhythmia, however, is rare; only two cases of exit block have been reported in the literature of paroxysmal atrial tachycardia.2,3 To the best of our knowledge exit block in atrial tachysystole resembling atrial flutter has never been described.

We present a rare and interesting case of atrial tachysystole simulating atrial flutter with exit block as documented by unipolar and bipolar right intra-atrial electrograms in a postsurgical cardiac patient who subsequently showed sino-atrial block after conversion to normal sinus rhythm. A permanent QRS synchronous demand pacemaker was subsequently inserted because of a slow ventricular rate.

Report of Case
V. H., a 61-year-old white woman, was admitted to Mount Sinai Hospital on December 2, 1968, with a clinical diagnosis of rheumatic heart disease, cardiomegaly, mitral stenosis, mild aortic regurgitation, and atrial flutter, class III-D. Cardiac catheterization performed a week later confirmed the diagnosis of severe mitral stenosis and moderate aortic insufficiency. She subsequently underwent mitral valve excision and replacement with a prosthesis on December 27, 1968.

On admission, the electrocardiogram (ECG) showed atrial flutter with varying degrees of atrioventricular (A-V) block, the atrial rate being 231/min (fig. 1A). Twelve days after surgery the rhythm was the same, but there were pauses which measured 520 to 1,040 msec during which there were no flutter waves (fig. 1B). Exit block of the ectopic atrial impulses was suspected, and therefore simultaneous recordings of right intra-atrial bipolar and unipolar electrograms were obtained via an electrode catheter together with a lead II ECG, as has been previously described.4 These revealed atrial activity at a basic rate of 231/min (fig. 2A and B). Long intervals with no atrial activity, ranging from 510 to 780 msec, were roughly twice and three times that of the basic cycle length of 260 msec. This supported the clinical suspicion of exit block from the ectopic atrial pacemaker which prevented the impulses from reaching the atrial...
myocardium. Unequivocal confirmation of the exit block could have been obtained only by recording the depolarization of the pacemaker at its origin and then showing the block of the pacemaker impulses from surrounding multiple recording sites.

During carotid sinus massage, the long pauses were abolished and regular atrial activity without exit block was apparent with cycle lengths of 260 msec (fig. 2C). It is well known that vagal stimulation shortens the refractory period of the atrial tissues so that more impulses are able to reach the atrial myocardium.

In view of the rapid ventricular rate (120/min) an attempt was made to convert the atrial flutter to atrial fibrillation with a slower ventricular rate or to normal sinus rhythm by overdriving the atrium. This was accomplished by connecting the electrode catheter to a stimulator (Cordis Synchrocort II) and applying rapid single pulse stimulation to the atrium at 5 milliamperes. The atrium was paced at rates starting from 300 to 1,200/min. The rhythm was converted to atrial fibrillation after 14 sec of rapid stimulation at 1,200/min (fig. 3B and C). However, the next day the rhythm reverted to that of atrial flutter, and repeat rapid atrial stimulation at a rate of 1,200/min failed to convert the rhythm to atrial fibrillation. Two days later, after having received digitalis, the patient reverted to a normal sinus rhythm (fig. 1C). P-P cycle lengths measured 600 msec, corresponding to an atrial rate of 100/min.

Figure 1

Lead II electrocardiograms of patient. (A) Control, showing atrial flutter with variable degrees of A-V block. Interatrial cycle length is 260 msec corresponding to an atrial rate of 231/min. Ventricular rate (VR) is 64/min.

(B) Long pauses with no evidence of atrial activity as compared to A. Pauses measure 520 and 1040 msec, which correspond to two to four times the basic cycle length of 260 msec, which is the least common denominator.

(C) Lead II ECG obtained 5 days after B. Patient has converted to normal sinus rhythm. Cycle length of 600 msec corresponds to an atrial rate of 100/min. Two long pauses measuring 2,400 and 1,240 msec, respectively, are periods of sino-atrial block. They are four times and two times the basic cycle length of 600 msec.
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Figure 2
Simultaneous tracings of lead II (L-2) of the surface electrocardiogram, intra-atrial bipolar (ABE) and unipolar electrogram (AUE) taken on the same day as figure 1B. Tracings were recorded at a paper speed of 50 mm/sec with 1 sec photographic time lines. Lead II electrocardiogram shows atrial flutter. (A and B) ABE and AUE reveal smaller inter-atrial (a-a) cycle lengths of 250 to 270 msec corresponding to atrial depolarization at a rate of 222 to 240/min. The longer a-a cycle lengths of 510 to 780 msec are roughly two times and three times that of the basic cycle length of 260 msec corresponding to an atrial rate of 231/min. There is, therefore, an exit block from the ectopic atrial pacemaker which prevents the impulses from reaching the atrial myocardium. (C) ABE and AUE showed fixed a-a cycle lengths of 260 msec with no exit block during carotid sinus massage. Atrial rate is 231/min and ventricular rate is 115/min (2:1 A-V response).

Discussion
Exit block has been defined as failure of an ectopic impulse, usually from the A-V junction or the ventricles, to propagate to the adjacent myocardium. Its occurrence in atrial tachysystoles is rare. Exit block has been likened to the abnormally increased refractory periods created around an ectopic pacemaker by its own impulses and those of the primary pacemaker. They may be intermittent, and their absence may lead to control by the rapidly discharging ectopic center.

Atrial tachycardia and atrial flutter are two of the common atrial tachyarrhythmias, and their differentiation has always been the
subject of discussion. One group contends that a basic difference exists and that it is possible to differentiate the two.6,7 Lewis8 stated that the most important criterion for the electrocardiographic diagnosis of atrial flutter is the continuously moving base line as seen in most leads. Paroxysmal atrial tachycardia, in contrast, manifests quiescent intervals between P waves. To him the atrial flutter range is 220 to 370/min. Prinzmetal et al.9 who consider both rhythms to be produced by an ectopic pacemaker discharging at a rate faster than the sinus node, but slower than atrial fibrillation, classify atrial flutter as a form of atrial tachycardia. They consider the undulating F wave of atrial flutter to be specific for the arrhythmia with an atrial rate of 260 to 320/min. Others maintain that there is no clear-cut separation between the two and, therefore, propose the term “atrial tachysystole.”10

We believe in a unitary concept to explain the mechanism of the two arrhythmias and therefore prefer to use the term “atrial tachysystole.” In the case reported herein, intermittent exit block prevented capture of the atrial myocardium by a rapidly discharging ectopic atrial pacemaker.

Figure 3
Intra-atrial unipolar (AUE) and bipolar (ABE) electrograms and lead II electrocardiogram. All recorded on the same day as figure 2.

(A) Simultaneous tracings of lead II, intra-atrial unipolar and bipolar electrograms. The latter have been recorded at filter settings different from that of figure 1. Tracings reveal a-a cycle lengths of 240 to 250 msec, corresponding to atrial activity of 240 to 250/min. There is a ventricular response to every two atrial impulses.

(B) Right atrium is being rapidly paced by single pulse stimulation. Pacer impulse (PI) rate is 1,200/min. After 14 sec of rapid stimulation, ventricular activity becomes irregular suggesting conversion to atrial fibrillation.

(C) Simultaneous tracings of lead II, AUE, and ABE reveal and confirm conversion to atrial fibrillation.
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References
Minuscule Review

The Possible Facade of Unsupervised Electrocardiographic Monitoring


When a medical advance has gained universal recognition, it is often difficult to identify and assign relative importances to the various factors in the management of the patient which have resulted in favorable changes. This is so even if the data documenting it are revealed in mortality rates. No one would now minimize the salutary contribution of electrocardiographic monitoring to the care of the patient with acute myocardial infarction. Such monitoring has documented the alarming frequency with which arrhythmias occur and has often alerted the staff to carry out lifesaving procedures promptly which would have been ineffective or less effective, if delayed for only a few moments. At the time of the introduction of the coronary care unit, there were some who believed that a system of electrocardiographic monitoring with an alarm signal could suffice, even if isolated at the bedside of patients and it was not necessary to concentrate the patients in a special care area, provided that there was a plethora of nurses and residents on the ward.

It has seemed to have been the universal experience that the most important person in the 24-hour care of the patient with acute myocardial infarction is the nurse who has been trained to recognize threatening catastrophes and who is able to initiate proper therapy. The adequate supervision of coronary care units requires dedicated vigilance; otherwise the electrocardiographic monitoring equipment and playbacks may not be utilized to their maximal advantage.

Dr. Hubner and his associates report that routine cardiac monitoring in the management of acute myocardial infarction outside a coronary care unit gives minimal aid to therapy, in that cardiac monitoring with ECG oscilloscopes, in the general medical ward apparently did not decrease the mortality rate as compared with therapy in the same type of ward and hospital without monitoring. Their summary states, "A mortality of 25% for acute myocardial infarction was the same for a hospital without a coronary unit where monitoring was routinely performed and for two neighbouring hospitals which did not routinely use monitoring during the period of analysis." The authors point out that monitors do stimulate interest in the detection of arrhythmias. Thus, monitors are useful, but their limited contribution to modification of mortality rate is emphasized. The authors believe that the important factor in the decrease of mortality rate in coronary care units is related to the almost continuous observation of the patient and monitoring by trained personnel.

The need to sponsor continuing training programs for nurses and paramedical personnel is underscored. These are the people who make the remarkable practical contribution and who become invaluable in any coronary care setting.

H.B.B.
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Circulation. 1969;40:179-184
doi: 10.1161/01.CIR.40.2.179

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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