Exit Block Around a Junctional Pacemaker

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An arrhythmia displaying the phenomenon of exit block around a junctional pacemaker in a patient with atrial tachycardia and complete A-V block at the junctional level is described. The importance of the exit block concept for a rational interpretation of cardiac arrhythmias is discussed.

The phenomenon of exit block is defined as a failure of normally elaborated impulses to capture the surrounding myocardium. The block can follow the pattern of progressive delay in conduction (type 1) or occur without warning (type 2). Conceptually, this conduction disorder can interfere with the function of any cardiac pacemaker, including artificial pulse generators. The occurrence of exit block around an A-V junctional focus is, however, not well documented in the literature, the review of which revealed only two relevant references. Because of the significance of the exit block concept for a rational interpretation of some cardiac arrhythmias, we thought it worthwhile to report this recently observed case.

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Description of Tracings

The tracings reproduced in Figures 1 and 2 are rhythm strips from a monitor lead recorded in a 72-year-old man, suspected of having "sick sinus node" syndrome. The patient was on no medication, had no evidence of cardiac or respiratory failure, and had normal serum potassium levels.

Figure 1A shows atrial tachycardia, with a rate of 200/min. Slight variations in the length of the P-R intervals are present on close observation. The QRS complexes are narrow and of normal configuration. The basic ventricular rate is 65/min. On several occasions, however, as shown in the middle portion of this rhythm strip, longer pauses appear, reducing abruptly the rate to 33/min, one-half of the basic frequency.

Figure 1B shows two examples of the same arrhythmia, in which the longer pauses are interrupted by ventricular escape beats. These beats are not followed by a compensatory pause and disrupt the basic QRS rhythm. The interval between the end of the ventricular escape beats and the onset of the next normal QRS complex is similar to the basic R-R interval.

Figure 2, recorded with a different monitor lead, displays the same ventricular rate as in Figure 1. The atrial rate, however, is now 250/min.

Interpretation and Discussion

Because the basic ventricular rate (65/min) was approximately one-third of the atrial rate (200/min), the arrhythmia displayed in Figure 1A was initially interpreted as atrial tachycardia with 3:1 block. The longer pauses between the QRS complexes were explained by increase in the degree of A-V block to 6:1. It seemed strange, however, that no other intermediate degrees of block, such as 4:1 or 5:1, were noted on the numerous rhythm strips recorded. An alternative possibility of complete A-V block with junctional pacemaker driving the ventricles at a rate of 65/min was therefore considered. The longer pauses, which were double the basic R-R interval, could then be explained by the phenomenon of exit block around the junctional pacemaker.

Additional observations supported this second interpretation. Although the slightly different lengths of the P-R intervals noted in Figure 1A already suggested complete dissociation between the atria and the ventricles, this was confirmed when increases in atrial rate up to 250/min (Fig 2) did not alter the basic ventricular rate, which remained at 65/min.

Figure 1A. Atrial tachycardia of 200/min, with complete A-V block. Junctional pacemaker (arrows) is driving heart at rate of 65/min. Longer pauses reflect phenomenon of exit block around junctional pacemaker (open arrows). For further details see text. (B) Two examples of same arrhythmia. Escape ventricular ectopic beats interrupt long pauses and reset junctional pacemaker (arrows). For further details see text.
Also of significance were the changes noted when some of the longer R-R pauses were interrupted by ventricular escape beats (Fig 1B). No compensatory pause followed as long as those beats depolarized the ventricles, the original heart rate resumed. Such a pattern could best be explained as a result of depolarization of the junctional focus by the propagated ectopic ventricular impulse, leading to resetting of the pacemaker. Indeed, if the QRS complexes had been conducted from the atria rather than originating in the A-V junction, one would have expected either no disruption of the ventricular rhythm, or possibly even an abolition of the atrial tachycardia, assuming that such was due to a reentry mechanism.

The electrocardiographic diagnosis of type 1 exit block can be made when the intervals between the QRS complexes become gradually shorter, with the long interval including the blocked beat being shorter than the two preceding intervals. Type 2 exit block, as in our case, should be considered when the long pauses occur unexpectedly and are the multiples, most frequently double but occasionally triple, of the basic cycle length.

It should be emphasized that the failure of normally elaborated impulses to capture the surrounding myocardium reflects a conduction disturbance rather than myocardial inexcitability. Were the conduction normal, the junctional pacemaker would have captured the surrounding myocardium since the impulses arrived outside its refractory period. That the myocardium was excitable is indicated by the ability of the ectopic ventricular focus to be expressed. While the exit block around the junctional pacemaker allowed the ectopic ventricular beat to emerge, there was absence of an entrance block around this focus bringing about its resetting. Thus, the conduction defect seems to be unidirectional.

Initially advanced as an explanation for the failure of parasystolic foci to activate the heart outside its refractory period, exit block is no longer a purely electrocardiographic concept. Recent experimental studies have provided electrophysiologic evidence supporting its existence. Using direct recordings at the cellular level, Greenspan et al. reproduced the phenomenon of exit block in both canine and human preparations. Their observation clearly suggested that failure to capture is most often due to type 1 or 2 block rather than to an alteration of impulse formation. Exit block was also recently elicited by vagal stimulation of the intact heart in open-chested, anesthetized dogs.

At a clinical level, the importance of the exit block phenomenon is well exemplified by the case under consideration. Were the exit block concept not considered, the arrhythmia would not be well understood and an erroneous diagnosis or even therapeutic decisions could easily have been reached. Indeed, if the diagnosis of atrial tachycardia were accepted, either digitalis therapy or cardioversion might have been considered. On the other hand, in a patient with complete heart block and exit block around the junctional focus, artificial pacemaker therapy might have been indicated. At the very least, one would choose to carefully monitor this patient for some time to guard against an untoward slowing of the ventricular rate.

REFERENCES


Enterococcal Lung Abscess: Medical and Surgical Therapy*

James F. Morris, M.D.,** and J. Edward Okies, M.D.†

Enterococci are rarely implicated in the causation of pneumonia or lung abscess. Two patients are reported who developed large lung abscess cavities from enterococcal pneumonia. Combination penicillin and aminoglycoside antimicrobial therapy and drainage with a transthoracic tube resulted in complete cavity closure and healing. Enterococci should be considered as possible etiologic agents of pneumonia. Percutaneous chest tube drainage appears helpful in certain types of lung abscess.

The microbial agents causing pyogenic lung abscess have been identified with increasing specificity in recent years. Greatest attention is being directed lately to the anaerobic organisms, including Bacteroides species, Peptostreptococcus and Fusobacterium. Other causative organisms include Staphylococcus aureus and

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CHEST, 65: 6, JUNE, 1974

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