Name That Beat

To the Editor:

In the article "Wedensky Facilitation: Electrotonic Potentiation during Complete A-V Block" (Chest 1986; 89:557-60), the authors have delivered a big jolt to the credibility of scalar electrocardiography. According to the article, the relatively common phenomenon of sporadic conducted beats in complete A-V block may actually be ventricular escape beats. The authors substantiated their argument by demonstrating shorter H-V intervals for these escape beats. Demonstration of a similar mechanism in a number of similar cases needs to be documented before this new phenomenon can be recognized. One small question: how are the authors going to defend themselves against a puritan's alternative diagnosis of this anecdotal case as a conducted beat with a short H-V interval due to, say, a Mahaim type of pre-excitation,1 due to bypass fibers which are unavailable (for some unknown reason) to the regular junctional beats? Premorbid electrocardiograms of this patient, if available, as well as follow-up studies (especially His bundle studies if patient reverts to normal sinus conduction) may help resolve the mystery in this case.

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REFERENCE


To the Editor:

We thank Dr. Balasubramanian for his interest in our paper. Our purpose is not to challenge the value of standard electrocardiographic examination, but to suggest that sometimes the diagnosis of rhythm disturbances can be far more difficult than appears.

The alternative explanation for our case, based upon conduction of some sinus impulses through Mahaim fibres, is theoretically possible. Indeed, assuming a prolonged refractory period of the A-V node, late P-waves can be conducted. If Mahaim fibers arise from the lower part of the A-V node, the sinus impulses could be conducted to the ventricles with a short H-V interval. Escape impulses originating in a pacemaker distal to the Mahaim fibers would be conducted to the ventricles through the normal pathways. It is unlikely, however, that RB beats are actually conducted beats, as reflected in figure 1. This diagram is relative to a section of the original tracing reported in figure 2 of the paper.

P1 reflects a conducted sinus impulse, and gives rise to the ventricular complex labelled RB. The refractory period of the upper A-V junction is longer than the sinus cycle, since two consecutive RB beats do not occur. Accordingly, P1 is blocked in the upper A-V junction, allowing an escape pacemaker sited below the block to emerge and give rise to beat RA. The escape conducted impulse, however, is not retrogradely conducted to the atria. The sinus P-wave labelled R passes the upper block zone, but is blocked distally since the lower A-V junction is now refractory due to the preceding escape beat. P2, in turn, is blocked in the proximal zone, while P3 is conducted as it occurs outside the refractoriness of both the upper and the lower A-V junction. P4 is then blocked proximally, and P5 is nonconducted, occurring at a short interval from the preceding escape impulse. On the basis of the diagram, P6 cannot be conducted despite its occurrence at a long interval (1.52 sec) from the preceding QRS complex (RA). Thus, beat RB cannot be explained by conduction of P5 despite the fact that such a P-wave occurs at an RA-P interval commonly associated with assumed conduction.

It is worth noting that, in the complete and very long tracing, every one of the P-waves which occurred with an RA-P interval ranging from 1.24 to 1.6 sec were followed by an RB complex. On the basis of the situation postulated in the diagram, however, some of the P waves would have to be non-conducted despite being associated with RA-P intervals which are in the range of conduction. This is because different levels of block occur: one in the upper A-V node, and the other in the lower A-V junction, the latter due to refractoriness induced by the escape beats. Accordingly, it is impossible that the only factor governing the conduction of sinus P-waves is the RA-P interval. This could only occur if the escape impulse reaches the upper site of block, resetting the refractoriness of this zone. But the escape impulse was obviously not conducted into the upper A-V node, since retrograde atrial activation or atrial fusion beats were not observed. Thus, provided that all the P-waves associated with RA-P intervals ranging from 1.24 to 1.6 sec are

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**Figure 1**

<table>
<thead>
<tr>
<th>A</th>
<th>P1</th>
<th>P2</th>
<th>P3</th>
<th>P4</th>
<th>P5</th>
<th>P6</th>
<th>P7</th>
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<td>AVJ</td>
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</tr>
<tr>
<td>V</td>
<td>RB1</td>
<td>2.03</td>
<td>RA1</td>
<td>1.99</td>
<td>RA2</td>
<td>1.81</td>
<td>RB2</td>
<td>2.02</td>
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<tr>
<td></td>
<td>RB1-P2 = 1.04</td>
<td>RA1-P5 = 1.24</td>
<td>RB2-P6 = 0.77</td>
<td>RA3-P8 = 1.52</td>
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</tbody>
</table>

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followed by premature RB beats, we must assume that RB complexes do not express conducted sinus impulses but are, in fact, escape beats, also suggested by their short H-V interval.

The diagram of Figure 1 reflects a 2:1 form of A-V nodal block. This phenomenon, however, would have been the same with any other form of second-degree A-V block provided that two levels of block occur, and no retrograde conduction of the escape impulses is manifested.

Other arguments against conduction of sinus impulses through Mahaim fibers include: no delta wave is evident in RB beats, and the direction of the initial vectors of ventricular activation is the same for both the RA and RB beats.

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Predicting Postpneumonectomy FEV1, Half Empty or Half Full?

To the Editor:

Drs. Ladurie and Ranson-Bitker1 are to be congratulated on reporting one of the largest and longest series of patients in whom FEV1 was predicted postpneumonectomy using split function tests. This technique has been used clinically now for over ten years and seems to have gained some utility and support.2,3 These investigators, however, express disappointment with the accuracy of the FEV1 predicted postoperatively using either bronchospirometry, ventilation/perfusion scanning with multiple detectors, or Anger camera. FEV1 measured one year after pneumonectomy and then annually for up to ten years in some of their 159 patients seemed, to them, to vary excessively from the predicted value. Their results recall the pessimist who views the glass as half empty; is there another viewpoint? If viewed optimistically, 87 percent of their patients were potentially benefitted in that the test was accurate enough to permit a surgical approach to a disease which without surgery has a grim prognosis.4 As with any good study, as many questions are raised as answered.

Was there any difference in predicted compared to observed postpneumonectomy FEV1 in the patients with preoperative FEV1 <2 L vs >2 L? It would be interesting to know this since the patients in the study had a mean FEV1 of 2.4 L. I have never applied (nor advocated) this technique with patients whose FEV1 exceeds 2 L preoperatively.

What were the criteria used to accept or reject patients as to their physiologic operability? Most investigators have used a predicted postpneumonectomy FEV1 of 0.8 to 1.0 L as the lower limit, but we have suggested prospectively testing 30 percent of predicted normal as a possible lower acceptable limit.5

Was there any immediate (<30 days) postoperative cardiorespiratory death? In our experience, despite the prediction, morality in the patients with preoperative FEV1 <2 L may be as high as 18 percent. Having only six patients with difficulty postoperatively and two with cor pulmonale at ten years seems to be an excellent result.

Were all patients maximally treated for their COPD at the time of preoperative (and postoperative) FEV1 measurement, and preoperative reparation? We have seen patients preoperatively who, after therapy, experience improved FEV1, so much as to no longer need split function testing. Is it also possible that the right-left distribution of ventilation and perfusion vary from day to day in COPD? This has not been tested using radionuclide techniques, but is a real problem in using the lateral position test.6

In summary, the prediction of postpneumonectomy FEV1 may not be the best test for predicting postoperative cardiorespiratory function. Its primary advantage is that it seems to work and is less invasive than pulmonary artery balloon occlusion and bronchospirometry, which it supplanted. Will it in turn be supplanted by 152max?7

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References

To the Editor:

Here are some answers to Doctor Olsen's questions. There is no difference in predicted compared to observed postpneumonectomy FEV1 <2 L vs >2 L (FEV1 <2 L, 48 patients, 6 conflicting results; FEV1 >2 L, 111 patients, 29 conflicting results). In our hospital the lower result for operability is around 30 percent of predicted FEV1 value. Of course the predicted values used should be indicated (we use the CECA values, which are rather high) and some patients with only one functional lung have been operated on with a much lower FEV1.

This series concerns the post-surgical FEV1; and all the patients survived at least one year; however, we studied another series of 609 patients who underwent pulmonary resection between 1961 and 1983. Postsurgical mortality inhospitals does not depend significantly on FEV1, being under or above 60 percent of the predicted value. After pneumonectomy, mortality is 15 of 305 (4.9 percent); FEV1 <60 percent, four of 56 (7.1 percent); and FEV1 >60 percent, 11 of 290 (3.8 percent). After lobectomy, mortality is nine of 304 (2.9 percent); FEV1 <60 percent, one of 69 (1.5 percent); and FEV1 >60 percent, eight of 239 (3.4 percent). In the same series, the difference in

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