

# Tachycardia-dependent left posterior hemiblock

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**SUMMARY** A patient with intermittent tachycardia-dependent left posterior hemiblock is reported. Electrocardiographic patterns of complete and incomplete block were documented. Identification of the electrocardiographic characteristics of intraventricular conduction defects is aided when they are intermittent. The difficulty in diagnosing incomplete left posterior hemiblock, and the possible masking of the signs of previous inferior infarction by left posterior hemiblock are emphasised.

We report a patient with intermittent left posterior hemiblock, not associated with other intraventricular conduction defects. Given the rarity with which this type of block has been described and the controversy concerning its identification, meaning, and electrocardiographic characteristics,<sup>1-5</sup> this case is interesting both from a diagnostic and an electrophysiological point of view.

## Case report

A 70-year-old man was admitted because of syncopal attacks. Eight years previously, he had had an acute myocardial infarction. Subsequently he had episodes of angina at rest and on effort about once a month. During the three days before admission he had three episodes of weakness accompanied by sweating and

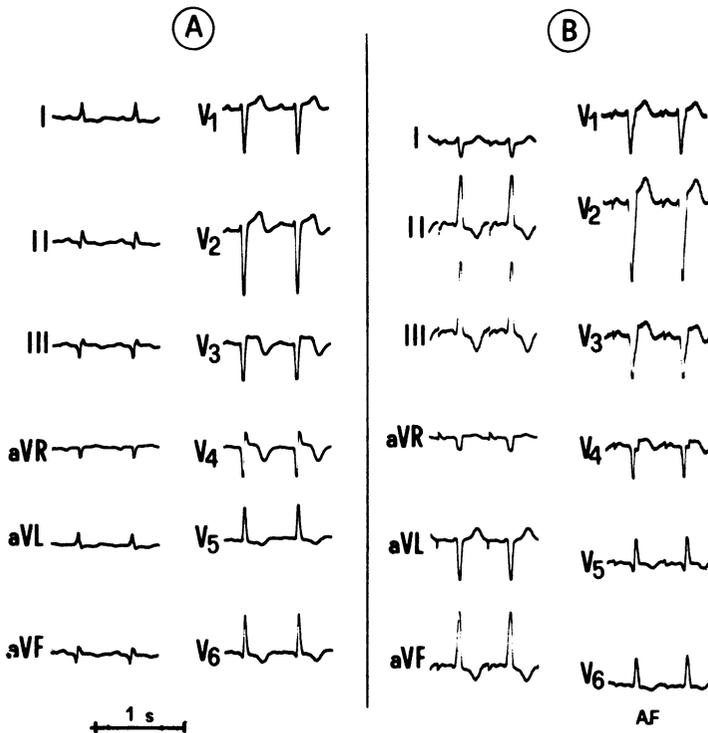


Fig. 1 (A) Twelve-lead electrocardiogram recorded during sinus rhythm in absence of LPH (R-R cycle: 620 ms). (B) Electrocardiogram recorded during atrial pacing in presence of left posterior hemiblock (R-R cycle: 580 ms). See text for further explanation.

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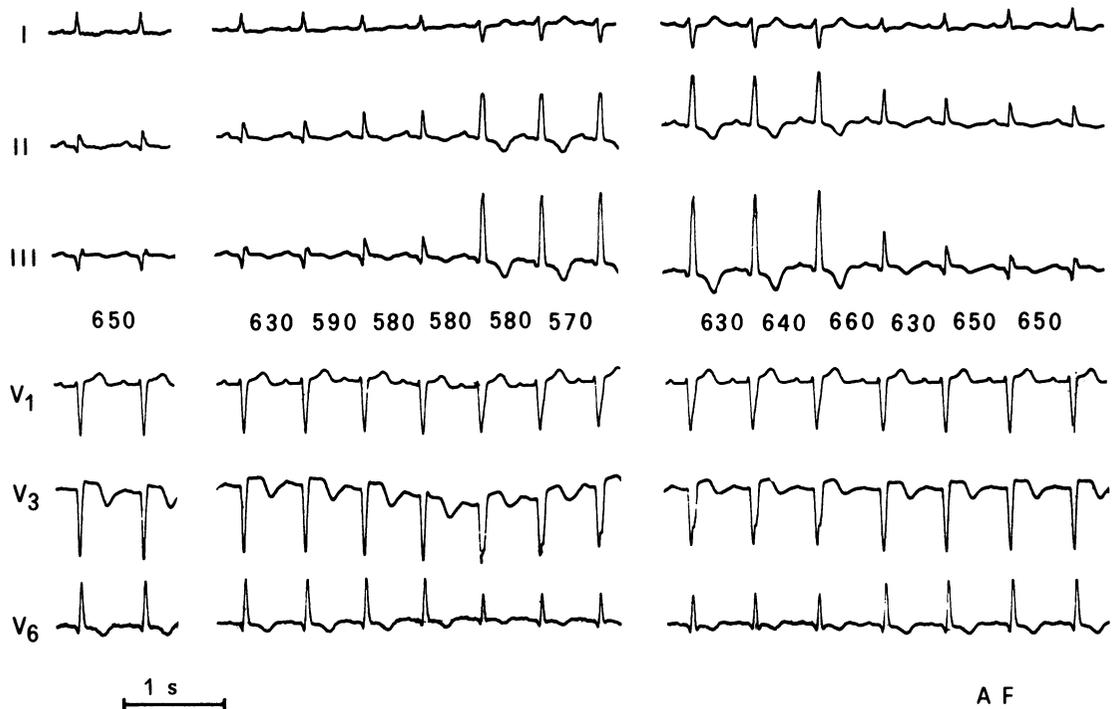


Fig. 2 Intraventricular conduction is normal at a cycle length of 650 ms (left panel); spontaneous increase in sinus rate (middle panel) induces the appearance of left posterior hemiblock, which disappears when heart rate decreases (right panel). Beats showing incomplete left posterior hemiblock are also present. See text for further explanation.

dizziness which lasted three to five minutes and resolved spontaneously, and then two syncopal attacks. Physical examination was unremarkable except for occasional pulmonary rales; no murmurs or abnormal heart sounds were heard. Chest films disclosed moderate cardiac enlargement and calcified plaques in the anterior pericardium. The blood pressure was 130/80 mmHg. During electrocardiographic monitoring episodes of ventricular tachycardia were observed; some resolved rapidly and spontaneously while others lasted longer, caused loss of consciousness, and required cardioversion. After starting oral procainamide (300 mg six times a day) episodes of tachycardia were no longer observed and the patient was discharged after 21 days. On the same treatment at home he did well, but one year later died suddenly.

#### ELECTROCARDIOGRAPHIC FINDINGS

The findings recorded in the absence and presence of left posterior hemiblock, before starting any treatment, are reported in Fig. 1. With normal intraventricular conduction (Fig. 1A) the QRS duration is 0.09 s. The mean QRS axis is about 0°. Pathological Q waves are present in leads II, III, and aVF; in the

precordial leads the R wave decreases from V1 to V3 and a deep Q wave is present in V4. Negative T waves are seen in leads II, III, aVF, V3 to V6, indicating a previous inferior and anteroapical infarction.

Atrial pacing at an increasing rate consistently induced the appearance of left posterior hemiblock at a cycle length of 600 ms. In the presence of hemiblock (Fig. 1B) the QRS duration is 0.12 s. The mean QRS axis is about +110°; a deep S wave appears in leads I and aVL and a tall R wave in leads II, III, and aVF. The Q wave in leads II, III, and aVF is 30 ms wide, with a voltage of about 0.1 mV. Thus, the electrocardiographic signs of inferior infarction are masked by the left posterior hemiblock, while in the precordial leads the signs of the anterior extension of the infarction remain.

In Fig. 2 the spontaneous appearance and disappearance of the left posterior hemiblock and its tachycardia-dependency are illustrated. In the left panel normal intraventricular conduction is present at a cycle length of 650 ms. When the cycle shortens from 630 to 580 ms (middle panel) the Q wave progressively decreases and the R wave increases in voltage in leads II and III, while the opposite occurs when the cycle lengthens from 630 to 650 ms (right

panel). The appearance and disappearance of left posterior hemiblock are preceded by beats showing different degrees of incomplete hemiblock. It should be noted that cycles of equal length (580, 630 ms) terminate with beats showing an electrocardiographic pattern of either a complete or a minimal degree of left posterior hemiblock. The same sequence of events was consistently recorded during atrial pacing at increasing and decreasing rates.

## Discussion

Transient pure left posterior hemiblock is not frequent<sup>1 6 7</sup> and an intermittent one is exceptional.<sup>1 8 9</sup> The reasons are probably multiple. First, according to Rosenbaum *et al.*<sup>1</sup> the posterior division of the left bundle-branch is the least vulnerable of the whole intraventricular conduction system because its blood supply comes from branches of both the left and right coronary arteries and because it is short and relatively large. Moreover, if left posterior hemiblock is not complete, the electrocardiographic pattern is not at all characteristic or impressive and the diagnosis may be difficult or impossible unless the block remits spontaneously. Finally, since the distribution of the divisions of the left bundle-branch may vary conspicuously,<sup>5</sup> it is possible that a distinct posterior fascicle responsible for the Purkinje network in the inferoposterior wall of the left ventricle does not exist in everyone.

The electrocardiographic characteristics of left posterior hemiblock are the object of discussion.<sup>1-4</sup> The pattern of complete hemiblock observed in our case is similar to that described by Rosenbaum *et al.*<sup>1</sup> as well as that observed in cases of transient left posterior hemiblock.<sup>6</sup> As emphasised by Halpern *et al.*,<sup>8</sup> however, a different and often insignificant electrocardiographic pattern can occur with incomplete degrees of block. Fig. 2 suggests that the progressive inferior deviation of the mean QRS axis and particularly the voltage of the R wave in leads II, III, and aVF, is the most sensitive index of an increasing degree of left posterior hemiblock. Since in our case a Q wave was already present in the inferior leads in the absence of hemiblock, the appearance of the latter did not change the initial vector of the QRS complex, an important sign of the diagnosis.<sup>6</sup>

The possibility that left posterior hemiblock may mask, and not only simulate, the electrocardiographic signs of inferior infarction<sup>10 11</sup> is confirmed in our patient even with only incomplete degrees of hemiblock (Fig. 2). In the absence of inferior infarction, activation of the anterolateral wall no longer balanced by activation of the inferoposterior wall, because of the delay caused by the left posterior hemiblock, explains the origin of the Q wave in the inferior leads.

The presence of an inferior infarction would not therefore be expected to modify the pattern of the initial vector in the inferior leads.<sup>11</sup> In contrast, in our case the Q wave decreased in voltage and duration, the change of Q wave amplitude presumably caused by the loss of anterior forces consequent upon the anteroapical infarction.

The appearance, disappearance, and degree of left posterior hemiblock in our patient correlated well with the length of the cardiac cycle. Complete hemiblock was observed in beats ending cycles shorter than those terminated by beats with incomplete hemiblock or normally conducted beats. The electrophysiological interpretation of tachycardia-dependent blocks has already been amply discussed.<sup>9 12-15</sup> Some degree of overlap was observed between cycles terminated by beats presenting a pattern of complete and incomplete hemiblock. This phenomenon, frequent in intermittent intraventricular conduction defects, has also already been discussed.<sup>9 12 13</sup> Various factors can be responsible for it. In our case retrograde conduction of the impulse in the fascicle blocked in an anterograde direction probably played an important role in causing persistence of the conduction defect in cycles longer than those in which it first appeared.

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### Erratum

In the paper by Beach *et al.* on pages 285-9 there was an unfortunate transposition of two lines of type. The first line of the right hand column of p. 285 should be the first line of the left hand column of page 286, and vice versa. The printers apologise for this error.