Transient Left Posterior Hemiblock
Report of Four Cases Induced by Exercise Test

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SUMMARY
Four cases with transient electrocardiographic features which have been attributed to left posterior hemiblock (LPH) are reported. These features were induced by the exercise test in patients with severe coronary artery disease. In all of them the following exercise-induced changes were noted: (1) A shift of the main QRS forces inferiorly and to the right (between +90° and +120°). (2) A definite shift of the initial 0.02 QRS vectors superiorly and to the left, causing a small Q wave to appear in leads II, III, and aVF and/or to disappear from leads I and aVL. (3) A S1Q3 pattern. (4) A leftward displacement of the precordial transition zone. (5) An increase of QRS duration in about 0.02 sec. Gradual disappearance of the exercise-induced axis shift was observed in all four cases and these findings were compatible with multiple degrees of “incomplete” LPH. The occurrence of transient LPH patterns was related to the development of acute, transient injury in the posteroinferior wall of the left ventricle in the presence of segmental or widespread coronary artery disease and chronic posteroinferior damage. Before the exercise test, two patients had electrocardiographic patterns suggesting old myocardial infarction and the other two had repolarization changes related to inferior myocardial ischemia according to the angiographic findings.

Additional Indexing Words:
Hemiblocks Incomplete forms of left posterior hemiblock Intrinjury block Coronary arteriography Transient and intermittent conduction disturbances Exercise test

It has been postulated that left posterior hemiblock (LPH) shifts the main QRS forces inferiorly and to the right, between +90° and +120°. However, since a similar QRS axis direction may occur in right ventricular hypertrophy, pulmonary disease, extensive lateral wall myocardial infarction, and an extremely vertical heart it becomes obvious that the diagnosis of block in the posterior division of the left bundle branch cannot always be made from the ECG alone, except in cases of transient or intermittent LPH, not reported in the literature until now.

While performing exercise tests for evaluation of coronary artery disease in a series of 100 patients, we have been surprised to find that four developed the typical features that have been assumed to be the result of “pure” LPH. These four cases, described below, provide very good material for the study of the electrocardiographic changes induced by this type of fascicular block in the human.

Case Reports

Case 1
A 44-year-old man, of medium body build, was referred because of daily episodes of retrosternal chest pain at rest. An ECG obtained when the patient was free of pain (fig. 1, control) showed slight S-T-segment depression in leads III and aVF, and negative or diphasic T waves in the same leads. This subject performed a maximal exercise tolerance test in the supine position on a bicycle ergometer.* ECG recording was done

*Universal Ergometer, Type UEM 3, Mijnhardt, Odijk, Holland.
using a 12-lead system. Continuous tracings were taken before and during exercise, and for 3 min immediately after exercise. Thereafter recordings were repeated at 1-min intervals for a further period of 12 min. The same method was used for all four cases.

During and immediately after the test, the patient developed S-T-segment elevation in leads II, III, and aVF; S-T depression in leads V_2-V_6; and shift of the QRS axis inferiorly and slightly to the right. Such changes disappeared gradually and the ECG returned to the preexercise features after 12 min (fig. 1).

The exercise-induced changes in QRS complex were as follows: (1) A shift of the main QRS forces inferiorly and to the right, from 0° to +110°. (2) Definite shift of the initial 0.02 QRS vectors superiorly and to the left. (3) Increase of the QRS duration, from 0.08-0.09 to 0.10-0.11 sec, that is, about 0.02 sec. (4) Displacement of the precordial transition zone to the left.

Thirty hours after performing the exercise test, the heart rate was artificially increased, with the intravenous administration of 1.0 mg of atropine sulfate. Conduction disturbances did not occur when the increase in heart rate was similar to that which occurred during the exercise test.

Coronary arteriography revealed a severe segmental stenosis of the midportion of the right coronary artery (fig. 2, top); the left coronary artery was normal. Injection of the contrast medium into the right coronary artery induced ST-T wave changes suggestive of inferior wall ischemia and a shift of the QRS axis inferiorly and to the right, associated with increased voltage of R wave in lead III. No changes in the initial 0.02 QRS vectors have been noted (fig. 2, bottom). The left coronary injection produced repolarization changes suggestive of lateral wall ischemia and a significant left-axis deviation.
Case 1. (Top) Right coronary arteriogram (lateral projection) showing severe segmental stenosis of the midportion (indicated by arrow). (Bottom) Electrocardiographic changes appearing during right coronary arteriography. The ST-T wave changes are suggestive of inferior wall ischemia. A shift of the QRS axis inferiorly and to the right is associated with increased voltage of the R wave in lead III. No changes in the initial 0.02 QRS vectors are noted.

Case 2

A 61-year-old man of heavy body build was admitted with a history of chest pain of 3 months duration manifesting itself occasionally at rest without any apparent cause, and always present with exertion. The ECG taken when the patient was free of pain (fig. 3, control) showed a suspicious pattern of old myocardial infarction (confirmed by VCG). During the exercise test and immediately after the test, the patient developed slight S-T-segment elevation in leads II, III, and aVF; S-T depression in leads V3-V6; and a shift of the mean QRS forces inferiorly and to the right. The electrocardiographic changes were remarkably similar to those described in case 1. Also in this case, the S-T-segment alterations and the QRS axis shift decreased gradually, and the ECG returned to the preexercise features after 12 min (fig. 3).
The heart rate was artificially increased in this patient 24 hours after performing the exercise test by using inhalation of amyl nitrate. No conduction disturbances were observed although the heart rate was 10 beats/min superior to the rate induced by the exercise test.

Coronary arteriography revealed subtotal obstruction at the upper third of the right coronary artery and atherosclerosis of the anterior descending branch of the left coronary artery. The circumflex artery was normal.

Case 3

A 45-year-old man of heavy body build was admitted with a 2-month history of chest pain, occasional at rest and always present with exertion. The control ECG (fig. 4) revealed an old myocardial infarction (confirmed by VCG). An exercise test was performed and, as in cases 1 and 2, the inferior QRS axis shift and the S-T-segment elevation in leads II, III, and aV_F were observed to occur, and to disappear gradually. The ECG returned to the preexercise features after 6 min (fig. 4). The exercise-induced changes were remarkably similar to those described for cases 1 and 2. No coronary arteriography was performed in this case.

Case 4

A 54-year-old man of heavy body build was admitted because of typical angina pectoris of 3 weeks duration. A control ECG (fig. 5) showed inverted T wave in leads II, III, and aV_F. During the exercise test and immediately thereafter the patient developed marked S-T-segment depression in leads II, III, aV_F, and V_4–V_6, and inferior QRS axis shift. Such changes disappeared gradually and the ECG returned to the control features after 6 min (fig. 5). The QRS changes
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C.A. 45-year-old man

Figure 4

Case 3. Control. ECG taken when the patient was free of pain: pattern suggestive of old inferior myocardial infarction. Exercise. Tracing taken after exercise test. Note S-T-segment elevation in leads III and aVF; S-T depression in the anterolateral chest leads; shift in direction of the mean QRS forces inferiorly and slightly to the right, from $-10^\circ$ to $+105^\circ$; increase of the QRS interval by 0.02 sec; definite change of direction of the initial QRS forces; S$_1$Q$_{III}$ pattern and leftward displacement of the precordial transition zone. The exercise-induced changes disappear gradually and the ECG returns to the preexercise features after 6 min.

were remarkably similar to those observed in cases 1, 2, and 3.

Coronary arteriography revealed a dominant right coronary artery with subtotal obstruction at the midportion; the circumflex artery arose from the left coronary sinus, while the anterior descending artery was seen to arise from the right coronary sinus. The circumflex artery and the anterior descending artery showed diffuse atherosclerosis.

Discussion

Our four cases can be considered typical examples of the electrocardiographic findings which have been attributed to "pure" LPH in previous publications.$^{1-10}$ In all of them, the exercise-induced changes accounting for LPH disappeared gradually, within 6–12 min.

Common findings in all four patients were: (1) A shift of the main QRS forces inferiorly and slightly to the right, between $+90^\circ$ and $+120^\circ$, which generates a relatively tall R wave in leads II, III, and aVF, and a deep S wave in leads I and aVL. (2) A definite change in the direction of the initial 0.02 QRS vectors, which were shifted superiorly and to the left. (3) The changes described in (1) and (2) are responsible for the occurrence of an S$_1$Q$_{III}$ pattern, which simulates a fictitious clockwise rotation of the heart on the longitudinal axis. (4) A displacement of the precordial transition zone to the left. (5) All these changes occurred with a small widening of the QRS interval $\leq 0.02$ sec.
The QRS changes noted in these patients are similar to the electrocardiographic findings which were postulated to be the result of LPH. However, the possibility of a delay in activation of the posteroinferior wall of the left ventricle due to injury should not be dismissed. Perhaps, an "intrainjury" conduction delay due to hypopolarization of the affected fibers can be excluded by extrapolating information obtained by right coronary injection in patients without significant coronary artery disease. In these instances, inferior ischemia occurs associated with a shift of the QRS axis inferiorly and to the right. However, the predictable variations in the initial 0.02 QRS vectors of LPH are not seen during this procedure.

Actually, in our case 1, during the right coronary artery injection, a shift of the QRS axis was noted without any change in the initial 0.02 QRS vectors (fig. 2, top), which were shifted superiorly and to the left during the exercise test (fig. 1). These findings suggest that extensive posteroinferior focal or parietal conduction delays are different to block in the posterior division of left bundle branch. Hence, the changes in these four cases are more probably due to LPH than to an intrainjury block.

Perhaps the most remarkable contribution of the present group of cases is related to the gradual disappearance of the exercise-induced axis shift that we ascribed to different degrees of incomplete LPH, or to step-by-step decrease in the intrainjury block. However, for

Figure 5
Case 4. Control. ECG taken when the patient was free of pain: inverted T wave in leads II, III, and aVF. Exercise. Tracing taken after exercise test. Note S-T-segment depression in leads II, III, aVF, and V4-V6; shift of the mean QRS forces inferiorly and slightly to the right, from $+60^\circ$ to $+110^\circ$; increase of the QRS interval by 0.02 sec; definite shift of the initial 0.02 QRS vectors superiorly and to the left (in lead III an initial positive force is replaced by a small Q wave); and clockwise rotation ($S_QI^I$ pattern and leftward displacement of the precordial transition zone). The exercise-induced changes disappear gradually and the ECG returns to the preexercise features after 6 min. In leads aVF, V3, and V4, recording is technically imperfect.
the reasons which were discussed above, they possibly represent different grades of LPH. If this is so, it further corroborates that a control tracing showing a QRS axis starting at 0° and then shifting to −60° may already indicate a fairly high degree of LPH. However, under such conditions the diagnosis will only be possible when the axis direction is changing from beat to beat, or from tracing to tracing as in our cases.

It has been reported that LPH may simulate the existence of an inferior infarction because of the occurrence of Q waves in leads II, III, and aVF. This is particularly evident in case 1, but could also be seen in case 4. In cases 2 and 3, however, LPH decreased the size of Q waves already in leads II, III, and aVF, thus tending to mask the signs of an inferior myocardial infarction, as recently reported by others. In cases 2 and 3, there was a gradual reappearance of the abnormal Q waves simultaneously with the reversion of the LPH features.

The control or preexercise tracings showed patterns suggestive of an old myocardial infarction in two cases (cases 2 and 3) and inverted or diphasic T waves in the inferior leads in the other two (cases 1 and 4). The relationship of the LPH pattern to the previous existence of damage in the posteroinferior wall of the left ventricle was thus obvious in every case, as expected. However, the question arises as to whether the sudden and transient occurrence of the LPH pattern was related to the development of a new, transient, and fresh injury, or to the concomitant increase in heart rate because of the exercise test, as may happen in intermittent forms of intraventricular block. This latter possibility was excluded in cases 1 and 2 by artificially increasing the heart rate and being unable to elicit an LPH pattern.

It was stressed that while left anterior hemiblock is relatively common in extensive anterior myocardial infarction, left posterior hemiblock is rare in posteroinferior infarction. The difference has been attributed to the fact that the posterior division of the left bundle branch is much less vulnerable than the anterior division, and to the fact that the posterior division seems to have a dual blood supply (from both the anterior and the posterior descending arteries), while the anterior division depends totally on the anterior descending artery. Accordingly, it seems likely that for the occurrence of pure LPH, even when it is directly related to damage in the posteroinferior wall of the left ventricle, both the right and the left coronary arteries should be involved. Concerning this point, it should be mentioned that when coronary arteriography was performed cases 2 and 4 had both the right and the left coronary arteries severely diseased whereas case 1 had exclusively the right coronary artery diseased. If LPH was really present in this patient with single-vessel disease, then we can conclude that the blood supply of the posterior division of the left bundle branch is principally, if not entirely, derived from the right coronary system, as others have indicated.

Finally, the fact that we were able to obtain four cases of transient LPH during the performance of exercise tests on not too large a series of cases suggests that LPH is perhaps more common than previously thought, particularly when taking into consideration that incomplete forms of LPH may perhaps occur and be overlooked under similar circumstances.

Addendum

After this paper was submitted for publication, case 1 developed permanent electrocardiographic pattern suggesting high degree of LPH. A further case, showing typical LPH features induced by exercise test, has been observed. In this case, the angiographic findings showed a severe single narrowing of the right coronary artery whereas the left coronary artery was normal.

References


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