

Left posterior fascicular block (LPFB) - 2010

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Para poder fazer o diagnóstico de LPFB o hábito asténico deve estar ausente e no presente.

O diagnóstico de LPFB sempre deve ser clinico-eletrocardiografico e obrigatoriamente nao pode ter SVD, ex enfisema, habito asténico longilíneo ou infarto lateral.

Em referencia ao aumento da deflexao intrinsecoide (R peak time = ou > 45ms) no BDPI ocorre nas inferiores aVF e V6 e no na lateral alta aVL.

Lembre-se que o primeiro vetor no LPFB aponta para acima e a esquerda isto é para aVL e só mais tarde para a região inferior que se inscreve com um atraso medio de 20ms.

Por outra parte os sinais de LPFB não necessariamente devem todos estar presentes.

Finalmente fijese em V5 possui S final. A ausencia de S final em V6 pode (e deve) ser por má posicionamento de eletrodo precordial fato extremamente frequente

Envio um artigo interessante em English escrito por Morton Arnsdorf acerca do raro LPFB.

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In the discussion that follows, it is assumed that the reader understands the general concepts of cardiac vectors, asynchronous activation of the ventricles (delayed as in fascicular or bundle branch block, or early as in preexcitation), and the effects that asynchrony has on the duration, morphology and amplitude of the QRS complex.

FASCICLES OF THE LEFT BUNDLE BRANCH j^a The classic hypothesis proposed by Rosenbaum and his coworkers was that the left bundle branch divides into two fascicles of rapidly conducting Purkinje fibers (ie, phase 0 dependent on the rapid inward sodium current) [1]. These fascicles primarily affect the direction of depolarization:

- The left anterior fascicle crosses the left ventricular outflow tract and terminates in the Purkinje system of the anterolateral wall of the left ventricle.
- The left posterior fascicle appears as an extension of the main bundle and fans out extensively posteriorly toward the papillary muscle and inferoposteriorly to the free wall of the left ventricle.

In addition, a third fascicle, called the left median or centroseptal fascicle, is found in nearly 65 percent of people [2,3]. This fascicle runs to the interventricular septum, and can arise from the common left bundle or from the anterior, posterior or both fascicles.

Support for the trifascicular nature of the left bundle comes from the observations in animals and humans that depolarization of the left ventricle begins in three areas corresponding to the terminal portions of the anterior, posterior and septal fascicles [4,5]. In the normal heart, the three fascicles of the left bundle are simultaneously depolarized.

Blood supply j^a The proximal part of the left posterior fascicle is supplied by the artery to the atrioventricular (AV) node and, at times, by septal branches of the left anterior descending (LAD) artery. The distal portion has a dual blood supply from both anterior and posterior septal perforating arteries. The left anterior and median fascicles are supplied either by septal branches of the LAD or by the AV nodal artery.

ELECTROCARDIOGRAM IN LEFT POSTERIOR FASCICULAR BLOCK

i^a Anterior and posterior fascicular blocks mainly affect the direction but not the duration of the QRS complex, because the conduction disturbance primarily involves the early phases of activation. (See "Left anterior fascicular block"). The electrocardiographic criteria for left posterior fascicular block (LPFB, or left posterior hemiblock) are depicted the ECG (show ECG). The changes that are seen reflect alterations in the different phases of activation.

- Early activation i^a Early activation by the normally conducting anterior and septal fascicles causes the initial vector to be directed to the left, anteriorly, and superiorly producing initial small r waves in leads I, V1, and V6.

- Mid-temporal and terminal activation i^a The mid-temporal and terminal vectors in LPFB are directed to the right, posteriorly, and inferiorly due to delayed depolarization of the areas normally activated by the left posterior fascicle. This leads to the characteristic rightward axis of $+90^\circ$ to $+180^\circ$ [6]. As a result, there is a qR morphology in leads II, III, aVF and Y and an rS morphology in leads I and aVL.

The QRS duration usually does not exceed 0.10 sec (100 msec), although the WHO/ISFC Task Force allows up to 0.12 sec or 0.02 sec above the previous baseline [6].

Diagnostic problems i^a There are a number of settings which may produce ECG findings similar to LPFB.

- The above criteria apply only in the absence of other causes for a rightward axis, such as right ventricular hypertrophy due to valvular heart disease or lung disease with cor pulmonale.

- A high lateral or anterolateral myocardial infarction (MI) can mimic LPFB. With an infarct, however, the initial r wave in leads I and aVL is absent and only a Q wave is seen.

- The presence of right bundle branch block (RBBB) might suggest right axis deviation because of the deep terminal S wave in

leads I, aVL and V5, and V6. However, these S waves reflect delayed right ventricular activation, not left ventricular forces. An additional potential source of confusion is that RBBB can occur in association with LPFB.

- The small q waves in the inferior leads in LPFB may cause confusion with an inferior wall MI.

CLINICAL CONSIDERATIONS

ⁱ^a The left posterior fascicle branch is the first branch of the left bundle and is large in its initial course. It then fans extensively throughout the posterior and inferior left ventricle. The left posterior fascicle is exposed to lower pressures and less turbulence than the left anterior fascicle; it also has a dual blood supply. These characteristics probably explain why isolated LPFB is a rare finding.

Isolated LPFB can, however, be seen in the setting of extensive arteriosclerotic cardiovascular disease, as an association with inferior MI and extensive coronary disease has been suggested [7]. LPFB can also occur with cardiomyopathies, including those which result from hypertension and Chagas disease, myocarditis, hyperkalemia, acute cor pulmonale, and chronic degenerative and fibrotic processes of the conducting system.

TREATMENT

ⁱ^a There are no symptoms induced by an isolated block in one of the left bundle fascicles. Therapy should be considered only in patients with persistent bifascicular or trifascicular block. (See "Course and treatment of chronic bifascicular and trifascicular block").

1. Rosenbaum, M, Elizari, MV, Lazzari, JO. The Hemiblocks. Tampa Tracings, Tampa, 1970.
2. Demoulin, JC, Kulbertus, HE. Histopathological examination of the concept of left hemiblock. Br Heart J 1972; 34:807.
3. Uhley, HN. Some controversy regarding the peripheral distribution of the conduction system. Am J Cardiol 1972; 30:919.
4. Myerburg, RJ, Nilsson, K, Gelband, H. Physiology of canine intraventricular conduction and endocardial excitation. Circ Res 1972; 30:217.
5. Durrer, D, Van Dam, RT, Freud, GE, et al. Total excitation of the isolated human heart. Circulation 1970; 41:899.

6. Willems, JL, Demedina, EO, Bernard, R, et al. World Health Organization/International Society and Federation of Cardiology Task Force. Criteria for intraventricular conduction disturbances. *J Am Coll Cardiol* 1985; 5:1261.
7. Godat, FJ, Gertsch, M. Isolated left posterior fascicular block: A reliable marker for inferior myocardial infarction and associated severe coronary artery disease. *Clin Cardiol* 1993; 16:220.