Phase 4 Left Septal Fascicular Block

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Case Report

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Abbreviation list

LSFB: Left septal fascicular block
MI: Myocardial infarction
LAF: Left anterior fascicular block

Introduction

The existence of a trifascicular left intraventricular system remains under debate. A recent consensus showed some discrepancies and, despite agreeing on the possible existence of an anatomical left septal fascicle, the electrocardiographic characteristics of its block were not universally accepted\textsuperscript{1,2}. One of the criteria requested to confirm the existence of a left septal fascicular block (LSFB) was its intermittent nature\textsuperscript{1}. So far, few cases of ischemia-induced LSFB have been published, but no rate-related LSFB has been described to date\textsuperscript{3}.

The aim of this presentation is to describe a bradycardia-dependent LSFB in an otherwise asymptomatic patient.

Case presentation

An 89-year-old patient with prior history of hypertension, dyslipemia and myocardial infarction (MI) attended the clinic for regular checkup. He was treated with atenolol 25 mg/day, rosuvastatin 10 mg/day and aspirin. A12-lead ECG was obtained showing sinus rhythm (cycle length: 960 ms), PR interval at 370 ms, extreme left axis deviation of the QRS at - 65° with rS pattern in leads II, III and aVF and SIII > SII, qR pattern in leads I and aVL indicating left anterior fascicular block (LAFB). Incomplete right bundle branch block (QRS duration 110 ms) and old transmural antero-apical MI can be also seen (Fig. 1).

A second 12-lead ECG was performed due to perception of irregular pulse depicting second degree AV block with alternating 4:3 and 2:1 conduction (Fig. 2). After the pause generated by the non-conducted P-waves (1,720 ms), the following conducted
beats with a fixed shorter PR interval at 270 ms depicted a different QRS morphology suggesting LSFB (Figure 2, beats 4th, 7th and 8th). Changes respect to previously conducted beats included: the initial q-waves in leads I and aVL and the initial r-waves in leads II, III and aVF have disappeared; in the right precordial leads, a sudden increase of R-wave voltage (Prominent Anterior Forces) and a discrete increment in the QRS duration (10 ms) can be seen. The interpretation of this phenomenon was bradycardia-dependent or phase 4 LSFB. Due to further 2:1 persistent AV block, a dual chamber permanent pacemaker was implanted.

Discussion

The concept of a bifascicular left hissian system has prevailed for decades4-5. The idea of a third septal fascicle, despite its anatomical demonstration, the detailed description of the electrocardiographic characteristic of the LSFB, and previously published consensus, remains under debate1,2,6. The ones that do not completely accept its existence, do not dispute that most of the pictures that illustrate classic text books on hemiblocks, showed also a group of fibers directing to the septum7. However, there are multiple anatomic variations of the left septal fascicle, that may depart from the other left fascicles (mainly the posterior division) and not from the main left bundle branch and even may be absent. Proof of transient LSFB, as part of the requisites to recognize a new ECG dromotropic disturbance, is considered mandatory. There were few cases of transient ischemic LSFB in the literature (associated with proximal obstruction of the left anterior descending coronary artery before the first septal perforator branch or ischemia triggered during an exercise stress test3,8), however; no rate-dependent LSFB was yet found, leaving a gap in knowledge and raising suspicion about the LSFB existence. The only mention to a rate-dependent septal block in the literature belongs to a manuscript from Gambetta et al. in 2 cases of septal myocardial infarction9. Rate-dependent blocks are the best model to study any new conduction disturbance in the conducting tissue, as they are free of possible “contaminants” associated with transient injuries to the surrounding tissue, as it happens in ischemia.

The electro-vectorcardiographic expression of LAFB and left posterior fascicular block (LPFB) are manifested mainly in the frontal plane by extreme deviation of the QRS electrical axis to the left around -60° (LAFB) and to the right around +120° (LPFB)4,7.
LSFB behaves differently, as its most conspicuous electro-vectorcardiographic manifestation occurs in the horizontal plane (precordial) by an anterior and to the left displacement of the QRS electrical forces (most of the QRS loop area is located in the anterior left quadrant)\(^2\). This is reflected in the right and middle precordial leads of the surface ECG as an increment on the R-wave voltage (prominent anterior forces) from leads V1 to V4 in an in crescendo pattern\(^2\). An alternative explanation for the prominent anterior forces could be due to a higher degree of right bundle branch block. In our case, however, the QRS complex widened only 10 ms and the lack of broad S-waves in the left precordial leads and lack of terminal slurred R-wave in lead aVR turns this possibility highly unlikely. Most of all changes in the initial QRS forces (lack of the initial q-wave in lead I and left precordial leads), reflects the lack of activation of the middle third of the left septal surface from left to right, dependent on the left septal fascicle and are unrelated to a right bundle branch block. Confirmation with intracardiac recordings would be ideal but was not available in this case.

As occurs with LPFB which diagnosis is electrocardiographic and clinical (to rule out right ventricular enlargement or vertical position of the heart), LSFB should only be suspected if other causes displacing the QRS forces in the horizontal plane have been ruled out\(^10\). The differential diagnosis should include lateral myocardial infarction, left accessory pathways, hypertrophic cardiomyopathy, muscular dystrophy, endomyocardial fibrosis and fusion beats (escapes) originated either in the fascicles or the unspecific myocardium. Regarding a differential diagnosis with fascicular escapes, the phenomenon reported here was repetitive over a full 10 min recording during 2:1 AV block, with mild changes in the PP intervals and PR intervals, and slight variations in the RR intervals without discernible changes in QRS morphology, as expected in a case of variable fusions between an escape rhythm and conducted sinus beats, providing strong evidence against that possibility. The clue to the electro-vectorcardiographic diagnosis of LSFB is transient prominent anterior forces which helps ruling out all other causes\(^2\).

In the case presented here, prominent anterior forces are depicted in an intermittent basis in an otherwise asymptomatic individual, associated with preceding pauses triggered by second degree AV block. Upon recovery of conduction in the AV node and resolution of pauses, prominent anterior forces (and LSFB) disappears. This indicates a phase 4 or bradycardia-dependent mechanism. To the best of our knowledge, this is the
first report of a rate-dependent LSFB, confirming prior observations of its existence and contributing to the better understanding of the physiopathology of the left hisian system.

Conclusion

Phase 4 LSFB is demonstrated in this case of intermittent second degree AV block. This observation contributes to the growing evidence of a trifascicular left intraventricular electrical system.

References


Legends to the figures

**Figure 1.** Surface 12-lead ECG during routine check-up.

**Figure 2.** Surface 12-lead ECG depicting second degree AV block and Phase 4 or bradycardia-dependent LSFB.