

In search of left septal fascicular block

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Background In most humans, the left bundle branch divides into 3 fascicles. Electrocardiographic changes resulting from conduction abnormalities of the left anterior and left posterior fascicles are well described and commonly diagnosed. Existence of conduction defects of the left septal fascicle is controversial, without generally accepted criteria for diagnosis.

Methods Approximately 26,000 electrocardiograms were reviewed to find examples consistent with left septal fascicle block according to criteria derived from review of the literature.

Results Electrocardiograms meeting proposed criteria were found in about 0.5% of tracings. Illustrative examples are shown with discussion of reasons for their selection.

Conclusions Left septal fascicle block seems to exist, is polymorphic, and may explain some previously inadequately understood electrocardiographic abnormalities. (*Am Heart J* 2002;144:948-56.)

Conduction defects involving the anterior and posterior fascicles of the left bundle branch, defined as and called "the hemiblocks" more than 30 years ago by Rosenbaum et al,¹ have become accepted aspects of clinical electrocardiography.² Anatomical studies, however, show that as it divides, the left bundle branch gives off fibers to the left endocardial surface of the interventricular septum (IVS), as well as to its better recognized anterior and posterior divisions.³⁻⁷ In some cases, these septal fibers are distributed broadly over the left septal surface like an interconnecting network.^{3,4,6} Histologic reconstructions of the left conduction system by Demoulin et al⁷ in 1972, however, showed that septal fibers can be identified in most cases as a distinct third division of the left bundle branch, as illustrated in Figure 1.

In 1970, Durrer et al⁸ found that earliest activation of the human left ventricle occurred simultaneously in 3 endocardial regions corresponding roughly to myocardial insertions of the 3 fascicles of the left bundle branch.^{7,8} Right ventricular activation began about 5 ms later. Activation waves resulting from anterior and posterior areas of initial left ventricular activation progress in directions approximately opposite to each other, and the potential fields created tend to cancel each other.⁵ This leaves activation of the IVS, starting from its left septal surface, to dominate the QRS in its first 10 ms, despite a small, opposite, slightly delayed potential from activation of the right septal surface

through the right bundle branch.^{2,8} Net initial QRS forces are directed rightward and anteriorly, causing small q waves in leads I, V₅-V₆. These have been termed "septal q waves."⁹

There are reasons why accepted criteria do not exist for block of the middle or septal division of the left bundle branch, or left septal fascicular block (LSFB). The anatomy of this fascicle, where it exists as a distinct entity, is more variable than that of the other 2 left fascicles.⁷ These septal fibers usually have many interconnections^{4,7} and the shortest refractory period of conduction fibers originating from this bundle branch.¹⁰ The amount of septum activated by way of the right bundle branch varies among individuals.⁸ Septal orientation also varies, and as the direction of initial QRS forces is related to orientation of the IVS,¹¹ this may alter the effect of LSFB on the electrocardiogram (ECG). LSFB may present with multiple morphologies and is frequently combined with other conduction abnormalities that disguise it.^{5,12}

Different criteria for LSFB have been proposed on the basis of anecdotal reports,^{6,12-20} results of experimental incision of septal fibers in the dog,^{21,22} electrical-anatomic models,⁵ and computer-based projections of QRS morphology.^{23,24}

Hypothetical models of LSFB

Activation of the IVS is normally accomplished by a double envelopment starting from both left and right septal surfaces, as contrasted to the endocardial to epicardial direction of activation of the free walls of the 2 ventricles.^{8,25} Activation of the right septal surface is initiated through the right bundle branch no more than 5 ms after first activation of the left septal surface.^{8,25} The activation wave traverses the septum in either direction in about 40 ms; septal activation is

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nearly completed within 35 ms of the beginning of ventricular depolarization,^{8,25} except for the basal septum, which has a relative dearth of Purkinje fibers.^{2,6}

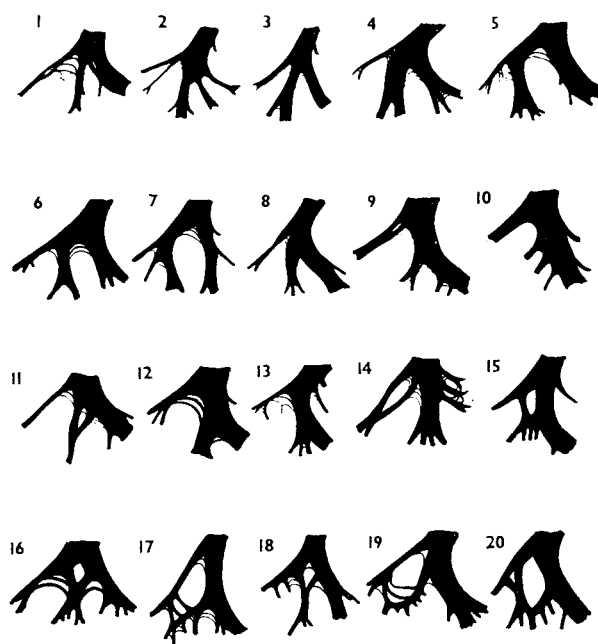
When conduction in the right bundle branch is normal, mild delay in activation of the left septal surface caused by slow conduction in septal fibers of the left bundle branch results in only slight delay in initiation of septal activation and little prolongation of the QRS. The contribution of left-to-right septal activation to orientation of initial QRS forces is attenuated or abolished, with diminution or disappearance of septal q waves.^{5,18,23,26} In contrast to the situations in left anterior and left posterior fascicular blocks (LAFB and LPFB), little or no effect occurs on direction or magnitude of QRS forces during the middle and late parts of ventricular activation.⁵

Greater delay in left septal activation could additionally result in reversal of average direction of septal activation to right-to-left and front-to-back, similar to what is usually seen in incomplete left bundle branch block (ILBBB).²⁶ Unless counterbalanced by simultaneous anteriorly directed forces of right ventricular free wall activation, this could produce posterior orientation of initial QRS forces, with q waves or even QS deflections in right precordial leads.

If left septal activation is significantly delayed and right septal activation is also limited in extent or delayed, septal activation has to be initiated from depolarization waves reaching it from myocardium that has been previously activated through the anterior and posterior fascicles of the left bundle branch. Considerable portions of the septum will be activated late from left-to-right and back-to-front. In this case, in addition to loss of septal q waves, anterior deviation of mid-to-late QRS forces might result, analogous to the left superior or right inferior deviation of mid-late QRS forces seen respectively in LAFB or LPFB. Such prominent anterior forces would be appreciated only in precordial leads. When otherwise unexplained by right ventricular hypertrophy, posterior infarction, right bundle branch block (RBBB), or ventricular pre-excitation, they have been claimed to be the result of just such an LSFB.^{6,12-16,19,20} That some of these cases were associated with RBBB when the conduction defect worsened is consistent with this hypothetical model.^{15,16}

With LSFB, mean frontal plane QRS axis and ventricular activation times in aVL, aVF, and V₅-V₆ should remain normal, as activation of the left ventricular free wall and apex by way of the left anterior and posterior fascicles is undisturbed. Absence of slurring or delay in R wave upstroke in leads overlying the left ventricle helps differentiate this conduction defect from various degrees of ILBBB.²⁶

Figure 1



Sketches are shown of the left ventricular conduction system from 20 normal human hearts. These represent reconstructions of the anatomy from serial histologic sections of carefully oriented blocks of left septal myocardium. Orientation is as if viewing the left septal surface from the left: anterior is to the viewer's left and superior at the top. In most examples, a middle or septal fascicle can be seen to arise from the central part of the main left bundle (sketches 1-4), from the anterior fascicle (sketches 5-7), from the posterior fascicle (sketches 8, 9, 11-14), or from fibers originating from both of the latter fascicles (sketches 15-20). (From: Demoulin JC, Kulbertus HE. Histopathological examination of concept of left hemiblock. *Br Heart J* 1972;34:807-14. Reproduced with permission from the BMJ Publishing Group.)

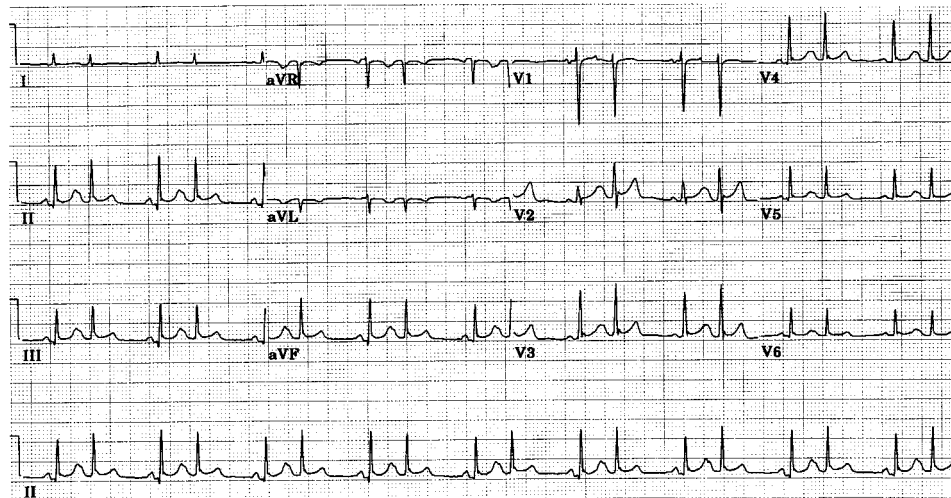
Methods

Approximately 26,000 consecutive ECGs processed in the ECG laboratory of the UCLA Medical Center were reviewed to identify those meeting criteria for LSFB derived from the literature (Table D). Where appropriate, hospital records were reviewed to find old tracings that might antedate changes in intraventricular conduction. ECGs shown below were recorded at a paper speed of 25 mm/s, calibrated at 1 mv = 1 cm, using paper with fine and coarse grid lines 1 mm and 5 mm apart, respectively. This protocol was approved by the UCLA Institutional Review Board.

Results

Some examples of probable and possible LSFB, with discussion

Tracings suggestive of LSFB were encountered in approximately 0.5% of ECGs reviewed.

Figure 2

Aberrant ventricular conduction after premature atrial beats causes loss of septal q waves.

Table I. Proposed criteria for uncomplicated left septal fascicular block

Loss of septal q waves (if present initially)*

Initial QRS vector directed to the left

Absence of other causes of abnormal septal activation*

e.g., Septal infarction, complete or incomplete LBBB, ventricular pre-excitation

QRS duration ≤ 110 ms; increased no more than 10 ms over preblock complex*

Ventricular activation time normal or unchanged in V_5 - V_6 , aVL, aVF*

No slurring or notching on R upstroke in I, V_5 - V_6 *

Little change in direction of mean frontal plane QRS axis*

Q wave in V_1 and/or V_2 †

Evidence of right-to-left, anterior-to-posterior direction of septal activation; possibly masked by presence of initial r due to right ventricular free wall activation

Evidence of delayed left-to-right and back-to-front septal activation†

a) Little or no change in 40 ms QRS vector, or

b) prominent anterior forces not explained by RVH or posterior infarction: $R/S > 1$ in V_1 and/or V_2 (maximal or mean QRS vector anterior to $+0^\circ$ (in horizontal plane); prolonged time to intrinsicoid deflection > 35 ms may be seen in V_1 - V_2)

Secondary repolarization changes†

*Items needed for diagnosis.

†Items not necessary for diagnosis.

Loss of septal q waves as the only change in LSFB. Absence of septal q waves in ECGs that are otherwise normal is the simplest way LSFB might present. This ECG pattern is not usually the result of acquired cardiac disease and probably represents a variant of normal found in about 7% of normal ECGs,²⁷ but such a pattern can result from aberration in intraventricular conduction. Figure 2 shows an example from a 20-year-old woman with pneumonia but no prior cardiac disease. Premature atrial beats were conducted with slight aberrancy probably resulting from LSFB. Transient disappearance of q waves from inferolateral leads occurred without change in QRS duration or frontal

plane mean QRS axis. There was increase in amplitude of anterior QRS forces at 30 ms. Aberrant beats were otherwise within normal limits, without secondary ST or T changes. Shortly after this ECG, atrial fibrillation developed with a ventricular rate of 195 per minute, during which all beats showed the same type of aberrant conduction. If the latter were to become permanent, without other abnormalities and without prior tracings, the resulting ECG would be indistinguishable from a normal variant as described above.

Examples of this type of transient LSFB were seen most commonly in subjects with atrial fibrillation. Short R-R intervals would sometimes result in disap-

pearance of septal q waves, as shown in Figure 3, taken on a 69-year-old woman without cardiac history.

Rate-related, transient loss of septal q waves in every beat was encountered in some cases of supraventricular tachycardia, and in atrial flutter with 2:1 atrioventricular block. One case was found with disappearance of septal q waves in every other beat during a regular supraventricular tachycardia, possibly reflecting a 2:1 LSFB.

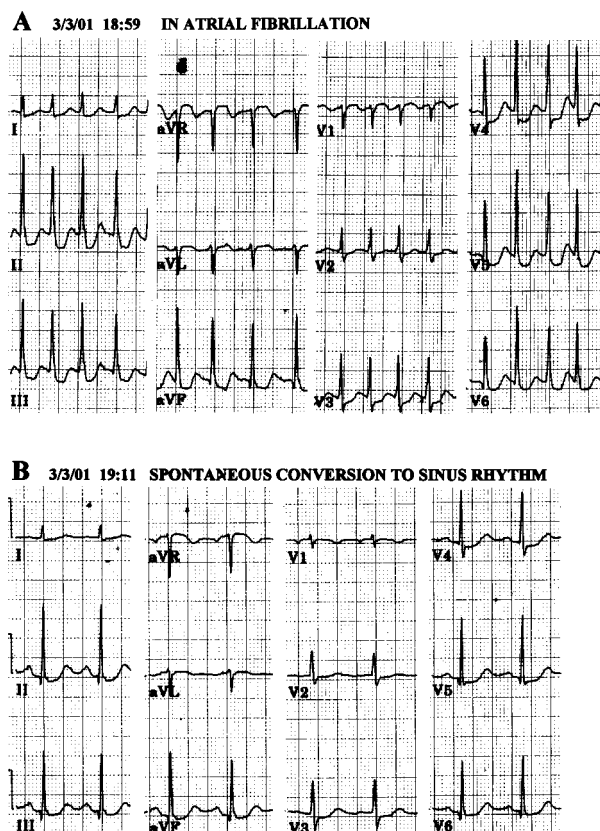
Posterior deviation of initial QRS forces in addition to loss of septal q waves. Occasionally, changes in initial QRS direction in aberrantly conducted beats varied with degree of prematurity of these beats. Figure 4 is an ECG from a 75-year-old man with coronary and peripheral vascular disease. Echocardiography showed inferior and posterobasal left ventricular hypokinesis. Some aberrant beats lacked septal q waves. In others, septal q waves were attenuated. With the most premature ectopic atrial beat, which occurred while right precordial leads were being recorded, a q wave appeared in leads V₁-V₃, suggesting posterior shift of initial QRS forces, which was probably the result of a greater degree of LSFB.

The ECG in Figure 5 is from a 90-year-old hypertensive woman with paroxysmal atrial fibrillation. Left atrial enlargement was the only echocardiographic abnormality. Atrial premature beats caused 2 distinct forms of aberrant QRS complexes. One type had changes indicating LAFB combined with RBBB, without discernable alteration of direction of initial QRS forces. In the second type, with slightly greater ectopic beat prematurity, there were loss of q waves from aVL, V₅, and V₆, loss of r waves in V₁-V₃, but no major change in frontal plane mean QRS axis. Increase in depth of S waves in V₁-V₄ and in height of R waves in V₅-V₆ occurred. Neither ventricular activation time nor QRS duration changed, nor was there slurring of R wave upstroke in V₅ or V₆, making ILBBB unlikely. This is probably another example of LSFB. The resulting QRS configuration mimics septal infarction and augments the voltage signs of left ventricular hypertrophy (LVH).

Diminution in amplitude or complete loss of septal q waves in the systolic overload pattern of LVH may be due to LSFB¹⁸ rather than to augmented left ventricular free wall forces that are not counterbalanced by left-to-right septal forces. This type of aberrancy has been reported with programmed premature electrical stimulation of the right atrium in subjects with known LVH¹⁸ and after spontaneous premature atrial beats.¹⁷ This manifestation of LSFB may be a cause of QS complexes in V₁-V₂ in some cases of severe LVH.^{2,18,28} It could also contribute to the low specificity of QS complexes in V₁-V₂ for diagnosing septal infarction.²⁸⁻³⁰

Chronic LSFB. The series of ECGs shown in Figure 6 probably also demonstrate this type of LSFB compli-

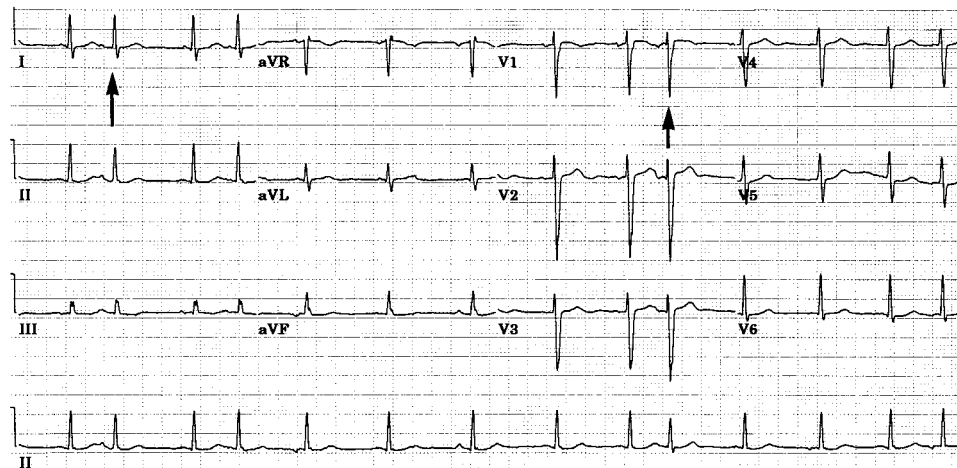
Figure 3



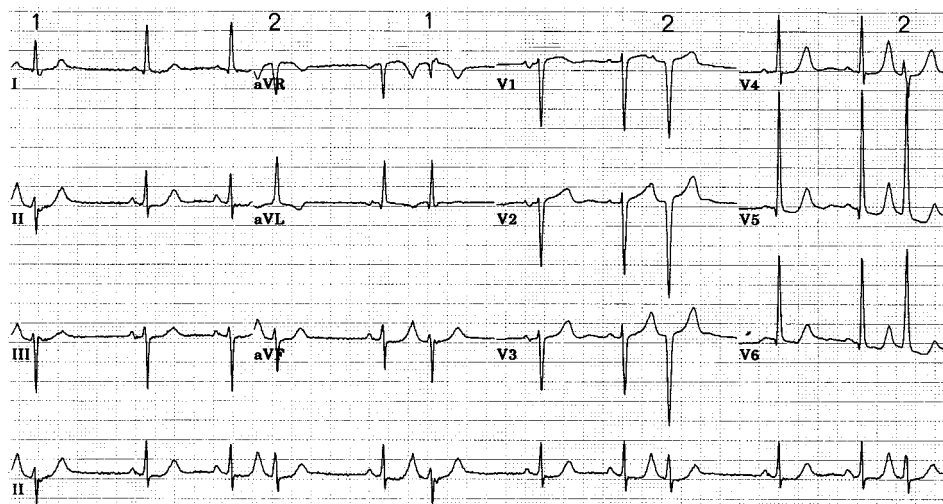
Transient, rate-related LSFB. **A**, During atrial fibrillation, variable aberrancy of intraventricular conduction related to preceding R-R interval produces loss of septal q waves from inferolateral leads and decrease in r in V₁ without change in QRS axis or duration. **B**, After conversion to sinus rhythm 12 minutes later, prominent septal q waves are present in inferolateral leads.

cating aortic balloon valvuloplasty in a 17-year-old boy with congenital aortic stenosis. Two preoperative tracings showed LVH with tiny q waves in leads I and V₆, and initial r waves in V₁-V₂. After valvuloplasty, he lost his r waves in V₁-V₂, and q waves were no longer seen in I and V₆. QRS duration and ventricular activation time in V₆ were not increased. Postoperative troponin I levels peaked at 0.54 ng/mL (normal ≤ 0.1), with normal total creatine kinase and creatine kinase-MB values, so major septal infarction was unlikely. No ST-T changes evolved. The fourth tracing in the series, taken 3 weeks later, showed return of initial r wave in aVL and V₂, but QS remained in V₁, without return of clear septal q waves in V₆.

No example was found of prominent anterior QRS forces, described by others as a manifestation of LSFB,^{6,12-16,19,20} which could be clearly differentiated

Figure 4

Ectopic atrial beats of differing prematurity cause varying aberrancy with slight changes in initial and middle parts of the QRS without major change in mean QRS axis. At the *first vertical arrow*, septal q waves disappear for a beat. At the *second vertical arrow*, at shorter preceding R-R interval, q waves appear in V₁-V₃, and S wave in V₃ is deeper, indicating possibly a greater degree of LSFB. QRS durations: sinus beat 100 ms; beat at *second arrow*, 110 ms.

Figure 5

Two types of aberrant intraventricular conduction are caused by premature atrial beats. In beats labeled *1*, there is no change in direction of initial QRS forces, but LAFB combined with RBBB develops. In beats labeled *2*, initial forces shift leftward and posteriorly without change in mean frontal plane QRS axis or QRS duration. Loss of septal q waves and of initial r in V₁-V₃ is probably caused by transient LSFB.

from a normal variant,³¹ or that did not have a readily apparent clinical explanation other than LSFB.

Combination of LSFB with other left fascicular blocks. The presence of 4 fascicles rather than the traditional 3 adds complexity to the possible combinations of fascicular blocks.⁵

Figure 7 shows sequential ECGs taken during a bout of spontaneous angina. This 60-year-old woman had mixed angina, and 50% diameter proximal anterior descending and 80% ostial right coronary arterial stenoses. As angina waxed, her ECG showed LAFB without q waves in lead I, with QS deflections in V₁-V₂, and

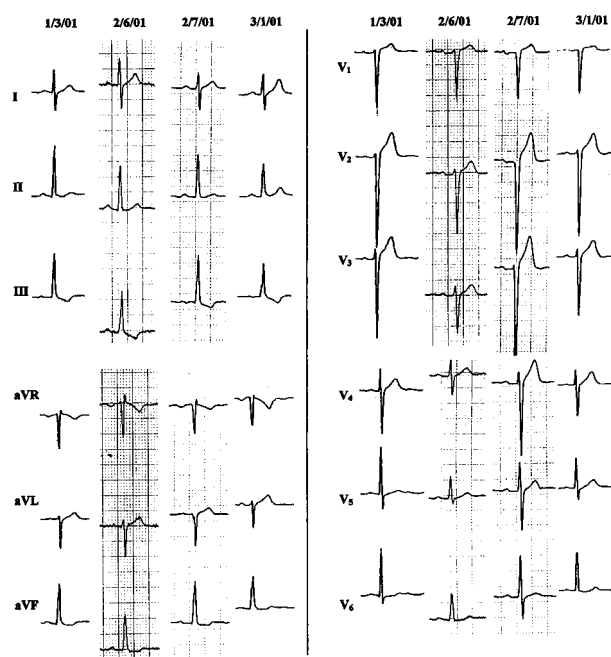
ischemic ST-segment depression in V_6 . After recovery from the attack, a q wave in lead I and initial r waves in V_1 - V_2 appeared, and ST depression disappeared, leaving her ECG exactly as it had been on contemporary but prior tracings.

The initial ECG of this series looks like LAFB complicated by a septal infarction with lateral subendocardial ischemia. The transient nature of the QS in right precordial leads and loss of q in lead I could be caused by transient LSFB, a conclusion suggested by others describing similar cases.³² Other possible explanations exist for these sequential QRS changes; however, if they were caused by transient LSFB, this case would illustrate that superimposed on chronic LAFB, LSFB can be associated with leftward and posterior rotation of initial QRS forces, as would be predicted from the hypothetical model described above. This suggests that a leftward orientation of initial QRS forces in LAFB, deemed inconsistent with a diagnosis of LAFB by Rosenbaum et al¹ but actually seen in many cases of this conduction abnormality,³³ might be caused by LAFB combined with involvement of left septal fibers as suggested by others,^{23,33} particularly when q waves are present in right precordial leads.³⁴ It is easily imagined how a lesion in the left anterior fascicle could produce such a conduction defect if the bulk of septal fibers of the left bundle branch originated distally as a branch of the left anterior fascicle, as seen in some examples in Figure 1.

No examples were found that could be called LPFB combined with LSFB, although such must exist. Part of the problem is that septal q waves in left-facing leads commonly disappear when LPFB develops because of superior and usually leftward shift of initial QRS forces.^{1,2,5} This means the addition of LSFB to existing LPFB might not cause a recognizable change in QRS configuration.

LSFB combined with RBBB with or without other left fascicular blocks. The presence of RBBB adds special considerations to the detection of coexisting LSFB. RBBB alone may cause slight decrease in amplitude of initial r waves in V_1 - V_2 but does not change the direction of initial QRS forces; hence, septal q waves are preserved when RBBB occurs.^{2,26} In the special case of proximal RBBB combined with LSFB, both right ventricular and interventricular septal activation are dependent on delayed left-to-right and back-to-front septal activation by depolarization waves from myocardium activated originally through the left anterior and posterior fascicles. Direction of initial QRS forces will depend on the orientation of net potentials generated by the start of left ventricular activation by the intact left fascicles. This might be variable but will not be modified by early forces from right ventricular free wall activation. It probably would be directed leftward.⁵ Thus, septal q waves would likely be absent

Figure 6



Loss of initial r in V_1 - V_2 and of septal q waves resulted from balloon aortic valvuloplasty on 2/6/01 in a 17-year-old boy with severe aortic stenosis and left ventricular hypertrophy. Limb leads of 4 sequential tracings are on the left half of the figure, and precordial leads are on the right. QRS durations: 90 ms on 2/6/01; 85 ms on 2/7/01. See text for details.

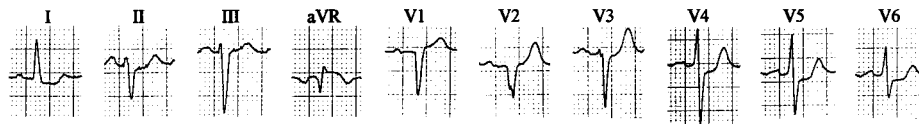
from left-facing leads. The presence or absence of initial r waves in right precordial leads would be hard to predict, however. Their absence in RBBB would add weight to a presumptive diagnosis of coexistent LSFB, but their presence would not necessarily rule it out.³⁵

The direction of forces in the middle of the QRS in this setting would be deviated anteriorly by delayed and relatively unopposed left-to-right and back-to-front activation of the IVS. This would be hard to separate from the anterior and rightward terminal QRS forces because of delayed activation of the right ventricular free wall. Hence, RBBB combined with LSFB might look just like RBBB except for atypical alterations in direction of initial QRS forces.

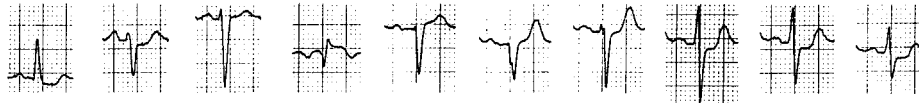
Figure 8 shows ECGs from a 96-year-old woman with hypertrophic cardiomyopathy and moderate aortic stenosis. Left ventricular wall motion and ejection fraction were normal. She never had hypertension or a clinical myocardial infarction. The 2 tracings shown are examples of the numerous ECGs done at 2 periods of her life. The first, dated in 1995, is typical of her ECGs before 1998. It showed typical RBBB, left atrial

Figure 7

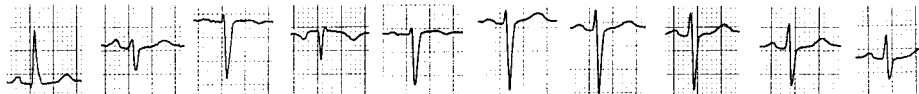
14:10 DURING 5+/10+ SPONTANEOUS CHEST PAIN



14:11 NOW CHEST PAIN 9+/10+



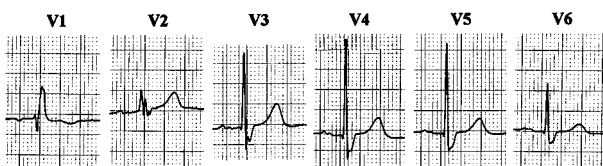
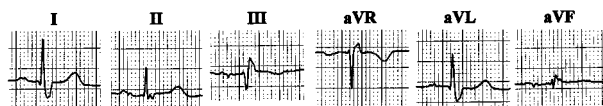
14:22 CHEST PAIN COMPLETELY GONE



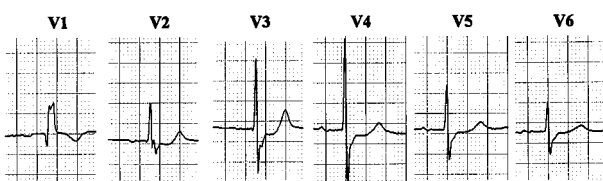
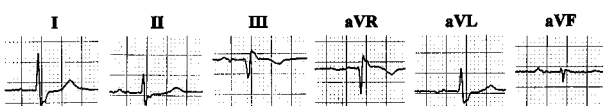
Sequential ECGs during spontaneous angina in a patient with chronic LAFB. Electrode positions were unchanged during the 12 minutes covered by these tracings. QRS durations: 105 ms in the first 2 tracings; 95 ms in the last ECG. See text for details.

Figure 8

9/6/95



8/12/99



Tracings before (**top**) and after (**bottom**) unexplained loss of septal q from I and V₆ and of initial r from V₁-V₂ in a woman with hypertrophic cardiomyopathy and chronic RBBB. QRS durations are 135 ms in both tracings. See text for details.

abnormality, and high R wave voltage in V₄-V₅ consistent with LVH. Initial QRS forces were directed to the right, anteriorly and slightly superiorly, resulting in clear septal q waves in left-facing leads, and initial r waves in V₁ and V₂. All ECGs after 1998 were like the second one shown, dated in 1999. Initial QRS forces were directed to the left, slightly posteriorly and superiorly. Septal q waves had disappeared from left-facing leads, and in right precordial leads initial r waves had been replaced by q waves. There were no significant changes in her echocardiograms between these 2 tracings. The alteration in direction of initial QRS forces responsible for these ECG changes is likely the result of the superimposition of LSFB on the RBBB.⁵

Discussion

Abnormalities of intraventricular conduction consistent with LSFB are more common than previously thought. As mid-late QRS forces may be minimally affected by LSFB, its diagnosis requires particular attention to changes in direction of the initial part of the QRS. Because mild degrees of LSFB may mimic a normal variation,²⁷ its recognition often requires comparison of ECGs taken before and after its development. That the examples illustrated above represent LSFB is predicated on the accuracy of hypothetical models of altered ventricular activation described in the introduction and of the criteria in Table I. Proof that this condition is met will require additional human investigation, including correlation of surface ECG patterns with electrophysiological studies of activation of the IVS.

The various electrocardiographic presentations of LSFB can tie together an assortment of otherwise inadequately explained phenomena: (1) loss of septal q waves by leftward shift of initial QRS forces transiently following short R-R intervals in some cases, permanently in others in the absence of septal infarction or ILBBB; (2) leftward direction of initial QRS forces in some cases of LAFB; (3) loss of initial r waves in right precordial leads accompanied by loss of septal q waves in severe LVH and in some cases of LAFB, or RBBB in the absence of septal infarction or ILBBB; (4) some cases of transient right precordial Q waves during myocardial ischemia in the absence of infarction; (5) atypical cases of ILBBB in which left ventricular activation time remains normal and the sense of initial septal activation may not be strictly right to left.³⁶

This study has not addressed important questions about the potential clinical significance of LSFB. Does alteration of the activation pattern of the IVS in LSFB produce recognizable changes in its pattern of contraction? A study of this might require analysis of septal motion more sophisticated than is used in clinical echocardiography or other imaging methods.³⁷ Are there prognostic implications to LSFB? As a transient aberration of conduction related to a short R-R interval, it probably has little clinical significance. If chronic, it implies the presence of disease involving at least a portion of the left bundle branch system. However, further study is needed to show whether LSFB is a harbinger of more advanced disease of the conduction system or myocardium, whether there are histopathologic correlations to its presence, and whether it carries prognostic significance similar to that of LAFB or LPFB.²

References

- Rosenbaum MB, Elizari MV, Lazzari JO. The hemiblocks: new concepts of intraventricular conduction based on human anatomical, physiological and clinical studies. Oldsmar: Tampa Tracings; 1971. p. 94.
- Surawicz B, Knilans TK. Chou's electrocardiography in clinical practice. 5th ed. Philadelphia: WB Saunders Co; 2001. p. 11, 51, 106-13.
- Tawara S. Das reizleitungssystem des säugetierherzens. Jena: Fischer; 1906. p. 35, 201.
- Massing GK, James TN. Anatomical configuration of the His bundle and bundle branches in the human heart. *Circulation* 1976;53:609-21.
- Uhley HN. The quadrifascicular nature of the peripheral conduction system. In: Dreifus L, Likoff W, editors. *Cardiac arrhythmias*. New York: Grune & Stratton; 1973. p. 339-48.
- Nakaya Y, Hiraga T. Reassessment of the subdivision block of the left bundle branch. *Jpn Circulation J* 1981;45:503-16.
- Demoulin JC, Kulbertus HE. Histopathological examination of concept of left hemiblock. *Br Heart J* 1972;34:807-14.
- Durrer D, van Dam R Th, Freud GE, et al. Total excitation of the isolated human heart. *Circulation* 1970;41:899-912.
- Burch GE. An electrocardiographic syndrome characterized by absence of Q in leads I, V₅ and V₆. *Am Heart J* 1956;51:487-88.
- Iwamura N, Kodama I, Shimizu T, et al. Functional properties of the left septal Purkinje network in premature activation of the ventricular conduction system. *Am Heart J* 1978;95:60-9.
- Kawai N, Sotobata I, Noda S, et al. Correlation between the direction of the interventricular septum estimated with transmission computed tomography and the initial QRS vectors. *J Electrocardiol* 1984;17:401-8.
- de Padua F, dos Reis D, Lopes VM, et al. Left median hemiblock—a chimera? *Adv Cardiol* 1978;21:242-8.
- Hoffman I, Mehta J, Hilsenrath J, et al. Anterior conduction delay: a possible cause for prominent anterior forces. *J Electrocardiol* 1976;9:15-21.
- Kulbertus HE, de Laval-Rutten F, Casters P. Vectorcardiographic study of aberrant conduction: anterior displacement of QRS: another form of intraventricular block. *Br Heart J* 1976;38:549-57.
- Nakaya Y, Hiasa Y, Murayama Y, et al. Prominent anterior QRS forces as a manifestation of left septal fascicular block. *J Electrocardiol* 1978;11:39-46.
- Reiffel JA, Bigger JT Jr. Pure anterior conduction delay: a variant "fascicular" defect. *J Electrocardiol* 1978;11:315-9.
- Gambetta M, Childers RW. Rate-dependent right precordial Q waves: "septal focal block." *Am J Cardiol* 1973;32:196-201.
- Piccolo E, Raviele A, Delise P, et al. The role of left ventricular conduction in the electrogenesis of left ventricular hypertrophy: an electrophysiologic study in man. *Circulation* 1979;59:1044-55.
- Sakai T. Left anterior fascicular block, left posterior fascicular block, left septal fascicular block [in Japanese]. *Ryoikibetsu Shokogun Shirizu* 1996;12:282-4.
- Moffa PJ, Ferreira BMA, Sanches PCR, et al. Bloqueio divisional ântero-medial intermitente em paciente com insuficiência coronária [in Portuguese]. *Arq Bras Cardiol* 1997;68:293-6.
- Uhley HN, Rivkin LM. Electrocardiographic patterns following interruption of the main and peripheral branches of the canine left bundle of His. *Am J Cardiol* 1964;13:41-7.
- Dabrowska B, Ruka M, Walczak E. The electrocardiographic diagnosis of left septal fascicular block. *Eur J Cardiol* 1978;6:347-57.
- Kulbertus HE. Concept of left hemiblock revisited: a histopathological and experimental study. *Adv Cardiol* 1975;14:126-35.
- Selvester RH, Wagner NB, Wagner GS. Ventricular excitation during percutaneous transluminal angioplasty of the left anterior descending coronary artery. *Am J Cardiol* 1988;62:1116-21.
- Scher AM, Young AC, Malmgren AL, et al. Activation of the interventricular septum. *Circ Res* 1953;3:56-64.
- Sodi-Pallares D, Calder RM. New bases of electrocardiography. St. Louis: CV Mosby Co; 1956. p. 282, 418, 489.
- MacAlpin RN. Absent septal Q waves in otherwise normal electrocardiograms: a variant of normal? *J Electrocardiol* 2001;34:207-14.
- Gunnar RM, Pietras RJ, Blackaller J, et al. Correlation of vectorcardiographic criteria for myocardial infarction with autopsy findings. *Circulation* 1967;35:158-71.
- Horan LG, Flowers NC, Johnson JC. Significance of the diagnostic Q wave of myocardial infarction. *Circulation* 1971;43:428-36.
- Hellerstedt M, Jonasson R, Orinius E. Electrocardiographic diagnosis of ventricular septal infarction. *Acta Med Scand* 1980;208:213-7.

31. Papparella N, Alboni P, Cappato R, et al. Prominent anterior forces: clinical, electrocardiographic and prospective study. *J Electrocardiol* 1987;20:233-40.
32. Hassett MA, Williams RR, Wagner GS. Transient QRS changes simulating acute myocardial infarction. *Circulation* 1980;62:975-9.
33. Jacobson LB, La Follette L, Cohn K. An appraisal of the initial QRS forces in left anterior fascicular block. *Am Heart J* 1977;94:407-13.
34. Antonin B, Roje J. The vectorcardiogram in left anterior hemiblock with qrs or qRS patterns in right precordial leads. *Adv Cardiol* 1976;16:507-11.
35. Dhala A, Gonzalez-Zuelgaray J, Deshpande S, et al. Unmasking the trifascicular left intraventricular conduction system by ablation of the right bundle branch. *Am J Cardiol* 1996;77:706-12.
36. Erickson Rv, Scher AM, Becker RA. Ventricular excitation in experimental bundle-branch block. *Circ Res* 1957;5:5-10.
37. Xiao HB, Gibson DG. Absent septal q wave: a marker of the effects of abnormal activation pattern on left ventricular diastolic function. *Br Heart J* 1994;72:45-51.