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Electrocardiographic manifestation of the middle fibers/septal fascicle block: a consensus report☆

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Abstract There are fibers in the left ventricle (LV) (LV middle network) that in around one third of cases may be considered a true septal fascicle that arises from the common left bundle. Its presence and the evidence that there are 3 points of activation onset in the LV favor the quadrifascicular theory of the intraventricular activation of both ventricles.

Since the 70s, different authors have suggested that the block of the left middle fibers (MS)/left septal fascicle may explain different electrocardiographic (ECG) patterns. The 2 hypothetically based criteria that are in some sense contradictory include: a) the lack of septal "q" wave due to first left and later posteriorly shifting of the horizontal plane loop and b) the presence of RS in lead V_2 (V_1-V_2) due to some anterior shifting of the horizontal plane vectorcardiogram loop. However, there are many evidence that the lack of septal q waves can be also explained by predivisional first-degree left bundle-branch block and that the RS pattern in the right precordial leads may be also explained by first-degree right bundle-branch block. The transient nature of these patterns favor the concept that some type of intraventricular conduction disturbance exists but a doubt remains about its location. Furthermore, the RS pattern could be explained by many different normal variants.

To improve our understanding whether these patterns are due to MF/left septal fascicle block or other ventricular conduction disturbances (or both), it would be advisable: 1) To perform more histologic studies (heart transplant and necropsy) of the ventricular conduction system; 2) To repeat prior experimental studies using new methodology/technology to isolate the MF; and 3) To change the paradigm: do not try to demonstrate if the block of the fibers produces an ECG change but to study with new electroanatomical imaging techniques, if these ECG criteria previously described correlate or not with a delay of activation in the zone of the LV that receives the activation through these fibers or in other zones.

Keywords: Intraventricular blocks; Block of middle fibers/left septal fascicle

What are the middle fibers of the left bundle?

Various authors have described fibers located between the superoanterior (SA) and inferoposterior (IP) division, using different nomenclature in the literature. For many authors,[1]

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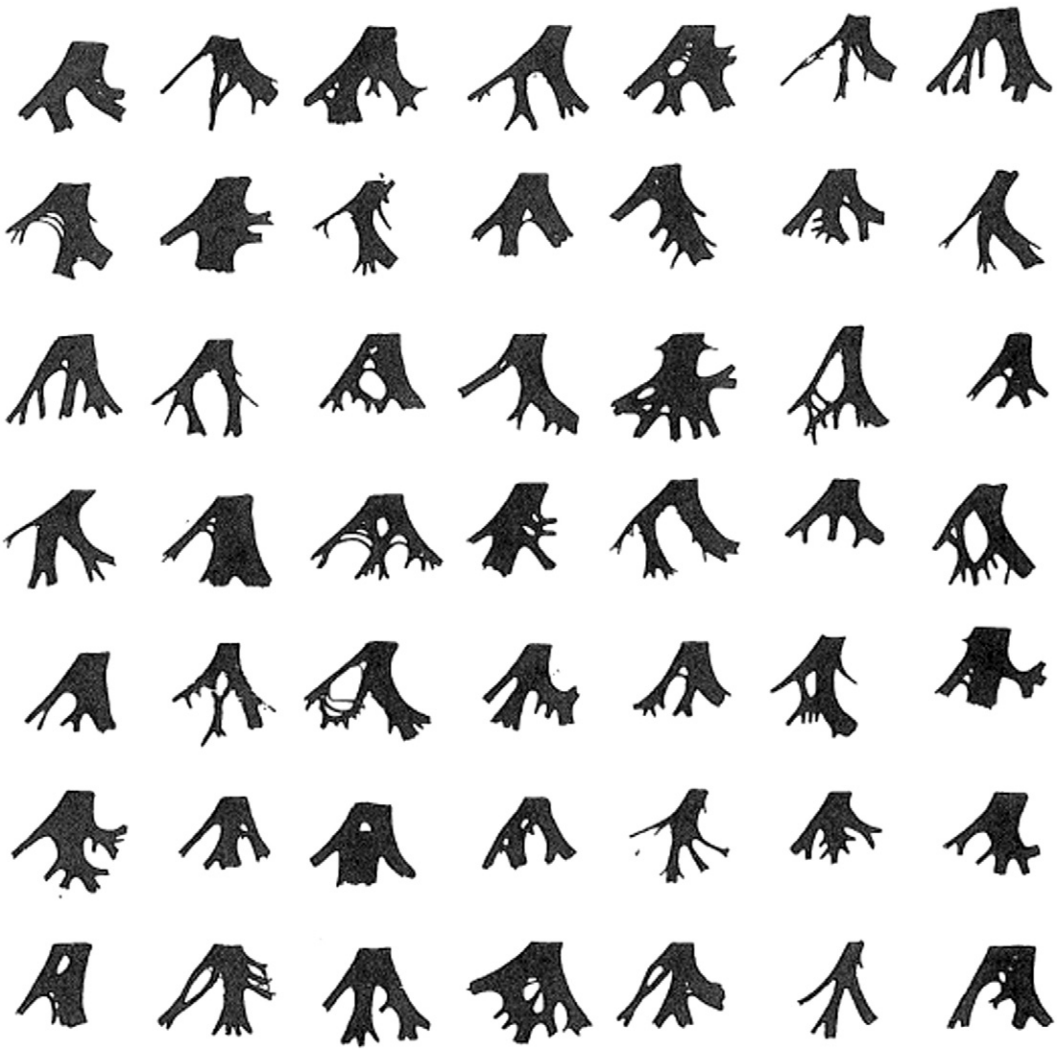

Fig. 1. Different pathologic aspects of the middle septal fibers of the left branch in 49 normal cases, according to Demoulin and Kulbertus.[4]

the best term was *left septal fascicle* (*SF*), whereas others[2] preferred the name *middle fibers* (*MF*). For the Rosenbaum School,[3] most of these fibers arise from the IP division, and for Demoulin,[4] it is a network of fibers that, in around two thirds of cases, a third subdivision could easily be identified but it arises from the common left bundle in only around one third of cases (Fig. 1). In this consensus paper, we will use the acronym MF/SF.

Ventricular activation: trifascicular vs quadrifascicular theory

Following the work of Rosenbaum et al,[3] it was believed that the intraventricular conduction system had 3 terminal divisions. According to this trifascicular theory of ventricular activation, the right bundle branch (RBB) represents 1 division, and the SA and IP divisions of the left bundle branch (LBB), which are true anatomical fascicles, represent the other 2 divisions. However, the middle anteroseptal fibers are thought by some authors to represent another division.[5,6] In fact, the experimental work of Durrer et al[7] in human hearts (Fig. 2) demonstrated that there are 3 points of activation onset in the left ventricular (LV) human heart, favoring the quadrifascicular theory of ventricular activation. Medrano et al[8] suggested that the LV activation may be preserved, in case of block of the SA and inferior division of the LBB, through these fibers. This may explain the pattern of left bundle-branch block (LBBB) with q wave in lateral leads, I, VL, V_5, and V_6 that it may be seen in some case of atypical LBBB pattern.

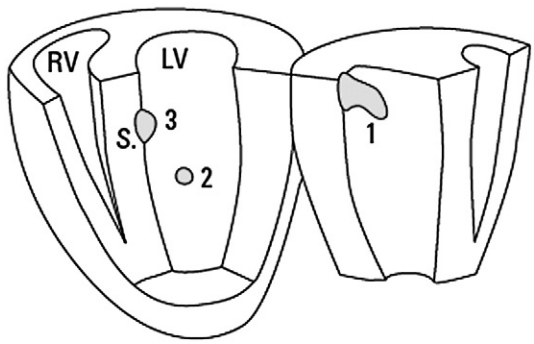

Fig. 2. The open LV shows the 3 points of LV activation according to Durrer et al.[7]

Do we agree on true universal electrocardiographic criteria for MF/SF block?

It has been suggested[9-11] that some electrocardiographic (ECG) changes can be caused by MF/SF block. We will comment on the ECG criteria for isolated MF/SF block, although the Brazilian School[11] has also reported the criteria of this block associated with right bundle-branch block (RBBB). The 2 well-known hypothetically based criteria that may be somehow contradictory include:

a) The block of MF/SF explains the initial QRS vector directed to the left with left posterior shift of the loop, expressed in the ECG by loss of septal q wave (R pattern) in leads V_5 and V_6. This criterion accompanied by other parameters (QRS duration ≤ 110 milliseconds; no slurring or notching on R upstroke in leads I, V_5, V_6) was emphasized by MacAlpin.[10]
b) The MF/SF block may explain that the activation of the anteroseptal area of the LV is delayed leading to some anteriorization of the QRS forces (loop) in the horizontal plane (HP), and this is associated to prominent anterior forces (PAF) that are manifested in the ECG as tall "R" in lead V_2 and occasionally in lead V_1. This criterion was first proposed by Hoffman et al[9] and later by others.[11-15] Currently, it is firmly supported by the Brazilian School.[1,11] Recently, major and minor diagnostic criteria of this type of block have been described.[1] The only major criterion, according to this hypothesis, was the presence of intermittent PAF, which is manifested in the ECG as prominent R wave in lead V_1 and especially in lead V_2 with progressive increasing of R voltage across the mid precordial leads and decreasing from leads V_5 to V_6. The minor criteria include a list of 13 patterns. According to Perez Riera et al,[1] the diagnosis of MF/SF block could be made with 1 major criterion or with 2 minor criteria.

Review of the major clinical studies

Although not exhaustive, the following is a list of the most important articles that described the 2 aforementioned criteria associated with MF/SF block.

Lack of septal q waves

Gambetta and Childers[16] had already suggested the possibility that septal-focal block may explain the rate-dependent right precordial q waves, and later, Hassett et al[17] published that transient QRS changes simulating acute anterior myocardial infarction may be explained by transient aberrancy in the SF, although he stated that a conclusive explanation was lacking. In addition, premature atrial stimulation (PAS) may reveal Q waves.[18] However, determining which fascicle is affected in case of lack of septal q waves and what was the degree of conduction delay remains highly controversial.

At the beginning of this century, MacAlpin[10,19] published 2 articles emphasizing that the block of MF/SF can induce changes in the initial part of the QRS, lack of q wave, that are more frequent than the presence of PAF. In fact, MacAlpin postulated after reviewing around 26 000 ECGs from the UCLA University that the major criterion for MF/SF block is the loss of septal q waves. In addition, MacAlpin affirmed that no example of PAF was found in his analysis. However, the examples of lack of septal q waves (Fig. 3) could also be explained by other types of aberrant complexes such as predivisonal first-degree LBBB.[20,21]

Presence of RS in V_1 to V_2

In 1976, Hoffman et al[9] were the first one to hypothesize that anterior conduction delay could explain PAF in patients apparently free of chronic coronary heart disease, right ventricular hypertrophy, or left anterior descending (LAD) ischemia. A conduction delay in the middle anteroseptal LV Purkinje network, he postulated, was a possible explanation for PAF because it may explain the anteriorization of the QRS loop in the HP.

In the same year, Kulbertus et al[6] published an article entitled "Anterior displacement of QRS: another form of intraventricular block," stating that, in 10% of patients with induced ventricular aberrancy, the vectorcardiogram (VCG) pattern was characterized only by PAF. He admitted that "this may correspond to some type of ventricular conduction disturbance (VCD) either of the RBB or the LV distal Purkinje network." He went on to say that "further studies are needed to determine on a firm basis which part of ventricular conduction system (VCS) is involved in the conduction delay responsible for these PAF."

Nakaya et al[12] published the case study of a patient with PAF occurring intermittently during exercise presenting also with intermittent advanced RBBB. He stated that PAF was a manifestation of transient SF block. However, the changes seen in this case could have been due to incomplete RBBB, especially in a patient that also developed intermittent advanced RBBB, in whom the necropsy study showed marked fibrosis in the SF, RBB, and IP fascicle.

In 1978, Reiffel and Bigger[22] published 2 cases of aberrancy produced by PAS that strongly suggested an additional form of ventricular conduction delay. They did not postulate which specific fascicle of the LBB was the anatomical substrate for it but observed that this PAF seemed independent of RBBB. However, Fig. 4 shows 1 sequence of panels (from A to D) that resembles a progressive RBBB pattern very similar to the ones recorded later by Piccolo et al[23]. It seems less likely that the aberrancy appears first as some type of LBBB (panel B) and later as RBBB (panels C and D), when progressive RBBB is more likely. However, the lack of q wave in the left precordial leads may be explained by the association with some other type of LV conduction defect.

In 1980, Piccolo et al[23] clearly demonstrated in healthy volunteers a progressive development of advanced RBBB pattern using PAS. He found that PAF and later different degrees of classical RBBB with R' in V_1 seemed to be generated by the same mechanisms: progressive right ventricular (RV) conduction delay. Furthermore, this

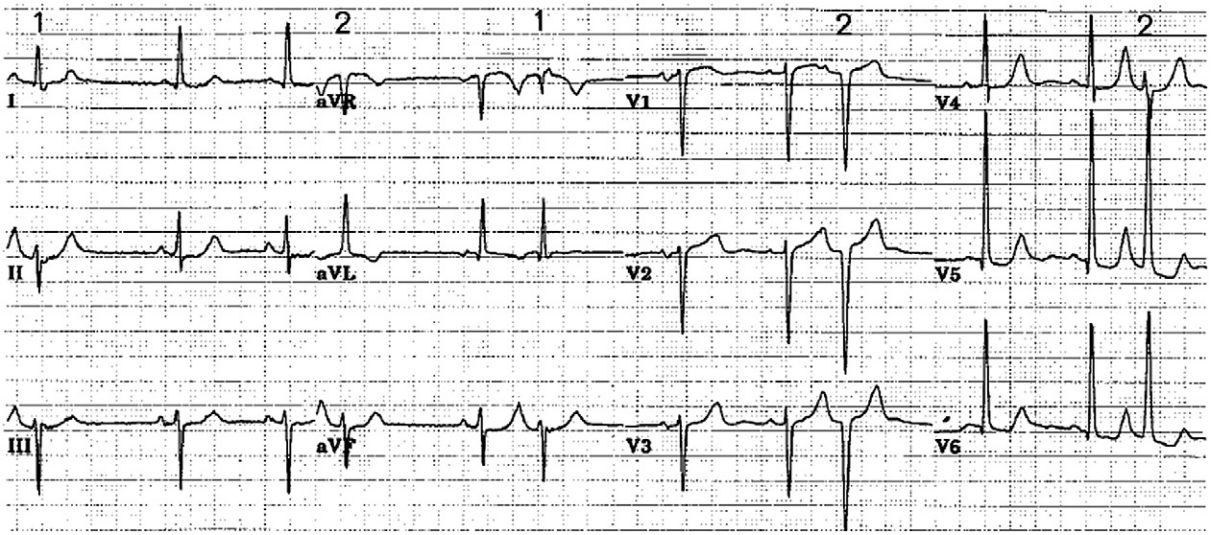

Fig. 3. From MacAlpin.[10] Two types of aberrant intraventricular conduction are caused by premature atrial beats. In beats labelled 1, there is no change in the direction of the initial QRS forces, but left anterior fascicular block combined with RBBB develops. In beats labelled 2, the initial forces shift leftward and posteriorly without changes in the mean frontal plane QRS axis or QRS duration. Loss of septal q waves and of initial r in leads V_1 to V_3 is probably caused by transient left septal fascicular block according the author (see text).

progressive appearance of RBBB was seen to be very similar to the progressive RBBB pattern that appears after "touching" the right bundle with a catheter[24] or the pattern observed in some recordings during increased heart rate.[3] Therefore, there is clear evidence that, in healthy people or in the presence of right ventricular enlargement, before the appearance of r' in lead V_1, there is an RS pattern that could be explained by anterior displacement of the VCG HP loop.

Alboni[25] stated that the pattern of permanent PAF in the ECG (rS or RS in leads V_1-V_2) in patients without heart disease is due to normal variants and never evolves to RBBB or LBBB during follow-up.

The Brazilian School published some cases, including those by Tranchesi et al[14] and Moffa et al.[15]

De Padua et al discussed the MF/SF block in many of his articles. He stated that it could be manifested as a PAF pattern in the HP (RS in leads V_1-V_2). However, the example used to illustrate this possible block[26] is identical to one shown by Alboni as an example of partial RBBB (Fig. 5).

In 2008, Pérez Riera et al[27] published a case describing a patient with ST-segment elevation myocardial infarction due to LAD occlusion with a transient increase of R wave in leads V_1 to V_2. Other similar cases have been also described[2] (Fig. 6). This change may be explained by a transient VCD, either in the RBB or the MF, or both, because both structures receive perfusion from septal branches. However, we cannot completely confirm that this is due to a MF/SF block.

Finally, in 2011 Pérez Riera et al[1] wrote a review discussing the anatomical and electrophysiological bases and the diagnosis of this type of intraventricular block. Those authors considered that the major criterion for the diagnosis of MF/SF block was the presence of transient PAF. However, although we can assure that some type of VCD exists during transient PAF, its location remains controversial. On the other hand, most of the minor criteria described

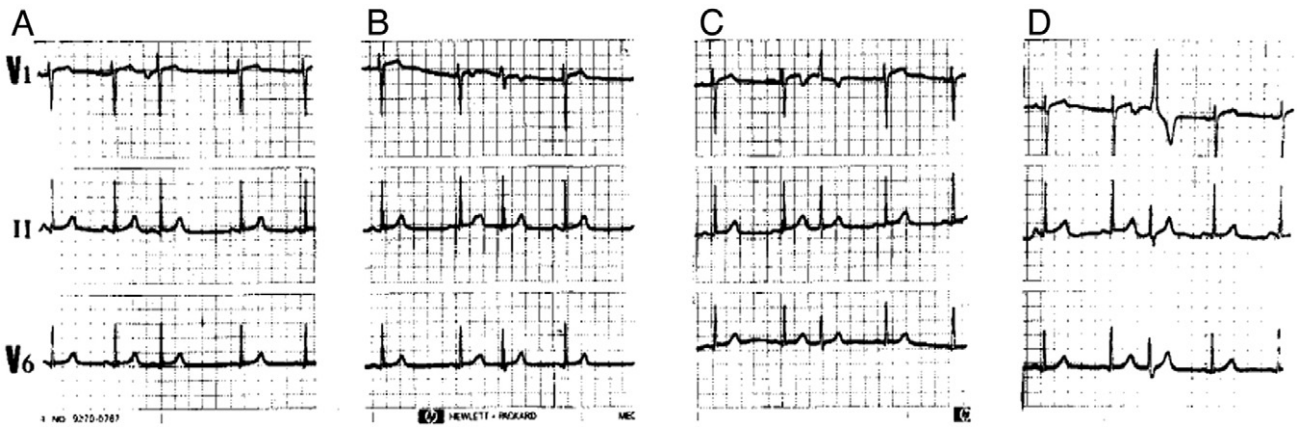

Fig. 4. From Reiffel and Bigger.[22] Composite of atrial premature beats obtained from an ECG rhythm strip (leads V_1, II, and V_6). With the shortening of the coupling interval (third QRS), a progressive pattern of RBBB pattern appears (from B to D). The author considers that the B pattern (RS in the third QRS complex) is explained by MF/LSF block and the others (C and D) by RBBB (see text).

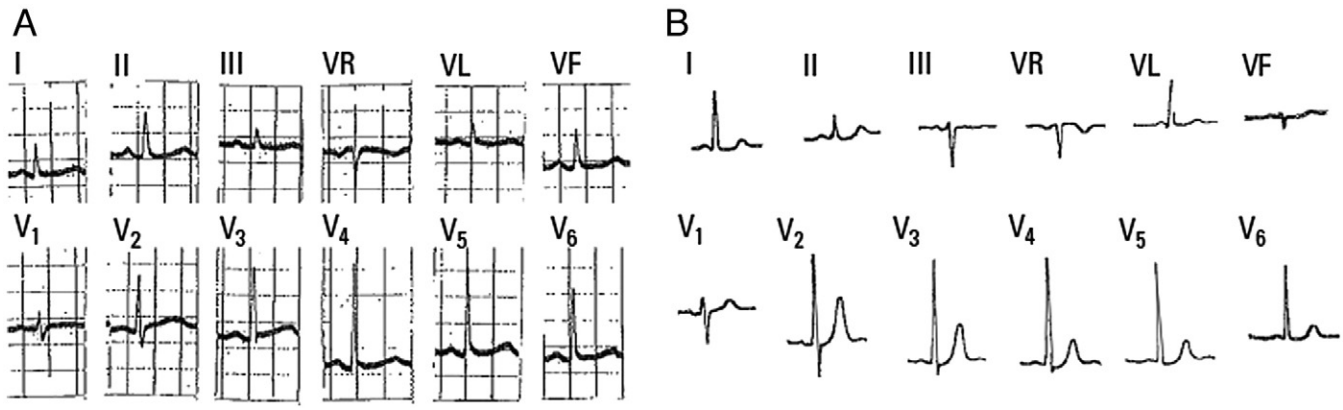

Fig. 5. A, Electrocardiogram with dominant R wave in right precordial leads (V_1 and V_2) with no other ECG abnormalities. Left medial fascicular block would be postulated (from De Padua et al[13]). B, Electrocardiogram with tall R wave in leads V_2 to V_3. This pattern may represent a variant form of incomplete RBBB (adapted from Alboni[25]).

in this review are hypothetical or based on parameters that may be found due to many circumstances such as predivisonal not advanced LBBB, differences in anatomy of the fibers, electrophysiological properties, size of the septum activated by the RBB, orientation of the first vector in relation to orientation of intraventricular septum, body habitus, distribution of LV Purkinje network, and others.

Can MF/SF block be experimentally reproduced?

There are a few experimental studies that have suppressed the middle Purkinje networks in dogs, which have an anatomical distribution similar to that in humans. The results of the obtained ECG patterns were not uniform.[3,8] Dabrowska et al[28] found that a lack of septal q wave, a pattern similar to incomplete LBBB, was the consequence of the block of the MF/SF fibers. Meanwhile, Uhley and Rivkin[29] and Nakaya et al[12] have demonstrated that there are different degrees of anterior shifting of the QRS loop in the HP after the section of the MF/SF. A question emerges: How can an isolated lesion in the middle septal fibers induce different ECG patterns?

Differences in the methodology used by different researchers or differences in the presence of additional local ischemia or electrolyte disturbances are possible explanations. Different types of middle septal fiber networks may also account for the discrepancies, and the anatomical structure and distribution of these middle fibers varied when compared with those in humans. In any case, experimental evidence demonstrating that middle fiber block may induce some ECG changes already exists, but confirmation and validation are still required.

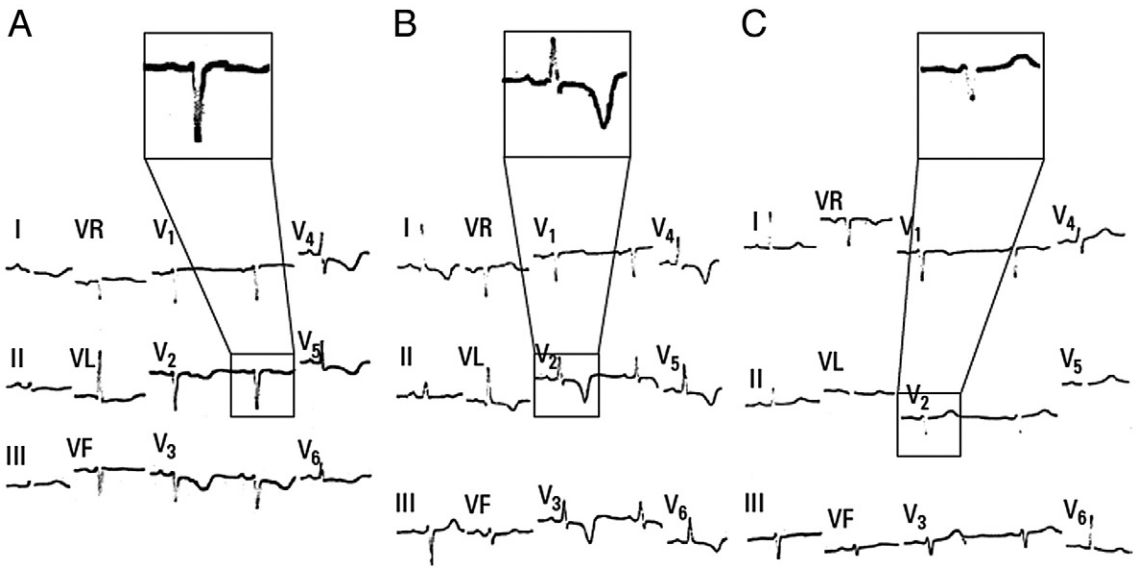

Fig. 6. A, Electrocardiogram from an ischemic heart disease patient (A) who, during an acute coronary syndrome (B), showed a significant morphologic change in lead V_2 (high transitory R), with very negative T wave in the right precordial leads (LAD involvement), which disappeared after some hours (C). As there is not any evidence of transient lateral ischemia, the transient pattern in lead V_2 (tall R wave) (B) may be explained by either a block of the middle fibers of the LBB or partial RBBB or both (see text).

Meanwhile, a fine-grid computer simulation[30] during middle LAD percutaneous coronary intervention (severe ischemia) has shown that when a conduction failure in the middle septal area happens, a transient decrease in the medial anterior vector occurs explaining the loss of voltage of R in lead V_2 instead of an increase in its voltage.

MF/SF block: myth or reality?

Even some of the strongest defenders of MF/SF block usually agree that no definitive evidence for the existence of an exact correlation between the presence of MF/SB block and its ECG manifestation in fact exists. Consequently, articles were often entitled with the question: "myth or reality?,"[19] "in search of septal fascicle block,"[10] or "septal fascicle block a chimera?"[13] It is true that transient ECG criteria are due to some type of block, but it is not always easy to determine where the block is anatomically located. The current knowledge of this problem is outlined below.

a) We will not discuss again on the nomenclature. This was extensively outlined in a recent review.[1] As said before, some of the coauthors of this manuscript prefer to name it middle fibers block because it better reflects the true nature of the problem accepting that only in around 33% of cases a true fascicle can be isolated and also because we would like to separate this type of block from the well-established blocks of the SA and IP fascicles described by Rosenbaum School in 1968. However, most of the articles that have been publishing about this issue prefer to use the term *SF block*.[1]
b) The experimental works previously discussed have demonstrated that some ECG changes occur after section of the septal middle fibbers or using simulation methods of activation. However, we have exposed that the ECG changes are not always the same and need further validation.
c) The pathologic involvement of the VCS is much more extensive than what it is suggested by ECG pattern changes.[31,32] As much as we know, there is only 1 case of histologic examination of the VCS[12] in a patient who presented transient prominent R in V_1 to V_2 during angina during an exercise testing. This patient also presented sometimes a transient RBBB pattern. The transient ECG pattern may also be explained by first-degree RBBB but was considered by the author typical MF/SF block. The histopathologic study showed marked fibrosis in RBB and middle fibers and left posterior division.
d) The transient lack of septal q wave is explained by some type of VCD. However, although this block may hypothetically be considered located in the MF, the lack of septal q wave could also be explained by a predivisonal not advanced (first-degree) LBBB[20,21] (Fig. 3). The transient presence of PAF may also be explained by transient intraventricular conduction defect (right bundle [RB] or perhaps MF/SF) and even transient WPW.
e) The permanent ECG pattern of PAF has been described as due to incomplete RBBB,[25,33] some type of WPW preexcitation, RV enlargement, lateral infarction, or possible MF/SF block.[11,26,27] In case of incomplete RBBB, a slurred or small notch in the ascending limb of the S wave is often observed. However, in other instances, a fine RS pattern is seen and in the VCG study of Piccolo et al,[23] there was no more slowness of the terminal part of the loop than before the anterior shifting of the loop.

Based on all these data, we could state that the current perspective about the presence of PAF (RS in leads V_1-V_2 with RS in lead V_6) is the following:

a) In healthy people or in individuals without LV heart disease, the transient appearance of PAF during the development of advanced RBBB, as has been demonstrated by Piccolo et al[23] and Peñaloza et al,[24] highly likely corresponds to a predivisional first-degree RBBB.

b) In healthy people, the presence of permanent PAF may be due to many normal variants, including a variety of LV Purkinje distribution (less Purkinje fibbers in the anteroseptal zone that induces a delay in the activation of this area without pathologic block). This last hypothesis, the different distribution of Purkinje fibers, has been also used to explain the presence of S1 S2 S3 pattern in healthy people.[34] This explains why the ECG pattern remains unmodified during the long term.[33,34]
c) In people with LV heart disease, especially LAD occlusion without lateral ischemia, the transient appearance of PAF may be explained by some type of VCD, block of the MF/SF, and/or some type of RBBB. It is conceivable that a block of both structures may be involved. The right bundle and the middle septal fibers receive perfusion from the LAD septal branches, and therefore, the obstruction of the LAD may induce block of either of the structures or both.
d) The presence of permanent PAF in patients with LV heart disease (LV hypertrophy, ischemic heart disease, or congestive heart failure) may be, in some cases, due to some type of VCD, including MF/SF block. However, it is mandatory to rule out the presence of septal hypertrophy, lateral ischemia, biventricular enlargement, and others.

It is probably due to these uncertainties that the guidelines on VCD from the American Heart Association[35] accept that MF/SF block may exist but that it is not possible to be conclusive about it and literally states: "The term *left septal fascicular block* is not recommended because of the lack of universally accepted criteria."

Recommendations for the future

To better clarify whether MF/SF block may certainly explain the prominent anterior shifting of the vectorcardiogram loop and the presence of rS/RS in leads V_1 to V_2, considered the most frequently accepted criterion, and/or to recognize if this block may explain the lack of septal q wave or other criteria, it would be necessary:

1) To change the current concept of block based on the site where the VCS is injured, by the concept that considers the site of delayed activation, probably in this case in some area of the RV or in the anteroseptal wall of the LV or both, trying to demonstrate using new electrophysiological-anatomical imaging techniques, which is the endocardial place of delayed activation when this ECG pattern is observed. Probably, this would be the best way to ascertain a delay of activation in the septal region or the more extensive anteroseptal or RV regions.
2) To perform new experimental well-designed studies to definitively confirm which are the ECG changes obtained after different types of section or injury of the LV Purkinje network.
3) To perform more studies (necropsy and heart transplant) to recognize the involved area of VCS in case of the presence of permanent and especially transient PAF and lack of septal q waves.

It is also advisable to learn more about the clinical significance of this conduction disorder, especially about the appearance of PAF during acute coronary syndromes due to LAD occlusion, during percutaneous coronary intervention and others.

We hope that a detailed scientific process including the studies mentioned above would allow concluding on a correlation between the ECG pattern and the location of this fascinating conduction disorder.

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