Coronary Angiographic Significance of Left Anterior Fascicular Block During Acute Myocardial Infarction

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The clinical and angiographic significance of isolated left anterior fascicular block occurring during the early stage of acute myocardial infarction was studied in 141 consecutive patients who underwent cardiac catheterization before hospital discharge. Left anterior fascicular block occurred in 15 of the 62 patients with an anterior wall infarction and in 13 of the 79 with an inferior infarction. None of the clinical characteristics differed among patients with or without left anterior fascicular block. The number of coronary vessels with significant stenosis, the Friesinger and the Gensini scores for severity of stenosis and the ejection fraction were also similar in the two groups. Patients with left anterior fascicular block had more severe narrowing of the coronary artery supplying the infarct zone (88 ± 21 versus 70 ± 35%, $p < 0.001$) and tended to have less developed collateral circulation (collateral score 0.7 ± 0.8 versus 1 ± 0.8, $p = 0.10$). A significant stenosis of the left anterior descending coronary artery was found as frequently in patients with as in those without left anterior fascicular block (64 versus 65%); 29% of the patients with inferior wall infarction and left anterior fascicular block had left anterior descending coronary artery stenosis compared with 47% of the patients without this conduction disturbance (no significant difference). When the infarction was located anteriorly, a significant stenosis of the proximal segment of the left anterior descending coronary artery was present in 47% of the patients with and in 45% of the patients without left anterior fascicular block.

In this study, the occurrence of left anterior fascicular block during the course of an acute myocardial infarction was not an indication of left anterior descending coronary artery involvement or of more extensive coronary disease. Other pathophysiologic mechanisms, such as a dual blood supply to the anterior fascicle of the left bundle branch or a longitudinal dissociation of conduction in the His bundle, could be involved in the etiology of left anterior fascicular block.

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The association between left anterior fascicular block and coronary artery disease has been recognized for many years (1–13). It has been suggested that the occurrence of such block in patients with coronary disease is a sign of proximal left anterior descending coronary artery stenosis (10–15) and that its coexistence with inferior wall myocardial infarction is suggestive of additional left anterior descending coronary artery involvement and more severe coronary disease (14).

However, studies correlating left anterior fascicular block and coronary angiography (10,14–16) are scarce; they have been performed retrospectively in small series of selected patients without reporting the time elapsed between the occurrence of the block and the angiographic study and without control patients. Acute myocardial infarction, however, is often associated with the appearance of new left anterior fascicular block (7,8,11) and presents a situation in which the appearance of the conduction defect can be correlated with an acute coronary event. Recognition of its association with a particular anatomic lesion could help in the noninvasive evaluation of the extent of coronary artery disease in the patient with myocardial infarction and may have relevant clinical and therapeutic implications.

To better understand the pathophysiology and the clinical significance of the presence of left anterior fascicular block, we studied prospectively the angiographic correlates of such block in a consecutive series of patients admitted to the coronary care unit with a diagnosis of acute myocardial infarction.

Methods

Study patients. From April 1982 to January 1983, there were 155 consecutive patients admitted to our coronary care unit because of an acute myocardial infarction who survived

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the acute stage and underwent cardiac catheterization before hospital discharge as part of a prospective study. Patients older than 70 years of age and those with previous coronary artery bypass surgery were excluded from the study.

Acute myocardial infarction was diagnosed in the presence of two of the following three criteria: myocardial ischemic pain lasting more than 30 minutes, serum levels of creatine kinase elevated above twice the upper limit of normal with presence of the MB isoenzyme and Minnesota code electrocardiographic criteria for evolving acute infarction (17). The infarction was considered anterior when electrocardiographic changes appeared in two or more of leads I, aVL, V_1, V_2, V_3 or V_4 and inferior when they appeared in leads II, III or aVF (18). True posterior wall infarction was diagnosed and classified as inferior when an R/S ratio of 1 or more appeared in leads V_1 and V_2 (19). When the electrocardiographic changes were present in leads V_5 or V_6, the myocardial infarction was considered anterior if associated changes were present in the precordial leads or as inferior if they occurred in the inferior leads. The 12 lead electrocardiographic tracing was obtained twice on the day of hospital admission, daily thereafter for 4 days and then every other day. Electrocardiograms were also recorded every time a patient reported chest pain and on the day before and after coronary angiography. All electrocardiographic tracings were obtained using a Marquette three channnel electrocardiographic recorder.

Coronary angiographic study. Coronary arteriography was performed before hospital discharge at a mean of 10 ± 3 days after the infarction by a percutaneous transfemoral approach using preformed catheters as previously described (20). Each artery was filmed in four to six projections including special angulated views in the sagittal plane (21). All images were recorded on 35 mm film at 50 frames/s, and reviewed on a Tagarno projector and interpreted by a consensus of three independent observers unaware of the clinical condition of the patients. Stenoses of coronary vessels were coded according to the criteria of the American Heart Association (22). Stenoses of 70% or greater of the arterial intraluminal diameter of the right coronary artery, left anterior descending coronary artery or circumflex branches of the left coronary artery and stenoses of 50% or greater of the left main coronary artery were considered significant. Stenoses of large diagonal or marginal branches were considered lesions of the left anterior descending and circumflex coronary artery, respectively. A stenosis of a large diagonal branch without other significant stenosis in the left anterior descending coronary artery was present in nine patients, four with anterior and five with inferior myocardial infarction. A proximal lesion in the left anterior descending coronary artery was defined by the presence of a stenosis located before the first septal branch.

The coronary collateral circulation was considered good when visible collateral vessels completely opacified the recipient coronary artery and its branches distal to the stenosis and poor when the opacification was incomplete. A collateral circulation score to the vessel supplying the infarcted zone was defined as 0 when no collateral vessels were visualized, 1 when they were poor and 2 when they were good. The Gensini and Friesinger scores (23,24) for severity of coronary stenosis were calculated for each patient. The degree of the remaining stenosis of the vessel supplying the infarcted zone was also determined. When both the left circumflex and the right coronary arteries were significantly involved in inferior infarction, the artery showing the more severe stenosis was considered responsible for the infarction. Left ventriculograms were obtained in the 30° right anterior oblique projection and the ejection fraction was calculated by the area-length method (25).

Electrocardiographic criteria for left anterior fascicular block. The criteria of Fisher et al. (26) partially modified by Zema (27), were used. Left anterior fascicular block was diagnosed when all of three following electrocardiographic criteria were met: 1) left axis deviation; 2) deep negative terminal deflection (S wave) in lead II wider than any preceding initial negative deflection (Q wave); and 3) a terminal positive deflection (R wave) in lead aVR (Fig. 1). A shift of the QRS axis beyond −30° was required in this study for the diagnosis of left axis deviation. Since the electrocardiographic recognition of left anterior fascicular block is not possible with these criteria in the presence of

Figure 1. Left anterior fascicular block in the presence of inferior myocardial infarction. The criteria used were those of Fisher et al. (26) and Zema (27). See text.

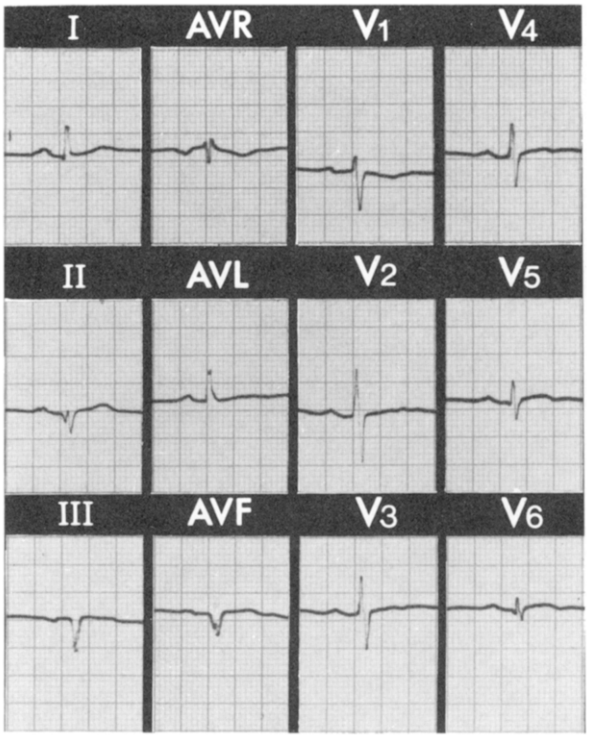

Table 1. Clinical Correlates of Left Anterior Fascicular Block in Patients With Acute Myocardial Infarction

	Left Anterior Fascicular Block*	
	Present (n = 28)	Absent (n = 113)
Age (yr)	54 ± 8	55 ± 8
Sex: male	21 (75%)	98 (87%)
History of hypertension	13 (46%)	41 (36%)
Previous MI	4 (14%)	25 (22%)
History of angina	10 (36%)	40 (35%)
Non Q wave MI	7 (25%)	36 (32%)
Peak CK (IU/liter)	1,742 ± 996	1,596 ± 1,370
Peak MB-CK (IU/liter)	260 ± 206	265 ± 262
Killip class	1.4 ± 0.6	1.4 ± 0.6
Early angina post MI	5 (18%)	20 (18%)
MI extension	2 (7%)	8 (7%)

*There were no significant differences in the variables cited between the patients with and without left anterior fascicular block. CK = creatine kinase enzyme; MB-CK = MB fraction of creatine kinase; MI = myocardial infarction; n = number of patients.

right or left bundle branch block, patients with these conduction disturbances were excluded.

Statistical analysis. Differences among continuous variables were analyzed by the unpaired *t* test. Discrete variables were compared with the chi-square test using the Yates' correction when appropriate. The null hypothesis was rejected when the probability values were 0.5 or less. Results were expressed as mean ± 1 standard deviation.

Results

Incidence. Of the total group of 155 patients, 9 developed right bundle branch block and 5 left bundle branch block during hospitalization and were excluded from further analysis. Among the remaining 141 patients, 28 met the criteria for left anterior fascicular block: 15 of the 62 patients with an anterior wall myocardial infarction and 13 of the 79 patients with an inferior myocardial infarction. In only one patient was the presence of the left anterior fascicular block documented before the infarction. In 10 patients (6 with an anterior and 4 with an inferior infarct), the left anterior fascicular block was present on the first electrocardiogram recorded and it was not possible to determine whether the block existed previously. The left anterior fascicular block was transient in seven patients. Twenty-five patients had spontaneous angina during their hospitalization; the electrocardiogram obtained in these patients during episodes of chest pain did not show appearance of a transient left anterior fascicular block. In one patient the shift in the QRS axis became more accentuated from −38 to −76°.

Clinical correlates of left anterior fascicular block. None of the clinical characteristics studied (age, sex, history of hypertension, previous myocardial infarction or angina, peak creatine kinase and MB isoenzyme values, Killip class during the acute stage, Q wave versus non Q wave myocardial infarction and angina and extension of myocardial infarction during the hospitalization) was associated with the occurrence of left anterior fascicular block (Table 1).

Angiographic correlates of left anterior fascicular block. The extent and the severity of the coronary artery lesions were not different in patients with and without left anterior fascicular block as assessed by the number of significantly stenosed vessels (1.8 ± 0.7 versus 1.8 ± 0.8, p = NS) (mean ± standard deviation) and the Gensini and the Friesinger scores (Table 2). Furthermore, stenosis of the left anterior descending coronary artery and its proximal segment was present in the same proportion of patients in the two groups. Left ventricular ejection fraction was also the same (50 ± 13 versus 50 ± 14%).

Patients with left anterior fascicular block had a significantly more severe degree of stenosis of the vessel supplying the infarct zone (88 ± 21 versus 70 ± 35%, p = 0.0008). The collateral circulation score to the infarct-related vessel was also lower but the difference did not reach statistical significance (0.7 ± 0.8 versus 1 ± 0.8, p = 0.10).

In the subset analysis of patients with an anterior wall myocardial infarction, none of the angiographic characteristics differed (Table 3). Forty-seven percent of the patients with and 45% of the patients without left anterior fascicular block had significant stenosis of the proximal segment of the left anterior descending coronary artery (p = NS). Similarly, patients with left anterior fascicular block could not be identified by the number of stenosed coronary vessels (1.8 ± 0.7 versus 1.7 ± 0.9, p = NS), the involvement of the left anterior descending coronary artery (100 versus 91%, p = NS), the percent stenosis of the vessel supplying

Table 2. Angiographic Correlates of Left Anterior Fascicular Block in Patients With Acute Myocardial Infarction

	Left Anterior Fascicular Block		
	Present (n = 28)	Absent (n = 113)	p Value
Number of diseased vessels* per patient	1.8 ± 0.7	1.8 ± 0.8	NS
Number of stenosed vessels*			
LAD	18 (64%)	74 (65%)	NS
Proximal LAD	7 (25%)	30 (27%)	NS
Proximal or mid LAD	16 (57%)	63 (56%)	NS
RCA	14 (50%)	77 (68%)	NS
LCx	17 (61%)	50 (44%)	NS
Stenosis infarct-related vessel (%)	88 ± 21	70 ± 35	0.0008
Gensini score	53 ± 33	50 ± 33	NS
Friesinger score	8.8 ± 2.9	8.6 ± 2.9	NS
Ejection fraction (%)	50 ± 13	50 ± 14	NS
Collateral score to infarct-related vessel	0.7 ± 0.8	1 ± 0.8	$p < 0.10$

* = ≥70% stenosis; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; n = number of patients; RCA = right coronary artery.

the infarcted zone (95 ± 7 versus 90 ± 18%), the collateral circulation score (0.7 ± 0.7 versus 0.6 ± 0.6, p = NS) and the ejection fraction (43 ± 10 versus 47 ± 15%, p = NS).

However, left anterior fascicular block in patients with inferior myocardial infarction was associated with two angiographic characteristics (Table 3): the degree of the residual stenosis of the vessel supplying the infarcted zone was more marked (81 ± 28 versus 56 ± 38%, $p < 0.02$) and the collateral circulation score to the infarct-related vessel was smaller (0.7 ± 0.9 versus 1.3 ± 0.8, $p < 0.05$). Twenty-three percent of the patients with left anterior fascicular block had a significant stenosis of the left anterior descending coronary artery compared with 47% of those without (p = NS). A proximal stenosis of the left anterior descending coronary artery was not seen in any of the 13 patients with left anterior fascicular block, but was present in 9 of the 66 patients without the block (p = NS). The number of stenosed vessels, the Gensini and Friesinger scores and the ejection fraction were similar in both groups.

The statistical analysis was repeated by excluding the 11 patients who had left anterior fascicular block on admission.

Table 3. Angiographic Correlates of Left Anterior Fascicular Block in Patients With Anterior and Inferior Wall Myocardial Infarction

	Anterior Myocardial Infarction (n = 62)		Inferior Myocardial Infarction (n = 79)	
	LAFB Present (n = 15)	LAFB Absent (n = 47)	LAFB Present (n = 13)	LAFB Absent (n = 66)
Number of diseased vessels per patient*	1.8 ± 0.7	1.7 ± 0.9	1.7 ± 0.7	1.9 ± 0.8
Number of stenosed vessels*				
LAD	15 (100%)	43 (91%)	3 (23%)	31 (47%)
Proximal LAD	7 (47%)	21 (45%)	0 (0%)	9 (14%)
Proximal or mid LAD	14 (93%)	39 (83%)	2 (15%)	24 (36%)
Diagonal branch alone	1 (7%)	3 (6%)	1 (7%)	4 (6%)
RCA	5 (33%)	17 (36%)	9 (69%)	60 (91%)
LCx	7 (47%)	18 (38%)	10 (77%)	32 (48%)
Stenosis infarct-related vessel (%)	95 ± 7	90 ± 18	81 ± 28	56 ± 38†
Gensini score	62 ± 30	54 ± 36	43 ± 36	46 ± 30
Friesinger score	9 ± 3	9 ± 3	8.6 ± 3	8.6 ± 3
Ejection fraction (%)	43 ± 10	47 ± 15	58 ± 10	52 ± 13
Collateral score to infarct-related vessel	0.7 ± 0.7	0.6 ± 0.6	0.7 ± 0.9	1.3 ± 0.8‡

*≥70% stenosis; †$p < 0.02$; ‡$p < 0.05$. LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; LAFB = left anterior fascicular block; n = number of patients; RCA = right coronary artery.

Again, in the 17 other patients with new left anterior fascicular block, a more severe residual stenosis (83 ± 25 versus 70 ± 35%, $p = 0.08$) and a lower collateral circulation score (0.58 ± 0.8 versus 1.04 ± 0.8, $p < 0.05$) were found. All other clinical and angiographic characteristics were similar to those previously described for the total group. We also examined the data by using different left axis deviation criteria for the diagnosis of left anterior fascicular block. Including patients with an axis between 0 and −30° added three patients in the group with an anterior wall infarction and block and three in the group with an inferior wall infarction but did not affect the overall results. Use of a more stringent criterion of more than 60° axis deviation left only six patients with left anterior fascicular block and anterior wall infarction and two with inferior infarction; of these last two, one had a stenosis of the left anterior descending coronary artery and the other did not.

Discussion

The appearance of left anterior fascicular block during the acute stage of myocardial infarction was not associated in this study with the presence of significant left anterior descending coronary artery stenosis or with more extensive coronary artery disease or more severe impairment in left ventricular function. These findings contrast with the generally accepted view that the presence of left anterior fascicular block is suggestive of disease of the left anterior descending coronary artery and stimulates interest to understand the pathophysiology of this conduction defect.

Previous correlations with coronary angiography. Rosenbaum and coworkers (12,13) first suggested in 1970 that the presence of this conduction disturbance in patients with angina pectoris was indicative of left anterior descending coronary artery involvement. McKeever et al. (16) also noted a significantly greater frequency of three vessel coronary disease (78%) and a strong probability (97%) of left anterior descending coronary artery disease among patients with left anterior fascicular block and old myocardial infarction who underwent cardiac catheterization for angina pectoris. Levy et al. (14) reported a series of 20 patients with left anterior fascicular block derived from 283 consecutive patients with angina and significant coronary artery stenoses. All 20 patients had extensive coronary artery disease and a significant lesion of the left anterior descending coronary artery; 4 had stenosis of the left main coronary vessel. In a later study, the same authors (15) reported the angiographic findings in six patients with unstable angina at rest who developed transient left anterior fascicular block during ischemic attacks; all had severe stenosis of the left anterior descending coronary artery.

Most investigators and clinicians consider that left anterior fascicular block in patients with coronary artery disease is associated with obstruction of the proximal left anterior descending coronary artery because it is generally believed that blood supply to the anterosuperior fascicle of the left bundle branch originates exclusively from the septal branches of the left anterior descending coronary artery (10, 12–15, 28). In our study of nonselected patients with acute myocardial infarction, we could not document this association between left anterior descending coronary artery disease and left anterior fascicular block. Indeed, only 23% of the patients with inferior infarction who developed left anterior fascicular block had significant stenosis of the left anterior descending coronary artery and none had stenosis in its proximal segment. These results suggest that left anterior fascicular block may not be related to a single pathophysiologic mechanism.

Etiology of left anterior fascicular block. In 1973, Frink and James (29) demonstrated in human subjects that the blood supply to the anterosuperior fascicle of the left bundle branch originated in 50% of the cases not only from the anterior septal branch of the left anterior descending coronary artery, but also from the atrioventricular (AV) nodal artery, a branch of the right coronary artery in 90% of the cases and of the left circumflex coronary artery in 10%. In 1 of the 10 patients studied, blood to the anterosuperior fascicle of the left bundle branch was supplied exclusively by the AV nodal artery. Thus, anatomic data support the observation that occlusion of the proximal segment of the left anterior descending coronary artery is not a prerequisite for the occurrence of left anterior fascicular block.

Other mechanisms could also be involved in the etiology of left anterior fascicular block and explain our findings. James and Sherf (30) and Watt and Pruitt (31) developed the concept of longitudinal dissociation of conduction in the human His bundle giving rise to abnormal patterns of ventricular activation and producing left anterior fascicular block without an anatomic lesion in the left bundle itself. This concept is based on the histologic demonstration that the His bundle is partitioned into narrow cords by collagen running in its long axis with relatively little cross connections within each strand (31). The entire conduction system can thus be isolated in various cords insulated from one another by collagen, providing the anatomic setting for asynchronous conduction or longitudinal dissociation. Narula (32), in an attempt to reproduce the physiologic counterpart of these anatomic findings, demonstrated normalization of the QRS duration and axis, the HV interval and the ventricular activation time in patients with partial or advanced bundle branch block by pacing the distal portion of the His bundle; advanced left bundle branch block was also induced in patients with a narrow QRS complex by selective His bundle pacing (33). El-Sherif et al. (34) reproduced experimentally the same results in dogs: ligation of the anterior septal artery resulted in intra-His conduction delay with split His bundle potentials and right or left bundle branch block; distal His pacing normalized conduction in 67% of these

dogs. Left anterior fascicular block has also been reported during right coronary arteriography (35) and right heart catheterization (36), reinforcing the concept of intra-His disturbance in the presence of longitudinal dissociation of conduction within the His bundle. This structure receives a dual blood supply form the anterior septal branch of the left anterior descending coronary artery and from the AV nodal artery (29). Thus, although the phenomenon of left bundle branch block produced by intra-His conduction delay in the presence of longitudinal dissociation of conduction within the His bundle has not been studied in patients with acute myocardial infarction, our results suggest that ischemia in the His bundle produced by interrupting flow to the septal branches of the left anterior descending coronary artery or to the AV nodal artery, or both, could lead to left anterior fascicular block with or without associated right bundle branch block.

Other possible mechanisms of left anterior fascicular block could be peri-infarction and functional blocks. Indeed, electrocardiographic-vectorcardiographic findings merely translate the summation of electric forces derived from the myocardium. Thus, intramural delay distal to the Purkinje-myocardium junction could conceivably result in patterns of conduction delay and of block usually attributed to lesions in the specialized conducting tissues (37).

Our results also suggest a protective mechanism of the collateral circulation for the occurrence of left anterior fascicular block. The quantity and the quality of the collateral circulation to the vessel supplying the infarcted zone as assessed by the collateral circulation score was greater in patients without the conduction disturbance than in patients with inferior wall infarction; although this result could represent a type I error caused by multiple examination of the data, this explanation is unlikely considering that the degree of stenosis of the infarct-related vessel was less severe in that group of patients (38).

Limitations of the study. Vectorcardiographic studies were not performed in this study and the results are only valid with the electrocardiographic criteria used. The electrocardiographic recognition of left anterior fascicular block in the setting of inferior myocardial infarction is difficult and often confusing (28). However, we have applied the recently reported criteria of Fisher et al. (26) partially refined by Zema (27) to increase the specificity. With these criteria, left anterior fascicular block can be recognized in the presence of inferior infarction with a near 100% specificity (26,27,39). Thus, our results are probably applicable to all cases of left anterior fascicular block and comparable with those from vectorcardiographic studies. Use of different degrees of left axis deviation in the diagnosis of the block did not change the results (26).

Other possible limitations of this study could result from a bias selection because patients who died in the early hospital phase and patients with advanced right or left bundle branch block were excluded. Furthermore, the results apply to patients with acute evolving infarction and may differ for some unknown reason from the results previously reported in other studies which were obtained in patients with chronic stable angina.

Conclusions. Our study demonstrates that the appearance of left anterior fascicular block during acute myocardial infarction is not a sign of a coexistent significant stenosis of the left anterior descending coronary artery or of more severe or extensive coronary artery disease. This study also suggests that in these patients, other mechanisms such as the degree of the coronary collateral circulation may play a role in the occurrence of this conduction disturbance and supports the experimental and clinical reports that left fascicular block may be due to lesions involving the His bundle by means of a longitudinal dissociation of this structure. Further electrophysiologic studies would be needed to document this hypothesis.

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