Inferior myocardial infarction

Correlation of selective cine coronary arteriographic and ventriculographic findings with form of QRS complex in lead AVF

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The correlation of the electrocardiogram with meticulous postmortem arteriographic and pathologic studies in inferior myocardial infarction was a major contribution of Myers et al.[1] The purpose of this paper is to correlate the results of arteriographic and ventriculographic studies with the electrocardiogram in the same disorder.

The electrocardiograms of 2,000 consecutive patients who had cardiac catheterizations were studied using magnified 70 mm transparencies. Those tracings with peculiarities of the QRS configuration in lead AVF were selected. The original electrocardiograms, recorded on high-fidelity photographic machines (Sanborn Twin Beam) at a speed of 25 mm/sec, were then classified according to a preconceived scheme (*Table 1*), without knowledge of the catheterization findings. The width of the Q wave was measured from its onset to the point at which the R wave crossed the baseline. The data obtained by cardiac catheterization, including selective coronary arteriography and ventriculography, were then studied and correlated with the various abnormalities of AVF. Of the 340 patients chosen for review, selective coronary arteriograms had been done in 337 and left ventriculograms had been done in 309.

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Table 1. Classification of electrocardiographic changes

Group	Description
A	(multiphasic) or splintered QRS complex not preceded by a Q
A_1	QRS 0.5 mv or less
A_2	QRS greater than 0.5 mv
B	splintered QRS preceded by a Q = or <0.03 sec
B_1	QRS 0.5 mv or less
B_2	QRS greater than 0.5 mv
C	qR or qRs Q/R ratio <0.25, Q < 0.03 sec; T wave abnormality
D	Q/R ratio 0.25+; <0.5 mv; Q = or <0.03 sec
E	rSr, or rS, coarsely slurred descending limb S
E_1	Q < 0.03 sec
E_2	no Q wave
F	rS or RS, distinct notched ascending limb of r or R
F_1	Q < 0.03 sec
F_2	no q wave
G	R < 0.1 mv preceding S or SR′ configurations
G_1	0.5 mv or less
G_2	greater than 0.5 mv
H	QRS complex with a Q > 0.03 sec
I	QS complex without left bundle branch block
J	QR complex with Q/R ratio >0.25; Q < 0.03 sec
K	qR or qRs complex, Q of 0.03 sec
K_1	no T wave abnormality
K_2	T wave abnormality
L	qrS or qRs, Q < 0.03 sec; abnormal left axis deviation

The coronary arteriograms were performed by the technique previously reported,[2] and the degree of obstruction in the various arteries was estimated. The ventriculographic findings are shown in *Table 2*. The abnormal ventriculograms are classified according to the distribution of defective contractility: inferior, generalized, or apical.

Results

The ventriculographic findings in 309 patients with abnormalities in AVF are shown in *Table 2*. Only in Group H (QRS complex with a Q > 0.03 sec) did the majority of patients have ventriculograms showing impaired or absent contractility of the inferior portion of the left ventricle (29 of 42). If the five patients in this group having generalized decrease in contractility are added to those having localized impairment, then 34 of 42 had abnormal contractility of the inferior portion of the left ventricle. One of the five patients had diffuse impairment of contractility and severe obstruction in the anterior descending artery only; he was thought to have primary myocardial disease as well as coronary disease. Another patient showing diffuse impairment had localized partial obstruction of the anterior descending artery and severe disease in the aortic valve. Five patients in group H had apical infarctions, but without apparent infarction of the inferior wall. One patient (in Group H) had idiopathic hypertrophic subaortic stenosis. Therefore, in Group H, 40 of 42 patients had some condition of the

left ventricle that could account for the abnormality in AVF. In the remaining two patients the ventriculogram was normal in one and not performed in the other.

The five patients from Group H whose ventriculograms showed apical infarctions, the one patient with a normal pattern, and the one patient in whom a ventriculogram was not performed, had severe obstruction in the right or circumflex artery or both (*Table 3*). The arterial narrowing was at least 90% in six patients.

Other groups (*Table 2*) in which a significant number of ventriculograms showed impaired contractility of the inferior wall of the left ventricle were: Groups G (7 of 18), I (3 of 7), J (20 of 53), and K (6 of 13). If patients having generalized impairment are added to those having inferior wall impairment the percentages are: Group G, 50% (9 of 18); I, 57% (4 of 7); J, 42% (22 of 53); and K_2, 62% (8 of 13). If all abnormal ventriculograms are considered the percentages become: Group G_2, 72% (13 of 18); I, 86% (6 of 7); J, 53% (28 of 53); and K_2, 77% (10 of 13). However, some individuals in each group had normal ventriculograms. Finally, if the numbers of patients having severe circumflex or right coronary lesions (*Table 3*) are added to the numbers having inferior or generalized impairment of contrac-

Table 2. Ventriculographic findings

Group	Number	Location of defect in contractility						Ventriculogram	
		Inferior				Apical			
		Only	Apical	A-L	General	Only	A-L	Normal	Not done*
A_1	22	1				1		20	
A_2	1								1
B_1	7	1						6	
B_2	10	1		1	3	1	1	2	1
C	31	6		1	1	3	1	19	
D	9							9	
E_1	2							2	
E_2	24	3		2		4	1	13	1
F_1	20	2			1	2	1	8	6
F_2	43	1		1	3	4	4	22	8
G_1	17	1	1			1	1	11	2
G_2	18	3	4		2	1	3	3	2
H	42	20	4	5	5	3	2	1†	1
I	7	2	1		1	2		1	
J	53	16		4	2	3	3	17	8
K_1	18	3			2			12	1
K_2	13	1	5		2	1	1	3	
L	1				1				

A-L = anterolateral wall of left ventricle.

* Catheterization findings in cases in which ventriculogram was not done: coronary disease—19 (A_2-1, F_1-1, F_2-5, G_1-1, G_2-2, H-1, J-7, K-1); dissecting aneurysm of aorta—2 (E_2 and G_1); aortic stenosis—1 (B_2); mitral insufficiency—1 (F_2); interatrial septal defect—1 (F_1); idiopathic hypertrophic subaortic stenosis—1 (H); coarctation of aorta—1 (F_2); normal—6 (F_1-4, F_2-1, J-1).

† One additional patient had idiopathic hypertrophic subaortic stenosis.

Table 3. Narrowing in right and circumflex arteries in cases having no impairment of inferior contraction or ventriculogram not done

Group	Obstruction in RCA or Cx		Normal arteriogram
	50%+	90%+	
A_1	6	5	9
A_2	1	1	0
B_1*	4	2	4
B_2	6	4	1
C	1	1	16
D	1	1	7
E_1	1	1	1
E_2	5	3	2
F_1	3	2	9
F_2	13	8	12
G_1	8	7	2
G_2†	7	7	0
H‡	7	6	1
I	2	2	1
J§	17	14	12
K_1	9	1	4
K_2	3	1	1
L	0	0	0

* One patient with complete occlusion of left main coronary artery also.
† One patient without coronary arteriography.
‡ One patient having idiopathic hypertrophic subaortic stenosis not included.
§ One patient with 90% narrowing of left main artery.

tility (*Table 4*), the percentages are: Group G_2, 89% (16 of 18); I, 86% (6 of 7); J, 74% (39 of 53); and K_2, 92% (12 of 13).

Localized abnormality in inferior ventricular contractility (*Table 2*) was associated with coronary disease in 89 of 90 patients; the single exception being a patient in Group H who was thought to have traumatic heart disease. Also, in all except one (a patient in Group K_2 who had total obstruction of the anterior descending artery and also had apical infarction) the arterial obstruction involved the circumflex or right coronary artery or both, although the anterior descending artery was also affected in many patients. The obstruction was severe in all and complete or almost complete in 86% (77 of 90).

In one patient (Group F) aortic valvular disease was also present. In 15 of 23 patients who had generalized impairment of contractility the abnormality was associated with coronary disease. Two patients (both in Group H) had severe obstruction of the anterior descending artery only. In addition one of these was thought to have primary myocardial disease, and one had aortic valvular disease. Of the remaining 13 patients, nine had total occlusion of one or more coronary arteries and one had almost total occlusion; the right or circumflex artery was affected in every instance. The other three patients had total occlusion of the anterior descending artery and severe obstruction in both the circumflex and right coronary arteries. The eight patients having generalized impairment of contractility in the absence of coronary disease had aortic valvular disease or primary myocardial disease.

Discussion

The specificity of an electrocardiographic abnormality is as important as its sensitivity. Very few electrocardiographic signs are both sensitive and specific for any anatomic cardiac abnormality. Myers et al[1] correlated the electrocardiogram and postmortem anatomic and arteriographic findings in a large series of patients who had inferior myocardial infarction. Pathologic criteria, based on anatomic findings, for the diagnosis of myocardial infarc-

Table 4. Severe obstruction of right or circumflex coronary arteries and contractile abnormality of inferior wall or generalized impairment

		Inferior		Generalized	
Group	Number	CAD	Remarks	CAD	Other causes
A_1	1	1 (1)			
A_2					
B_1	1	1 (0)			
B_2	5	2 (2)		2 (2)	1 PMD
C	8	7 (4)		1 (1)	
D					
E_1					
E_2	5	5 (3)			
F_1	3	2 (1)	One AS and AI also		1 AS and AI
F_2	5	2 (1)		1 (1)	1 AI and MI 1 PMD
G_1	2	2 (2)			
G_2	9	7 (7)		1 (0)	1 AI†
H	34	28 (25)	One traumatic ventricular aneurysm	5 (2)*	
I	4	3 (3)		1 (0)	
J	22	20 (19)			2 PMD
K_1	5	3 (3)		2 (2)	
K_2	8	6 (6)	On total AD occlusion only	2 (2)	
L	1				1 AS and AI

CAD = coronary artery disease.
Unbracketed numbers in CAD columns indicate number of cases having at least 50% narrowing; bracketed numbers indicate 90% to 100%.
AS = aortic stenosis; AI = aortic insufficiency; MI = mitral insufficiency; PMD = primary myocardial disease; AD = anterior descending artery.
* Includes one case of AS and AI and one of PMD.
† Coronary arteriograms not done; inferior infarct without significant CAD at autopsy.

tion are relatively easy to establish, but there are no criteria based on data obtained by cardiac catheterization. Localized decrease in contractility of an area of the left ventricular wall, as shown by ventriculography, is associated with clinical and electrocardiographic evidence of myocardial infarction in most instances.[3] Ventriculograms may show no definite abnormality when there is clinical and electrocardiographic evidence of myocardial infarction. On the other hand, ventriculograms may show localized areas of impaired contractility without electrocardiographic evidence of myocardial infarction. The size of the infarction, its location, the persistence of some residual contractile tissue, and the viewing angle of the films all affect the ventriculographic diagnosis. Therefore, one would not expect perfect correlation between electrocardiographic abnormalities and ventriculographic aberrations. Severe obstruction or total occlusion of an artery or arteries supplying an area of myocardium does not necessarily indicate that the region is infarcted. However, a normal ventriculogram in such a

patient does not always indicate that infarction has not occurred; infarctions have been found in such patients at subsequent operation or on postmortem examination.

In addition to these problems, apical infarctions may be reflected in lead AVF as well as in precordial leads. Generalized impairment of contractility of the left ventricle is difficult to evaluate in correlative studies. Finally, one would not expect to find normal coronary arteries in a significant percentage of patients thought to have myocardial infarction by a given electrocardiographic standard.

With these limitations in mind, a conservative attitude toward this type of correlative study must be maintained. Q waves more than 0.03 seconds wide in AVF appear to be reliable evidence of inferior infarction in most instances. If the Q wave is just 0.03 seconds wide the diagnosis is less certain. Q waves in the presence of QRS voltages of 0.5 mv or less are of doubtful significance unless the width is greater than 0.03 seconds. Relatively narrow but deep Q waves are not highly specific for myocardial infarction and were frequently encountered in patients who had normal coronary arteriograms and ventriculograms. QS deflections in AVF were found in only a few patients, including one in whom catheterization findings were normal. Minute initial R waves followed by prominent S waves, with a total QRS voltage of more than 0.5 mv, were associated with localized inferior, generalized or apical contractile abnormalities in most ventriculograms, but in some patients these electrocardiographic changes were associated with normal ventriculograms. This finding is an indication for further electrocardiographic or ventriculographic study to exclude the possibility of a strictly posterior infarction.

This investigation shows, as was pointed out by Myers et al,[1] that there is no highly sensitive specific electrocardiographic sign of inferior myocardial infarction. Of the signs that are suggestive, a wide Q wave is the most specific evidence, but it is not a sensitive criterion.

References

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