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Early angiography after myocardial infarction: What have we learned?

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Since the introduction of selective angiography in 1959, by Sones and Shirey,[1] a large body of knowledge of coronary anatomy in living humans has been accumulated. The relationship between angiographic findings and stable or unstable angina pectoris or chronic myocardial infarction is well known. Until recently, because of the potential lethal complications, angiography in acute myocardial infarction (AMI) and early thereafter was not recommended; a waiting period of 2 to 3 months after the acute event was advised.[2] However, angiography early after infarction[3-8] and even in the acute stage of AMI[9-11] has proved to be feasible and safe. Furthermore, acute angiography in evolving myocardial infarction is currently being used for applying fibrinolytic agents into the diseased coronary artery[12-17] or for assessing coronary lesions preceding emergency coronary artery surgical revascularization.[18] The knowledge which emerged from angiography after AMI is considered in this article.

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Transient occlusive obstruction in AMI. Until recently, the severity of coronary artery obstruction in

Table I. Frequency of total occlusion of infarct vessel in relation to time interval after onset of symptoms of AMI

		Time interval after onset of symptoms						
Reference	*Total No. of patients*	*0-6 hr Total occlusion (%)*	*6-24 hr Total occlusion (%)*	*2 wk Total occlusion (%)*	*4 wk Total occlusion (%)*	*6-8 wk Total occlusion (%)*	*4-6 mo Total occlusion (%)*	*7-12 mo Total occlusion (%)*
de Wood et al.[11]	208	86	—	—	—	—	—	—
Ganz et al.[15]	20	90	—	—	—	—	—	—
Rutsch et al.[20]	232	80.5	—	—	—	—	—	—
de Feyter et al.[17]	36	89	—	—	—	—	—	—
Timmis et al.[21]	72	90	—	—	—	—	—	—
Cowley et al.[19]	11	91	—	—	—	—	—	—
de Wood et al.[11]	114	—	67	—	—	—	—	—
Bertrand et al.[3]	106	—	—	53	—	—	—	—
Betriu et al.[8]	259	—	—	—	45	—	—	—
de Feyter et al.[26]	143	—	—	—	—	47	—	—
Pichard et al.[22]	18	—	—	—	—	—	50	46
Pichard et al.[22]	13	—	—	—	—	—	—	—

relation to myocardial infarction in living humans was unknown. Most of our knowledge had been derived from autopsy studies and thus involved a highly selective sample of the population studied. Angiography in the acute stages of myocardial infarction revealed that the rate of a complete coronary artery occlusion progressively declined as the time after onset of symptoms increased (Table I).[3, 7, 8, 11, 15, 19-22] The frequency of a total occlusion within 6 hours after onset of symptoms is 80% to 91%, at 6 to 24 hours 67%, at 2 weeks 53%, at 4 weeks 45%, at 6 to 8 weeks 47%, at 4 to 6 months 50%, and at 7 to 12 months 46%. Although these findings provide no direct evidence for spontaneous recanalization, they strongly suggest that this may occur after infarction. Direct evidence for spontaneous recanalization would require serial angiograms in the same person.

Spontaneous recanalization, probably as a consequence of endogenous thrombolysis, apparently occurs only in the first hours and weeks after AMI and levels off after 2 weeks. Whatever complex pathogenetic processes are involved, the onset of a transmural myocardial infarction is associated with total coronary artery obstruction, which apparently is transient in many patients. From recent experience with intracoronary streptokinase it appears that an occlusive thrombus, which can be dissolved in ±80% of patients, plays a major role in the process.[23] Wackers et al.[24] demonstrated spontaneous improvement in left ventricular ejection fraction within the first 24 hours after transmural AMI. This improvement may be associated with spontaneous recanalization, as has been suggested by Ong et al.[25] They studied 52 patients with AMI not treated with thrombolytic agents. They found that spontaneous recanalization, as evidenced by rapid release of CK-MB, was associated with spontaneous improvement of left ventricular function. de Feijter et al.[26] demonstrated better left ventricular function in patients with spontaneous recanalization than with persisting occlusion of the infarct vessel in patients with a first transmural AMI, who were catheterized 6 to 8 weeks after the acute event. These findings may have an important bearing upon the evaluation of the technique of intracoronary fibrinolytic treatment and stress the necessity of randomized studies.

Extent of coronary artery disease in survivors of AMI. As coronary angiography has not been routinely performed in patients who are asymptomatic after infarction, most studies tend to be biased toward

Table II. Extent of coronary disease in survivors of AMI

Reference	*No. of patients*	*Age (yr)*	*Female (%)*	*Eligible (%)*	*Previous MI (% of total)*	*Catheterization after acute event*
Bertrand et al.[3]	106	50.4 ± 8.6	?	?	?	16.0 ± 3 days
Taylor et al.[6]	106	48.6 ± 9.9	26	39	26	12.5 ± 1.6 days
Turner et al.[5]	92	54 ± 1	13	70	27	22 ± 1 days
Betriu et al.[8]	259	57.3 ± 5	0	91	9	<30 days
de Feyter et al.[7]	179	52 ± 5	10	81	8	<6-8 wk
Gibson et al.[27]	140	51 ± 8	13	72	19	Before discharge
Roubin et al.[28]	229	51	15	89	11	10 days-16 wk

*Significant coronary lesion was defined as an obstruction ≥50% of the luminal diameter in all studies except those of Turner et al.[5] and Roubin et al.[28] (≥70%); left main disease was considered as two-vessel disease (circumflex and left anterior descending).
†0-vessel disease: totally normal or a lesion less than 50% luminal diameter.

Table IV. Prevalence of multivessel disease in survivors of transmural and nontransmural myocardial infarction

	Transmural		*Nontransmural*	
Reference	*No. of patients*	*Multivessel disease %*	*No. of patients*	*Multivessel disease %*
Taylor et al.[6]	64	72	42	76
Turner et al.[5]*	94	76	23	74
de Feyter et al.[7]	143	54	21	48
Roubin et al.[28]	148	37	78	32

*Also included patients with catheterization later than 22 ± 1 days after acute event (18% had angiography after 30 days: mean 64 ± 7).

Table V. Prevalence of multivessel disease in survivors of anterior or inferior myocardial infarction

	Anterior		*Inferior*	
Reference	*No. of patients*	*Multivessel disease %*	*No. of patients*	*Multivessel disease (%)*
Bertrand et al.[3]	45	71	61	79
Taylor et al.[6]	29	59	35	83
de Feyter et al.[7]	62	41	81	73
Roubin et al.[28]	114	31	108	39

Table VI. Prevalence of multivessel disease in survivors of AMI with early postinfarction angina pectoris (within 8 weeks after acute event)

	Postinfarction angina		*Postinfarction angina absent*	
Reference	*No. of patients*	*Multivessel disease %*	*No. of patients*	*Multivessel disease %*
Turner et al.[5]*	26	92	91	70
de Feyter et al.[7]	52	79	127	45

*Included are patients with catheterization later than 22 ± 1 days after the acute event (18% had angiography after 30 days: 64 ± 7).

high-risk patients. Thus, much of our knowledge of coronary anatomy is derived from referred symptomatic patients. In this article we present only data from those prospective studies[2, 5-7, 27, 28] in which patient intake was not biased toward symptomatic postinfarct patients or complicated transfers from other hospitals, in an attempt to simulate the patient population with AMI seen in a community hospital. The study of Madigan et al.[29] was rejected because of its apparent bias toward high-risk

*Extent coronary artery disease**			
0-vessel† (%)	*1-vessel* (%)	*2-vessel* (%)	*3-vessel* (%)
1	23	45	32
0	26	21	53
2	25	32	30
7	34	33	25
2	43	41	14
3	39	36	22
6	58	26	10

Table III. Prevalence of left main stem coronary artery disease in survivors of myocardial infarction

References	*Total No. of patients*	*Left main disease* (%)
Taylor et al.[6]	106	11
Turner et al.[5]	92	11
Betriu et al.[8]	259	1
de Feyter et al.[7]	179	2
Gibson et al.[27]	140	2
Roubin et al.[28]	229	1

patients. In their study of 50 survivors of subendocardial infarction, 60% had multivessel disease, but 80% were catheterized for angina, which was unstable in 66%.

Only a few prospective angiographic studies after AMI have been performed, which give insight into the overall spectrum of coronary lesions associated with the infarction. The results of these studies vary, depending mainly on the selection criteria and the eligibility. In Table II the extent of coronary artery disease is presented in survivors of AMI. All patients were less than 70 years of age. Coronary angiography was performed 4 to 8 weeks after the acute event. Normal vessels or minimal lesions were present in 0% to 7%, one-vessel disease was present in 23% to 58%, two-vessel disease in 21% to 45%, and three-vessel disease in 10% to 53%. The prevalence of left main stem artery disease varied from 1% to 11% (Table III). The higher prevalence of left main disease (11%) in the studies of Turner et al.[5] and Taylor et al.[6] compared to the other studies[7, 8, 27, 28] may be caused by patient selection. Both studies had a higher prevalence of previous AMI (which is associated with a higher prevalence of multivessel disease) and a lower percentage of eligibility. The prevalence of multivessel disease did not differ in patients with a transmural or nontransmural AMI (Table IV). In transmural AMI multivessel disease was present in 37% to 76% and in nontransmural AMI this was 32% to 76%. The prevalence of multivessel disease is significantly ($p < 0.001$, Mantel-Hoenszel test) higher in survivors of a transmural inferior wall AMI than in survivors of a transmural anterior wall AMI (Table V). The prevalence of multivessel disease is significantly ($p < 0.001$) higher in patients with early postinfarction angina pectoris (79% to 92%) than in those without angina pectoris (45% to 70%) (Table VI). Finally, in patients with a previous AMI the presence of multivessel disease was higher (73% to 100%) than in patients with a first infarction (31% to 64%) (Table VII).

Left ventricular dysfunction in survivors of AMI. What is the extent of left ventricular damage caused by AMI in a nonselected population? An ejection fraction less than 30% was present in 7% to 17% of the patients, an ejection fraction of 30% to 49% was found in 44% to 55%, and an ejection fraction of 50% or more was present in 31% to 48% (Table VIII). Similar data were found with radionuclide determined ejection fractions.[27]

The ejection fraction was significantly lower in patients with multivessel disease than in those with single-vessel disease.[7, 8] In patients with a total occlusion of the infarct vessel the ejection fraction was lower and the impairment of left ventricular wall motion was higher than in those with a lesser degree of obstruction of the infarct vessel.[8, 26] Left ventricular damage was more severe with anterior wall myocardial infarction than with inferior wall myocardial infarction.[6-8, 26] A severely depressed left ventricular function was found in patients with previous myocardial infarction. Apparently, a well-developed collateral circulation improved the ejection fraction and decreased the size of akinetic segments.[8, 30]

Prognostic value of angiography and ventriculography soon after AMI. In patients younger than 65 years, the value of angiography and ventriculography as determinants of prognosis after infarction has been investigated.[6, 7, 28, 31] Sanz et al.[31] found that the ejection fraction and the number of diseased vessels were the only independent invasive predictors of survival during a follow-up study of 60 months. Patients with a normal ejection fraction, regardless of the number of diseased vessels, lived longest. The probability of survival of patients with an ejection fraction of 21% to 49% ranged from 78% for patients with three-vessel disease to 95% for those with single-vessel disease. The poorest prognosis

Table VII. Prevalence of multivessel disease in patients with and without previous myocardial infarction

	Previous myocardial infarction			
	Present		Absent	
Reference	No. of patients	Multivessel disease (%)	No. of patients	Multivessel disease (%)
Taylor et al.[6]	28	100	78	64
Betriu et al.[8]	24	91	235	55
de Feyter et al.[7]	15	73	164	53
Roubin et al.[28]	24	75	202	31

Table VIII. Left ventricular function in survivors of AMI

		Ejection fraction		
Reference	No. of patients	<30% % of total	30-50% % of total	>50% % of total
Taylor et al.[6]	106	7	44	48
Betriu et al.[8]	259	17	47	36
de Feyter et al.[7]	179	14	55	31
Roubin et al.[28]	229	—	58*	42

*Ejection fraction <49%.

corresponded to an ejection fraction less than 20%: 30% to 75% depending on the number of diseased vessels. According to the 30-month follow-up study of Taylor et al.,[6] univariate analysis showed that low ejection fraction, proximal left anterior descending coronary artery disease, and three-vessel disease were associated with a high risk of sudden cardiac death. However, multivariate analysis of 30 clinical and laboratory variables identified previous myocardial infarction and an ejection fraction less than 40% as predictors of death. Additional information was not provided by the other variables once these two variables were considered. According to de Feyter et al.,[7] patients with an ejection fraction less than 30% and three-vessel disease formed a high-risk group for cardiac death. During a mean follow-up of 28 months, 10 of the 11 cardiac deaths occurred in this high-risk group.

Roubin et al.[28] showed that three-vessel disease had a lower survival rate at 1 year than two- and one-vessel disease. Also, although not at a significant level, an ejection fraction lower than 50% was associated with a lower 1-year survival rate. Thus, it appears that cardiac death in survivors of AMI is related to the extent of coronary artery disease and the severity of left ventricular dysfunction.

Angiography in all survivors of AMI? Cardiac catheterization can be performed relatively safely early after a sustained myocardial infarction. Of a total of 486 patients who underwent angiography, there were no procedure-related deaths and major complications occurred in 1% to 3%.[3,5-7] Should we then proceed to perform coronary angiography before discharge in all patients after myocardial infarction in an attempt to guide therapy to assess prognosis?

Certainly, patients recovering from AMI, who are symptomatic, should be appropriately evaluated; this may include coronary angiography and left ventriculography. However, is there a need for angiography in asymptomatic postinfarction patients? Two recent studies[32,33] have shown that surgery is not superior to medical treatment for improving survival rates in asymptomatic postinfarction patients. On the other hand, to assess prognosis and to identify high-risk patients clinical variables or noninvasive tests such as exercise ECGs and radionuclide studies appear to be appropriate and only those with evidence of inducible ischemia should undergo cardiac catheterization.[34] Therefore, coronary angiography is reserved only for symptomatic patients not responding adequately to optimal medical treatment in order to assess operability.

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