Clinical characteristics of and prognosis in acute transmural anterior, transmural inferior and non-transmural myocardial infarction

A comparative retrospective study

C. J. VAN RENSBURG, J. Z. PRZYBOJEWSKI, J. SOOLMAN

Summary

This retrospective study was undertaken to determine whether there was any difference in the clinical characteristics of and prognosis in white patients admitted to the Intensive Coronary Care Unit (ICCU) at Tygerberg Hospital with acute non-transmural, transmural anterior and transmural inferior myocardial infarction (MI). The three groups were carefully matched, taking into consideration the possible influence of previous MI and congestive cardiac failure (CCF). There were 187 patients with non-transmural MI, and 176 with transmural anterior and 209 with transmural inferior MI. Patients with acute transmural anterior MI had the worst prognosis while at the ICCU, at 3 months' follow-up and at long-term follow-up (mean 22,2 months). This group had the greatest frequency of CCF, cardiogenic shock, acute pericarditis, ventricular premature beats, ventricular tachycardia, left anterior hemiblock and complete left bundle-branch block and the highest mortality. Acute transmural inferior MI was responsible for the highest frequency of ventricular fibrillation in the ICCU and had a worse prognosis than non-transmural MI. Acute non-transmural MI resulted in the highest incidence of early and late myocardial re-infarction; although death in the ICCU was least frequent, mortality among this group had increased dramatically by 3 months' follow-up. Hence, acute non-transmural MI is not benign and an unstable period exists for 3 months thereafter. Because of this, more aggressive diagnostic measures should be instituted during this period in order possibly to improve prognosis in this group. It would appear that this is the first such study undertaken in South Africa.

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Cardiac Clinic and Department of Internal Medicine, University of Stellenbosch and Tygerberg Hospital, and Institute for Biostatistics of the South African Medical Research Council, Parowvallei, CP
C. J. VAN RENSBURG, M.B. CH.B.
J. Z. PRZYBOJEWSKI, M.B. CH.B., F.C.P. (S.A.), F.I.C.A., F.A.C.C., F.C.C.P.
J. SOOLMAN, B.SC. HONS

The cardiological approach to the patient with an acute myocardial infarction (MI) has changed quite drastically over the last few years in that more 'aggressive' lines of therapy have been followed. Early interventions such as intracoronary thrombolysis with streptokinase infusion,[1,2] percutaneous transluminal coronary angioplasty (PTCA) immediately after intracoronary thrombolysis,[3,4] and aortosaphenous vein bypass grafting (CABG) soon after an acute MI,[5] are all designed to reduce myocardial 'infarct size' and thus improve short- and long-term morbidity and mortality. Early stress tests after acute MI are also advocated to detect the patient 'at risk' of a further acute MI or sudden death,[6-12] thus prompting early coronary angiography with a view to carrying out PTCA or CABG. Davies *et al.*[13] have recently shown that elevation of the creatine kinase iso-enzyme (CK-MB) level 24 hours after graded exercise stress testing probably indicates reversible myocardial ischaemia and could assist in deciding on the management of these patients.

There is still much controversy about acute 'non-transmural' MI ('ST infarction', 'non-Q-wave infarction', 'subendocardial infarction') and acute 'transmural' MI ('Q-wave infarction'). For a long time non-transmural MI was looked upon as 'mild' compared with transmural MI.[14-17] Most of the studies carried out in an attempt to clarify this controversy have concerned small series of patients and the number of patients in the transmural MI group was usually much greater than of those with a non-transmural MI. Another important controversial aspect is the inclusion of patients who have previously had an MI in these studies.

In order to establish the short- and long-term outcome in patients with acute non-transmural ('subendocardial') MI and the possible mechanisms influencing it, 187 patients with acute non-transmural MI were compared with 176 patients with an acute transmural anterior MI and a further 209 patients with an acute transmural inferior MI. It was hoped that this information would assist in selecting those patients at particularly high risk of complications and thus promote earlier intervention. The present study is one of the few to utilize statistically matched patient groups, and may be the first in South Africa.

Patients and methods

The hospital records of 4 000 consecutive white patients admitted to the intensive coronary care unit (ICCU) at Tygerberg Hospital during the period October 1978 - October 1980 were analysed. Out of this group a total of 572 patients who fulfilled the criteria for either an acute non-transmural or an acute transmural MI were selected.

The diagnosis of acute non-transmural MI was established on the basis of the history, an abnormal elevation in cardiac

enzyme levels (including CK-MB), and a positive ECG (as exemplified by persistent ischaemic T waves and/or persistent ST-segment depression or elevation which lasted more than 48 hours, and on which no Q waves were evident). All three criteria had to be fulfilled. For an acute transmural MI the first two criteria had to be present in addition to the evolution of pathological Q waves (0,04 second in duration, with a depth of at least 25% of the height of the R wave in the same QRS complex).

All patients were monitored in the ICCU. The clinical notes from the ICCU and the Cardiac Clinic follow-up folders were reviewed up to and including February 1983. All surviving patients were followed up for not less than 3 months. Twenty-three of the 572 patients were lost to long-term follow-up.

Matching characteristics

The matching clinical characteristics of the three groups of patients diagnosed as having either an acute non-transmural MI or an acute transmural anterior or inferior MI are detailed in Table I. The ratio of males to females in each group was not statistically significant, but the number of patients in each group who had suffered a previous MI proved to be statistically significant ($P = 0{,}0002$). There was also a statistically significant difference in the frequency of previous congestive cardiac failure (CCF) in each of the three groups ($P = 0{,}0101$). Because of these findings, previous MI and past CCF had to be taken into consideration in matching the three groups. In order to simplify statistical analysis, previous MI and prior CCF were combined. If either or both of these were present then 'previous heart disease' was documented.

TABLE I. MATCHING CHARACTERISTICS OF THE THREE GROUPS

	Types of MI					
	Non-transmural		Transmural anterior		Transmural inferior	
	No.	%	No.	%	No.	%
Men	**138**	**31,7**	**128**	**29,4**	**169**	**38,9**
Women	**49**	**35,8**	**48**	**35,0**	**40**	**29,2**
Prior MI	**88**	**41,9**	**65**	**31,0**	**57**	**27,1**
Prior CCF	**29**	**34,9**	**35**	**42,2**	**19**	**22,9**
Mean age (yrs)	**59,09**		**59,58**		**56,22**	

Percentages given are of the total number of patients in the category listed in the column on the left.
MI v. sex: $\chi^2 = 4{,}244$, df = 2, $P = 0{,}1198$; not significant.
MI v. prior MI: $\chi^2 = 16{,}635$, df = 2, $P = 0{,}0002$; significant.
MI v prior CCF: $\chi^2 = 9{,}199$, df = 2, $P = 0{,}0101$; significant.

Statistical analysis

Two interrelated statistical methods were employed: chi-square tests and log-linear models were applied to the data and combined in a series of frequency tables (three-way). Each factor was examined individually and a series of three-way frequency tables was established for MI versus previous heart disease versus the factor under consideration. The interactions between the three varying factors were then examined by the abovementioned statistical methods. Since simultaneous test procedures were utilized to determine the interaction between factors in a frequency table, the significance level of each individual test had to be controlled in order that overall significance could be maintained at an acceptable figure. The overall level of significance for each table was 0,16 (16%), and each individual test was carried out at the 2,5% level of significance. The data from the chi-square method are presented under 'Results'; the log-linear model was applied to the frequency tables. Any difference in the data obtained by using these two statistical methods is mentioned in the text.

Results

The incidence of precipitating factors (such as emotional stress, physical exercise, sexual activity and consumption of large meals) was evaluated (Table II), but no statistically significant difference could be shown between the three groups ($P = 0{,}9474$). In approximately 59% of the acute non-transmural MI group and 60% of the patients in the other two groups, precipitating factors were noted. Unstable angina during the week prior to the acute MI was equally common in the three groups ($P = 0{,}7727$).

Course in hospital

Arrhythmias and conduction disturbances

The incidences of various types of cardiac arrhythmias and conduction disturbances are indicated in Table III. There was no statistically significant difference in the frequency of sinus bradycardia ($P = 0{,}6997$) and active atrial arrhythmia ($P = 0{,}2735$); these complications occurred in less than 20% of each group. The incidence of ventricular premature beats and ventricular tachycardia was considerably less in the acute non-transmural MI group than in the group with acute transmural MI. Ventricular fibrillation was equally frequent in all groups ($P = 0{,}1329$). There was a statistically significant difference in the frequency of atrioventricular block (AVB); either Mobitz I or Mobitz II second-degree AVB and third-degree AVB or complete heart block (CHB) were commonest in patients with acute transmural inferior MI. CHB was detected in 23 patients (11%) with a transmural inferior MI and was the most common

TABLE II. PRECIPITATING FACTORS AND UNSTABLE ANGINA

	Non-transmural MI		Transmural anterior MI		Transmural inferior MI		
	No.	%	No.	%	No.	%	Total
Precipitating factors*							
No	**77**	**41,18**	**70**	**39,77**	**83**	**39,71**	**230**
Yes	**110**	**58,82**	**106**	**60,23**	**126**	**60,29**	**342**
Total	**187**		**176**		**209**		**572**
Unstable angina**							
No	**88**	**47,06**	**89**	**50,29**	**105**	**50,24**	**282**
Yes	**99**	**52,94**	**87**	**49,71**	**104**	**49,76**	**290**
Total	**187**		**176**		**209**		**572**

*$\chi^2 = 0{,}108$, df = 2, $P = 0{,}9474$; not significant.
**$\chi^2 = 0{,}516$, df = 2, $P = 0{,}7727$; not significant.

TABLE III. DISTURBANCES OF CARDIAC RHYTHM AND CONDUCTION

	Types of MI							
	Non-transmural (*N* = 187)		Transmural ant. (*N* = 176)		Transmural inf. (*N* = 209)		*P* value	Significance
	No.	%	No.	%	No.	%		
Sinus bradycardia	**28**	**14,97**	**32**	**18,18**	**36**	**17,22**	**0,6997**	**NS**
Active atrial arrhythmia	**32**	**17,11**	**23**	**13,07**	**40**	**19,14**	**0,2735**	**NS**
Ventricular premature beats	**63**	**33,69**	**89**	**50,57**	**93**	**44,50**	**0,0043**	**Sig.**
Ventricular tachycardia	**8**	**4,28**	**23**	**13,07**	**26**	**12,44**	**0,0065**	**Sig.**
Ventricular fibrillation	**9**	**4,81**	**16**	**9,09**	**21**	**10,05**	**0,1329**	**NS**
AVB							**0,0001**	**Sig.**
Mobitz I	**3**	**1,60**	**3**	**1,70**	**7**	**3,35**		
Mobitz II	**1**	**0,53**	**2**	**1,14**	**7**	**3,35**		
CHB	**1**	**0,53**	**10**	**5,68**	**23**	**11,00**		
LAHB	**14**	**7,49**	**39**	**22,16**	**20**	**9,57**	**0,0000**	**Sig.**
RBBB	**17**	**9,09**	**21**	**11,93**	**26**	**12,44**	**0,5338**	**NS**
LBBB	**3**	**1,60**	**16**	**9,09**	**5**	**2,39**	**0,0005**	**Sig.**

NS = not significant; Sig. = significant.

form of heart block. All forms of heart block were rare in patients with acute non-transmural MI. Only 1 patient developed Mobitz II second-degree AVB, and in a further patient CHB was registered, whereas only 3 patients developed Mobitz I second-degree AVB. In the acute transmural anterior MI group, CHB was the commonest form of heart block (10 cases), while in only 3 and 2 patients respectively was a Mobitz I or Mobitz II second-degree AVB documented. Disturbances in intraventricular conduction (incomplete and complete right bundle-branch block (RBBB) or left bundle-branch block (LBBB); fascicular heart block) were equally common in the acute non-transmural MI and acute transmural inferior MI groups, but statistically more frequent in the acute transmural anterior MI group. The relatively high occurrence of left anterior hemiblock (LAHB) of 22% and complete LBBB of 9% in the latter group mainly accounted for these conduction defects ($P = 0{,}0000$ and $P = 0{,}0005$ respectively). There was also significant statistical interaction between complete LBBB and 'previous heart disease'.

Complications in the ICCU

The complications occurring in the ICCU are shown in Table IV. The diagnosis of CCF was based on the presence of a third heart sound, basal crepitations and radiological signs of pulmonary congestion. The highest incidence of CCF was 51%, in patients with an acute transmural anterior MI; the corresponding figures for acute non-transmural MI and acute transmural inferior MI were 24% and 39% respectively. There was also a significant correlation between CCF and 'previous heart disease' Hypotension (systolic blood pressure < 90 mmHg in the presence of sinus rhythm) followed a similar frequency pattern. The frequency of pericarditis (pericardial friction rub with or without associated pericarditis pain and ECG changes) was not significantly different in the groups (provided that 'previous heart disease' was taken into consideration). The duration of ischaemic myocardial pain in the ICCU was also not statistically different.

Mortality

Mortality due to cardiac causes in the ICCU and during the first 3 months is given in Table V. It was significantly less frequent in the ICCU in the acute non-transmural MI group (4,3%) than in the acute transmural anterior (22,2%) and acute transmural inferior (13,9%) MI groups ($P = 0{,}0000$). There

TABLE V. MORTALITY WITHIN THE FIRST 3 MONTHS

	Types of MI					
	Non-transmural (*N* = 187)		Transmural ant. (*N* = 176)		Transmural inf. (*N* = 209)	
	No.	%	No.	%	No.	%
ICCU	**8**	**4,3**	**39**	**22,2**	**29**	**13,9**
Follow-up	**17**	**9,1**	**47**	**26,7**	**36**	**17,2**

$\chi^2 = 19{,}903$; df = 2; $P = 0{,}0000$

TABLE IV. CLINICAL EVENTS OCCURRING WHILE IN THE ICCU

	Types of MI				
	Non-transmural (*N* = 187)	Transmural ant. (*N* = 176)	Transmural inf. (*N* = 209)	*P* value	Significance
CCF	**45 (24,06%)**	**90 (51,14%)**	**81 (38,76%)**	**0,0000**	**Sig.**
Hypotension	**16 (8,56%)**	**54 (30,68%)**	**55 (26,32%)**	**0,0000**	**Sig.**
Pericarditis	**4 (2,14%)**	**17 (9,66%)**	**14 (6,70%)**	**0,0105**	**Sig.**
Mean duration of pain (d)	**1,717**	**1,966**	**1,813**	**0,0349**	**NS**
Mean duration of stay in the ICCU (d)	**4,834**	**5,051**	**4,962**	**0,4682**	**NS**

Sig. = significant; NS = not significant.

was also a significant relationship between 'previous heart disease' and death within the ICCU. During the 3-month follow-up period the mortality in the non-transmural MI group rose to a greater extent than in the other two groups; the difference between the three groups was still highly significant ($P = 0{,}0000$).

Course after discharge

Clinical condition

Table VI shows the clinical and functional features of the patients over a mean follow-up period of 22,2 months after discharge from hospital. Differences as regards angina or its class during follow-up were not significant; this symptom was exceptionally common, occurring in over 60% of all patients, and furthermore there was a significant correlation between angina pectoris and 'previous heart disease'. Syncope and CCF were equally frequent in the three groups, but there was still a significant correlation between 'previous heart disease' and CCF after discharge from the ICCU. There was no statistically significant difference as regards return to full-time employment (Table VI).

TABLE VI. LATE CLINICAL FEATURES

	Types of MI					
	Non-transmural ($N = 170$)		Transmural ant. ($N = 129$)		Transmural inf. ($N = 173$)	
	No.	%	No.	%	No.	%
Angina						
None	**60**	**35,3**	**48**	**37,2**	**61**	**35,3**
Class I - II	**69**	**40,6**	**48**	**37,2**	**70**	**40,5**
Class III - IV	**41**	**24,1**	**33**	**25,6**	**42**	**24,3**
CCF	**17**	**10**	**10**	**7,8**	**11**	**6,4**
Syncope	**47**	**27,6**	**43**	**33,3**	**38**	**22,0**
Working						
Full-time	**57**	**33,5**	**41**	**31,8**	**74**	**42,8**
Part-time	**14**	**8,2**	**11**	**8,5**	**13**	**7,5**
Not working	**99**	**58,2**	**77**	**59,7**	**86**	**49,7**

Recurrent MI

The incidence of further acute MI occurring during the follow-up period is indicated in Table VII. Differences between the three groups were statistically significant; the incidence of evidence of a new MI, or extension of a previous MI, was 19,4% in non-transmural cases (11,8% were shown by ECG to be transmural anterior in type), 17,1% in the transmural anterior MI group (14,0% again transmural anterior), and only 13,9% in the transmural inferior MI group. There was a statistically insignificant difference in the incidence of recurrent acute MI in patients with previously documented transmural or non-transmural MI with or without previous MI.

Patient survival and causes of death

Table VIII shows that the survival rate was greater in non-transmural MI and transmural inferior MI than in transmural anterior MI. There were also statistically significant differences as regards the cause of death ($P = 0{,}0280$); in the transmural anterior MI group 88,3% of deaths were directly due to cardiac causes. A statistically significant relationship between 'previous heart disease' and death due to cardiac causes was demonstrated in all groups.

Discussion

ECG classification of acute MI type

It is most important to appreciate that this study was of a clinical nature, excluding any form of autopsy. The type of acute MI was categorized by clinically acceptable criteria and

TABLE VII. REINFARCTION IN VARIOUS PATIENT GROUPS

	Type of MI					
	Non-transmural		Transmural anterior		Transmural inferior	
Follow-up acute MI	No.	%	No.	%	No.	%
None	**137**	**80,6**	**107**	**82,9**	**149**	**86,1**
Non-transmural	**7**	**4,1**	**1**	**0,8**	**2**	**1,2**
Transmural anterior	**20**	**11,8**	**18**	**14,0**	**10**	**5,8**
Transmural inferior	**6**	**3,5**	**3**	**2,3**	**12**	**6,9**
	170		**129**		**173**	

$\chi^2 = 14{,}935$; df = 6; $P = 0{,}0208$ — significant

TABLE VIII. CAUSE OF DEATH IN VARIOUS PATIENT GROUPS*

	Type of MI						
	Non-transmural		Transmural anterior		Transmural inferior		
Cause of death	No.	%	No.	%	No.	%	Total
MI during follow-up	**9**	**32,1**	**5**	**8,3**	**8**	**19,5**	**22**
Cardiac (excluding above)	**16**	**57,1**	**53**	**88,3**	**30**	**73,2**	**99**
Non-cardiac	**3**	**10,7**	**2**	**3,3**	**3**	**7,3**	**8**
Total	**28**		**60**		**41**		**129**

$\chi^2 = 10{,}877$; df = 4; $P = 0{,}0280$ — significant

*Mean follow-up period 22,2 months.

ECG changes recorded. The clinician is limited to the ECG characteristics in establishing a diagnosis of acute 'non-transmural' and 'transmural' MI, and limitations exist in this type of clinical classification. Spodick[18] has written a most elegant description detailing these limitations, and the accuracy of clinical categorization has been seriously questioned in various autopsy studies. 'Pathological' Q waves have always been said to define an acute 'transmural' MI, but both Horan *et al.*[19] and Abbott and Scheinman[20] have demonstrated that this type of acute MI can occur in the absence of Q waves, while abnormal Q waves, in addition to classic ST-T-wave changes, have been noted in patients with autopsy-proven acute 'non-transmural' MI.[21–23] Further confusion regarding the ECG classification of acute MI type has been contributed to by the so-called 'non-diagnostic' ECG.[24] In the absence of more refined clinical diagnostic criteria, the present ECG classification should probably still be employed in studying large series of patients who survive.

Prevalence of acute non-transmural MI

A review of the literature clearly indicates that assessment of the prevalence of acute non-transmural MI is made rather difficult by the fact that some series (both prospective and retrospective) include patients who have had a previous MI. Furthermore, some studies exclude females while others exclude patients $\geqslant$ 60 years of age. In our study, which included patients with a previous acute MI, this prevalence was 31,7% among men and 35,8% among women (Table I). Patients with acute non-transmural MI, 41,9% had suffered a previous MI, as against 31,0% and 27,1% respectively with transmural anterior and inferior MI.

The prevalence of previous MI is similar to those of 41% documented by Schultze *et al.*[25] and 46% by Hutter *et al.*[26] Lower figures have been recorded by Marmor *et al.*[27] (20%), Madigan *et al.*[28] (24%), Cannom *et al.*[29] (32%), and Szklo *et al.*[30] (34%). Others have analysed series of first acute non-transmural MIs thus excluding previous MI.[31–34] Coll *et al.*[32] found a 6% prevalence of non-transmural MI, similar to the 4% reported by Mahony *et al.*,[31] but Krone *et al.*[33] documented a higher prevalence of 15,8%. This marked variation in prevalence may be partly explained by the fact that a previous MI could prevent the appearance of new pathological Q waves and thus give a falsely high prevalence of acute non-transmural MI.[35,36]

CCF

The incidence of CCF appearing in the three groups while patients were still in the ICCU was statistically significantly different (Table IV). The Killip classification[37] of CCF complicating acute MI was not employed in this study. The finding that CCF was commonest (51,14%) in patients with acute transmural anterior MI was not surprising since CCF is logically related to the exent of myocardial damage. Infarct size is well known to be related to left ventricular ejection fraction and the complication of CCF. No attempt was made to determine infarct size by assessment of parameters such as cardiac serum enzyme levels. This study also demonstrated a significant correlation between CCF and previous heart disease (previous MI, previous CCF, or both), again emphasizing the importance of extent of myocardial damage.

Hutter *et al.*[26] also demonstrated an increased frequency of CCF in the acute phase in patients admitted with an acute transmural anterior MI (55%; $P < 0,02$). These workers noted a comparable frequency of CCF in transmural inferior and non-transmural MI, 37% and 25% respectively — figures very close to ours. Thanavaro *et al.*[38] found a higher prevalence of CCF in acute transmural MI (not differentiating anterior from inferior types) than in acute non-transmural MI (54% and 49% respectively), but this difference was not statistically significant. Sanz *et al.*,[39] in a unique prospective study of 259 men with acute transmural (Q wave) MI, demonstrated that severe CCF (Killip class III and IV) detected during the acute phase in the ICCU was an independent predictor of late mortality, the others being ejection fraction (the best predictor) and the number of diseased coronary arteries. Coll *et al.*[32] clearly showed that severe CCF during the early phase of MI was one of the most important determinants of long-term survival. Nicholson *et al.*[34] reported that of 86 patients with acute non-transmural (non-Q-wave) MI, 77 (90%) were in Killip class I (no CCF), 6 were in class II (mild-to-moderate CCF) and the remaining 3 were in class III (severe CCF). Moss *et al.*[40] and Moss,[41] in the recent Multicenter Postinfarction Risk Stratification Study, found that the degree of CCF in the ICCU was directly proportional to the 1-year cardiac mortality rate; with no CCF the 1-year mortality rate was only 3%, rising to as high as 38% in the presence of CCF. Sniderman *et al.*[42] emphasized the importance of the mass of myocardial necrosis, as well as the amount of potentially ischaemic myocardium, which is directly related to the occurrence of CCF secondary to 'left ventricular dysfunction', others[39,43–49] have substantiated these claims. Thompson *et al.*[50] demonstrated that CCF was more frequent with higher serum CK levels in patients who died after discharge, thus confirming the importance of 'infarct size' in determining short-term mortality. Sniderman *et al.*[42] emphasized the fact that 'patients in heart failure have not only an increased risk of arrhythmia, but their cardiac reserve may be so diminished as to make any additional ischaemia intolerable'.

CCF after discharge from the ICCU and during follow-up developed with equal frequency in the three groups (Table VI), but was far less than the frequency in the acute phase of observation in the ICCU, when it varied between 24,06% and 51,14% (Table IV). This finding suggests that reversible myocardial ischaemia leading to CCF played an important role in the acute phase. CCF during the follow-up period again correlated with 'previous heart disease'. Our data are at great variance with those of Hutter *et al.*,[26] who had much higher CCF rates in their three groups on follow-up, ranging from 28% (transmural inferior MI) to 37% (non-transmural MI), and approaching the frequency of CCF in the ICCU phase. Sanz *et al.*[39] noted a follow-up CCF frequency rate of 17% in their 259 patients with transmural MI, a frequency close to ours. Coll *et al.*[32] documented CCF during follow-up in 46 (10,6%) of those with an initial transmural MI but none in 28 patients with non-transmural MI. Cannom *et al.*[29] found a follow-up frequency of CCF of 47,6% in transmural MI and 39% in non-transmural MI, the difference not being statistically significant. Thus all the above studies show a trend to a higher incidence of CCF in both phases in patients with transmural MI, with a tendency for a decrease during the follow-up period.

Hypotension and cardiogenic shock

This complication occurred statistically significantly more commonly in transmural anterior MI (30,68%) than in acute transmural inferior (26,32%) and non-transmural (8,56%) MI (Table IV). Patients with this outcome are considered to be in Killip class IV,[40] so that the pattern is similar to that for CCF. Hutter *et al.*[26] found an almost identical frequency rate in transmural anterior MI (29%), with equal rates of 19% in both transmural inferior and non-transmural MI. However, the difference was not statistically significant in their study. In contrast, Thanavaro *et al.*[38] found a much lower frequency of cardiogenic shock — 7% in transmural MI and 3% in non-

transmural MI. They showed that this complication was determined by the peak serum glutamine oxalo-acetic transaminase level rather than the type of MI. Scheinman and Abbot[51] reported cardiogenic shock in 18% of transmural MI patients, as against 22% and 3% in patients with non-transmural MI and high and low serum enzyme levels respectively. Madias *et al.*[52] found no difference in the frequency of fatal cardiogenic shock in their groups of transmural and non-transmural MI patients, apart from the fact that those with non-transmural infarction had evidence of previous extensive MI. Poehlman and Silverman[53] encountered this complication in 4% of cases of non-transmural MI, Rigo *et al.*[54] in 6% of patients with both acute transmural and non-transmural MI, Szklo *et al.*[30] in 25,1% and 23,0% of patients with transmural and non-transmural MI, Madigan *et al.*[28] in none of 50 consecutive patients with acute non-transmural MI, and Cannom *et al.*[29] in 8,1% and 2,5% of patients with transmural and non-transmural MI respectively. In their prospective study of 86 patients with acute non-transmural MI, Nicholson *et al.*[34] recorded none. These marked variations may well be due to patient selection, particularly with regard to previous MI. The inaccuracy of the ECG in distinguishing between non-transmural and transmural infarction probably also has an important bearing on the frequency of cardiogenic shock.

Acute pericarditis

This complication has classically been described as only complicating transmural MI. Our study showed the greatest incidence of pericarditis in acute transmural MI (9,66%), followed by transmural inferior (6,70%) and non-transmural (2,14%) MI (Table IV). This difference only reached statistical significance ($P = 0{,}0105$) if 'previous heart disease' was excluded. Hutter *et al.*[26] found a 19% frequency of acute pericarditis in acute transmural inferior MI, followed by 18% and 6% in transmural and non-transmural MI respectively. This difference was not statistically significant. The occurrence in non-transmural MI can probably be explained by the fact that absence of Q waves does not necessarily mean that transmural infarction has not taken place.

Cardiac rhythm disturbances

Supraventricular arrhythmias were encountered in some 13 - 20% of patients, with no statistically significant differences between the three groups (Table III), but this was not the experience with ventricular arrhythmias. The controversial ventricular premature beats (VPBs) appeared most frequently (50,57%) in cases of transmural anterior MI, with lower rates of 44,50% and 33,69% in transmural inferior and non-transmural MI respectively. This difference was statistically significant ($P = 0{,}0043$). The incidence of ventricular tachycardia in the ICCU also showed a statistically significant difference between the groups; the highest figure (13,07%) was for transmural anterior MI, followed by 12,44% in acute transmural inferior and 4,28% in non-transmural MI ($P = 0{,}0065$). Ventricular fibrillation was slightly less frequent: 10,05% in transmural inferior MI, 9,09% in transmural anterior and 4,81% in non-transmural MI. However, these differences were not statistically significant ($P = 0{,}1329$).

Comparison with the study of Hutter *et al.*[26] highlights several important differences. Firstly, they found a statistically significant difference in the frequencies of supraventricular arrhythmias (sinus bradycardia; active atrial arrhythmia) in their three groups. Sinus bradycardia was seen most often as a complication of transmural inferior MI (25%), compared with transmural anterior (9%) and non-transmural MI (12%) ($P < 0{,}025$). Active atrial arrhythmias, such as acute atrial fibrillation, were encountered in 38% of patients with transmural inferior MI and 21% in both transmural anterior and non-transmural MI patients ($P < 0{,}05$). Exactly the opposite experience was recorded for ventricular arrhythmias, no statistically significant difference being found. VPBs were seen in 59% of transmural anterior MI, 46% of non-transmural MI, and 41% of acute transmural inferior MI patients. Ventricular tachycardia was commonest in transmural anterior infarction (30%), with lower rates in transmural inferior (25%) and non-transmural MI (19%). Ventricular fibrillation was encountered in 8% of transmural inferior infarctions and 6% of both transmural anterior and non-transmural MIs.

In contrast, Thanavaro *et al.*[38] had a statistically significant higher frequency of both supraventricular and ventricular arrhythmias in transmural MI than in non-transmural cases, although this significance diminished drastically when the groups were further categorized according to serum glutamic oxalo-acetic transaminase levels. This finding strongly suggested that this frequency was closely related to 'infarct size'. These authors wisely stated that 'the presence or absence of abnormal Q waves may be only a convenient way of indicating the extent of myocardial damage and may not reflect the true pathologic type of infarction', sentiments echoed by Krone *et al.*[33] Mahony *et al.*[31] also found a higher frequency of both supraventricular and ventricular arrhythmias during ICCU management in transmural MI than in non-transmural MI, but Szklo *et al.*[30] thought that patients with non-transmural MI ('incomplete infarction') were more unstable and therefore more prone to develop lethal ventricular arrhythmias, particularly in the early post-hospital phase. Similar frequencies of supraventricular and ventricular arrhythmias in the acute phase of transmural and non-transmural MI were encountered by Rigo *et al.*,[54] Madias *et al.*[52] Scheinman and Abbott,[51] and Cannom *et al.*[29] Thus, there is considerable discrepancy in the recorded frequency of arrhythmias in transmural and non-transmural MI, but most studies show a trend to higher frequencies in the former. Long-term comparative studies do not appear to have been carried out.

Conduction abnormalities

Some quite striking data were obtained about conduction disturbances. Complete LBBB was significantly ($P = 0{,}0005$) more common in transmural anterior MI (9,09%) (Table III), and correlated with 'previous heart disease'; its incidence was 2,39% in acute transmural inferior and 1,60% in non-transmural MI. These findings were at variance with those of Hutter *et al.*,[26] who noted that although bundle-branch block was most commonly seen in transmural anterior cases (18%), it was usually complete RBBB, while bundle-branch block occurred with equal frequency (6%) in both transmural inferior and non-transmural MI ($P < 0{,}01$). Thanavaro *et al.*[38] also documented a higher frequency (7%) of RBBB in transmural MI than in non-transmural MI ($P = 0{,}0200$). Our figures for RBBB in acute transmural inferior (12,44%), transmural anterior (11,93%), and non-transmural (9,09%) MI were not different. Hauer *et al.*[55] indicated the increased risk, within 6 weeks of hospital discharge, to patients with transmural anteroseptal MI complicated by either LBBB or RBBB, and Isomäki *et al.*[56] confirmed this. Fabricius-Bjerre *et al.*[57] found no difference in frequency of either LBBB or RBBB between transmural and non-transmural MI. There was a significantly increased frequency (22,16%) of LAHB in acute transmural anterior MI ($P = 0{,}0000$) in our study in contrast to transmural inferior (9,57%) and non-transmural MI (7,49%).

Our study also documented differences in incidence of various forms of AVB (Table III). The most common form of AVB in the three groups was CHB; all types of AVB were rare in non-transmural MI, the frequency of CHB being 0,53%.

CHB occurred in 5,68% of cases of transmural anterior MI and was the commonest form of AVB in this group. Hutter *et al.*[26] found a similar distribution of the various types of AVB, and a greater frequency of AVB was also encountered in transmural infarction by Thanavaro *et al.*,[38] although this could not be verified by Fabricius-Bjerre *et al.*[57] Mahony *et al.*[31] substantiated the findings of Thanavaro *et al.*[38] as well as those of Scheinman and Abbott.[51] Madias *et al.*[52] and Cannom *et al.*[29] also showed a statistically higher incidence of second- and third-degree AVB in transmural MI. The AVB was only temporary and rarely warranted insertion of a temporary pacemaker.

Angina pectoris after MI

Angina pectoris occurred with equal frequency in the three groups; in 64,7% of patients with non-transmural MIs and 62,8% and 64,8% of patients with transmural anterior and inferior MIs, respectively (Table VI). The more severe forms of angina pectoris were seen less often. These findings were somewhat surprising; one would have anticipated more angina pectoris after non-transmural MI, a type considered 'unstable' and 'incomplete'. This symptom correlated with 'previous heart disease'. Cannom *et al.*[29] found a much higher frequency of angina pectoris (61%) after non-transmural MI than after transmural infarction (36,2%). Both Hutter *et al.*[26] and Coll *et al.*[32] demonstrated a similar pattern, and noted no difference when previous MI was taken into consideration. Marmor *et al.*[58] made the pertinent observation that recurrent angina pectoris after acute non-transmural MI was a greater predictor of reinfarction than in transmural infarction, with the obese female especially at high risk.[27] Nicholson *et al.*[34] had a similar angina incidence of 62% after non-transmural MI. Taylor *et al.*[59] found that the frequency of post-infarction angina was further related to the number of stenosed vessels supplying viable but ischaemic myocardium, and Fuster *et al.*[60,61] made the unexpected observation of an increase in angina with the presence of coronary collaterals; this was further substantiated by Vaisrub.[62] Coronary vasospasm may play a part.[63,64]

Myocardial reinfarction

During the mean follow-up of 22,2 months after discharge the incidence of recurrent MI showed a statistically significant difference between the three groups. The highest rate was 19,4% in non-transmural MI, the great majority (11,8%) being transmural anterior in location (Table VII). Infarction recurred in 17,1% of transmural anterior cases and 13,9% of transmural inferior MIs. Again, a transmural anterior location was commonest (14%) in initial transmural anterior infarction, but a recurrent transmural inferior location (6,9%) was commonest in patients with an initial acute transmural inferior infarction. Previous MI had no influence on subsequent infarction in any group.

The general trend in our study was similar to that of Hutter *et al.*[26] Again, previous MI did not determine the future rate of MI in those patients with either transmural or non-transmural MI.

Marmor *et al.*[27] found an early recurrence rate of 43% in non-transmural infarction but only 8% in transmural infarction. These authors[58] also showed that obesity, female gender and recurrent angina pectoris were risk factors for MI and that early recurrent MI in patients with a non-transmural MI increased the mortality rate most significantly from 23% to 34%. Krone *et al.*[33] also established an increased frequency of MI at 60 days' follow-up in non-transmural MI, and Madigan *et al.*[28] had a reinfarction rate of 21% at a mean of 10,6 months in acute non-transmural MI; all of these went through a phase of unstable angina. Thus, in summary, most studies demonstrated the increased propensity for reinfarction after non-transmural infarction.

Mortality after MI

Analysis of the data revealed quite dramatic results. Death in the ICCU was significantly commoner in transmural anterior MI (22,2%) than in transmural inferior (13,9%) and non-transmural (4,3%) MI (Table V). A further notable finding was the fact that mortality in the ICCU was statistically related to 'previous heart disease'. A completely different picture emerged during the first 3 months of follow-up when a significant increase in mortality occurred in those patients with non-transmural MI; the increase in the other two groups was much less. Nevertheless, mortality was still greatest in transmural anterior infarction.

The long-term mortality rate (mean follow-up 22,2 months) was still greatest in transmural anterior infarction (Table VIII). Transmural inferior MI carried a lower death rate followed by non-transmural infarction. Cardiac causes of death were significantly commoner in transmural anterior MI (88,3%), with decreasing frequencies in transmural inferior (73,2%) and non-transmural (57,1%) MI ($P = 0{,}0280$). Furthermore, a statistically significant relationship was encountered between death due to a cardiac cause and 'previous heart disease' in all three groups.

The study of Hutter *et al.*[26] highlighted a similar pattern of short-term (in the ICCU) and long-term mortality; 20% in acute transmural anterior MI, and 19% and 9% in transmural inferior and non-transmural MI respectively. The 3-month mortality rate in the non-transmural MI group rose to 14% but it was still significantly higher in acute transmural anterior (29%) and transmural inferior (27%) infarction. Thus, the survival rate was distinctly higher in the non-transmural infarction patients in both phases. Furthermore, as in our study, the late prognosis in non-transmural MI was much less favourable than the early (ICCU) prognosis. Hutter *et al.*[26] also documented a higher frequency of myocardial reinfarction in this group of patients at 9 months' follow-up, and Szklo *et al.*,[30] Cannom *et al.*[29] and Thanavaro *et al.*[38] found much the same trend. Sanz *et al.*[39] showed that in transmural MI the ejection fraction, number of diseased vessels and absence of CCF in the ICCU were the only independent predictors of long-term survival; Taylor *et al.*[59] agreed about ejection fraction. Marmor *et al.*[58] noted that the mortality rate among their patients with non-transmural MI increased markedly if they experienced an early reinfarction (23% as opposed to 34%). However, Coll *et al.*[32] showed no difference in survival for first non-transmural and transmural MI in the ICCU and at 4-year follow-up (94% and 90% in the two groups respectively). Krome *et al.*[33] related mortality in the ICCU and during the early post-discharge phase to the increase in serum enzyme levels, whereas late mortaltiy was determined by a transmural site and increasing age of the patient. Sniderman *et al.*[42] believed that recurrent myocardial ischaemia, and not only acute MI size, was a most significant factor determining mortality and morbidity.

That there is much controversy about whether long-term mortality is really different in non-transmural and transmural MI is highlighted by the finding of Fabricius-Bjerre *et al.*[57] that there was no difference. Mahony *et al.*[31] showed that both categories were at greater risk of death (in both the short- and long-term) if additional QRS-complex abnormalities were present. Rigo *et al.*[54] found no significant difference in in-hospital mortality rate between non-transmural (13%) and transmural (22%) cases, but noted increased mortality (27%) in non-transmural infarction with QRS-complex abnormalities. Furthermore, they found no difference in the late mortality

rate between non-transmural (19%) and transmural (18%) MI, but did document a higher late mortality for non-transmural infarction with QRS-complex abnormalities. Scheinman and Abbott[51] stressed the important effect of a rise in serum enzyme levels on mortality. Madias *et al.*[52] found the ECG criteria unhelpful in predicting short-term prognosis and wisely stated that both transmural MI and non-transmural MI should be observed with equal attention. This statement tends to support the myth of acute 'mild' MI.[14]

Clinical implications of our study

This study could be criticized because it is retrospective, but meticulous patient matching in the three different groups overcomes several of the known problems encountered in retrospective studies. Consideration of the effect of a history of past MI and CCF allows more meaningful interpretation of data.

Controversy has always existed about prognostic differences between acute non-transmural and transmural MI diagnosed by ECG.[65-67] Although the prevalence rates of these types of infarctions were very similar, several important differences were noted with a bearing on the short- and long-term prognosis. Acute transmural anterior MI had the worst immediate, short- and long-term prognosis with highest incidence of CCF, cardiogenic shock and acute pericarditis in the ICCU; the greatest frequency of VPBs and ventricular tachycardia in the acute phase; the highest incidence of LAHB and complete LBBB; the greatest frequency of death from cardiac causes in the ICCU and at both 3-month and long-term (mean 22,2 months) follow-up. Furthermore, transmural anterior infarction gave rise to ventricular fibrillation slightly less often than transmural inferior infarction, but patients were more prone to myocardial reinfarction. Acute transmural inferior MI had a worse prognosis than non-transmural infarction, with the most important exception of myocardial reinfarction and death at 3 months.

Non-transmural MI patients had the highest incidence of myocardial reinfarction over short- and long-term follow-up and, although death in the ICCU was least frequent, the rate at 3 months increased more dramatically than in transmural infarction groups, although the transmural anterior type was still the most frequent cause of death at this time — this despite post-infarction angina occurring with equal frequency in the three patient groups. These latter data indicate that patients with non-transmural MI have not suffered a 'mild form of heart attack' and that perhaps a more active approach, such as determining the presence of any residual myocardial ischaemia and undertaking selective coronary arteriography with a view to possible PTCA or CABG, should be employed. The antagonists of this approach should ask themselves critically what the possible pathogenesis of reinfarction and sudden death is in patients with non-transmural MI. It is very likely that the most critical factor in this clinical setting is myocardial ischaemia, which may well be reversible and should therefore be energetically sought after.

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News and Comment/Nuus en Kommentaar

Musical morbidity

Like all occupations, the making of music carries with it its own particular hazards, but whereas athletes are now very well catered for by the medical profession, the same cannot be said for musicians (*JAMA* 1984; **252:** 985). String players can suffer from stiffness, tension, pain, soreness, spasms or numbness in the fingers, hands, wrists, arm, shoulder, back, neck and jaw. Trumpet players can develop massive dilatation of the hypopharynx, and other wind players have developed headaches, retinal haemorrhages, temporary soft palatal paralysis and laryngoceles. Other sections of the orchestra are not exempt, and cymbal player's shoulder, horn player's palsy, pianists's, violinist's and harpists's cramp, English horn player's thumb and cellist's dermatitis have all been described. Sensorineural deafness is a hazard faced by all instrumentalists (particularly those who spend much time playing music by Wagner, or who have to sit near the tympani). Singers are at risk not only through faulty techniques of breathing and vocalizing, but through changes in atmospheric temperature, humidity and pollution, and all musicians, particularly soloists, are subject to the sheer nervous tension of performing before an often highly critical public audience.

What happens when they seek medical advice? Unfortunately many of them do not, because there appears to be a strong feeling among many musicians that doctors do not understand their problems. Advice like 'Well, you'll just have to give it up', which is all too common, is about as sensible as telling the Prince that he must stop being Hamlet. Many musicians simply cannot afford to stop doing the one thing that they can do really well, and most will carry on making music until it is physically impossible simply because of the almost mystical significance attached to making music.

Doctors should be more aware of the specialized problems of musicians, and adopt a more positive attitude towards them.

Inflatable splints and limb ischaemia

Inflatable splints for stabilizing limb fractures during transportation from accident sites are effective, convenient, easily applied, and have become popular among members of emergency services. However, is it possible that, like tourniquets, they can cause ischaemia of the limb? It appears that they can (Shakespeare *et al.*, *Injury* 1984; **16:** 38). A moderate rise in muscle compartment pressure is quite common after injury due to traumatic swelling of the contents or haemorrhage; even elevation of the limb above the level of the heart can cause ischaemia. The uneven pressure applied to a limb by a compressive dressing does not normally cause pressure to rise, and the pressure decreases with time. In this investigation the anterior compartment of the leg of 4 volunteers was cannulated and connected to a pressure manometer. Pressure was then applied by inflating the splint. The pressure applied was transmitted directly into the tissues of the leg and was added to the pre-existing pressure in the anterior compartment. Care should be taken by those using these splints that the blood supply to the limb is not compromised.