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# Assessment for Prognosis during and after Myocardial Infarction

## A Plea for a Stratified Approach

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### *Introduction and Concepts*

This overview recapitulates some essential conditions which must be met for the recent rapid advances in myocardial infarction treatment to become applicable for the community at large. These conditions require the concept of coronary care to be expanded and stratified in a logical fashion of interlocking steps, as shown in table I.

Such a stratified network must be worked out for myocardial infarction from its most acute stage, phase I (table I–IV) onwards. This aggressive approach is much in the limelight now that recent trials with thrombolytic agents have shown the possibility to limit infarct size by 30%, to reduce ventricular fibrillation and cardiogenic shock by 50%, and to improve ventricular function by up to 8% in terms of ejection fraction and thereby reduce short- and long-term mortality to half of that in a randomized control group (9 rather than 16%; table II). This alone would influence the outcome of acute myocardial infarction in a dramatic fashion. In fact, with the steps indicated in tables III and IV, the coronary care concept must be broadened and be made systematic to encompass all patients in a continuous framework of care which begins with the first manifestations of coronary artery disease and ends with their ultimate death. While the needs for such a systematic approach are becoming more and more evident, resistance from the health care authorities and insurers appears to mount as well (table III–V). The only proper replies to their questions lies in providing the proof that earlier treatment is in fact cost effective as the cost of postmyocardial infarction care with its

*Table I.* The proper concept of coronary care between 1985 and 2000 depends on the perception of the problem

| Time | Now | Why |
|---|---|---|
| Phase I, 0–4 h includes: | self referral to precoronary care for evaluation, home care and/or mobile coronary care unit as well as early reperfusion | can MI be avoided/limited? |
| Phase II, 4 h to 20 days includes: | coronary care unit + intermediary care inclusive assessment for prognosis after care | can MI be optimally treated? |
| Phase III, remainder of life includes: | regular supervision revalidation reconstruction of coronary artery system | can MI be prevented from recurring? |

*Table II.* Intravenous and intracoronary streptokinase. Overall results, The Netherlands Study [1], 533 patients (percent values) treated v.s. controls

| | |
|---|---|
| Reperfusion achieved | 85 |
| Limitation infarct size (HBDH) | 740 vs. 1,120 U |
| Left ventricular ejection fraction | 53 vs. 47 |
| Cardiogenic shock | 13 vs. 24 |
| CHF during convalescence | 37 vs. 53 |
| Ventricular fibrillation | 38 vs. 61 |
| Mortality ⩽ 30 days (n = 533) | 16 vs. 31 |
| Mortality median 8 months (n = 533) | 23 vs. 42 |
| Reinfarction rate | 36 vs. 16 |

*Table III.* Stratified approach (costs)

Public health authorities are not really interested in reducing deaths, but in reducing costs; so

1 Since smaller infarcts follow early reperfusion, there will be a shorter hospital stay, less complications (shock, VF, CHF, MI) and less loss of employment

2 When (post)infarct assessment is systematized
post-myocardial infarction complications can be reduced
and unnecessary expenditures avoided

*Table IV.* Towards a stratified approach in myocardial infarction

| |
|---|
| *Secondary prevention* |
| A Early detection in community |
| B Rapid admission policy |
| C Early discharge policy |
| *Tertiary prevention* |
| D Early classification in OPD |
| E Advocate self referral |

*Table V.* Stratified approach, needs

| |
|---|
| More public awareness |
| Better referral |
| Better registration of events |
| Optimalization of transport |
| Quicker admission |
| Better classification upon admission |
| More careful stratification upon discharge |
| Better network 3° vs. 2° centers |

(avoidable) complications is in fact higher than their prevention, consisting of early and aggressive reperfusion efforts. The recent publication of the Netherlands Interuniversity Cardiology Institute Trial data [1] confirming some of the better designed earlier trials [2–4] and its similarity with just published data from the Italian trial [5] is in this regard most significant.

As efforts at supporting the heart during infarction mechanically, or through pharmacological means such as afterload reduction or inotropic therapy, have not been as successful as expected, attention has been directed towards prevention of ventricular failure and reduction of the myocardial infarct or even at outright avoidance of its occurrence through treatment with beta-blockade or calcium antagonists. A variety of agents seemed promising in the experimental animal in terms of limiting infarct size, yet recent large clinical trials with the early administration of beta-blockers and calcium antagonists have failed to influence mortality in a major fashion. The MIAMI [6] and ISIS trials [7] involving together more than 23,000 randomized patients, while showing a modest 13–16% reduction in acute mortality, largely in those treated in the first few hours, have been carried out in patients with a low mortality risk (4–6%). Furthermore, trials [8, 9] with late beta-blockade aimed at

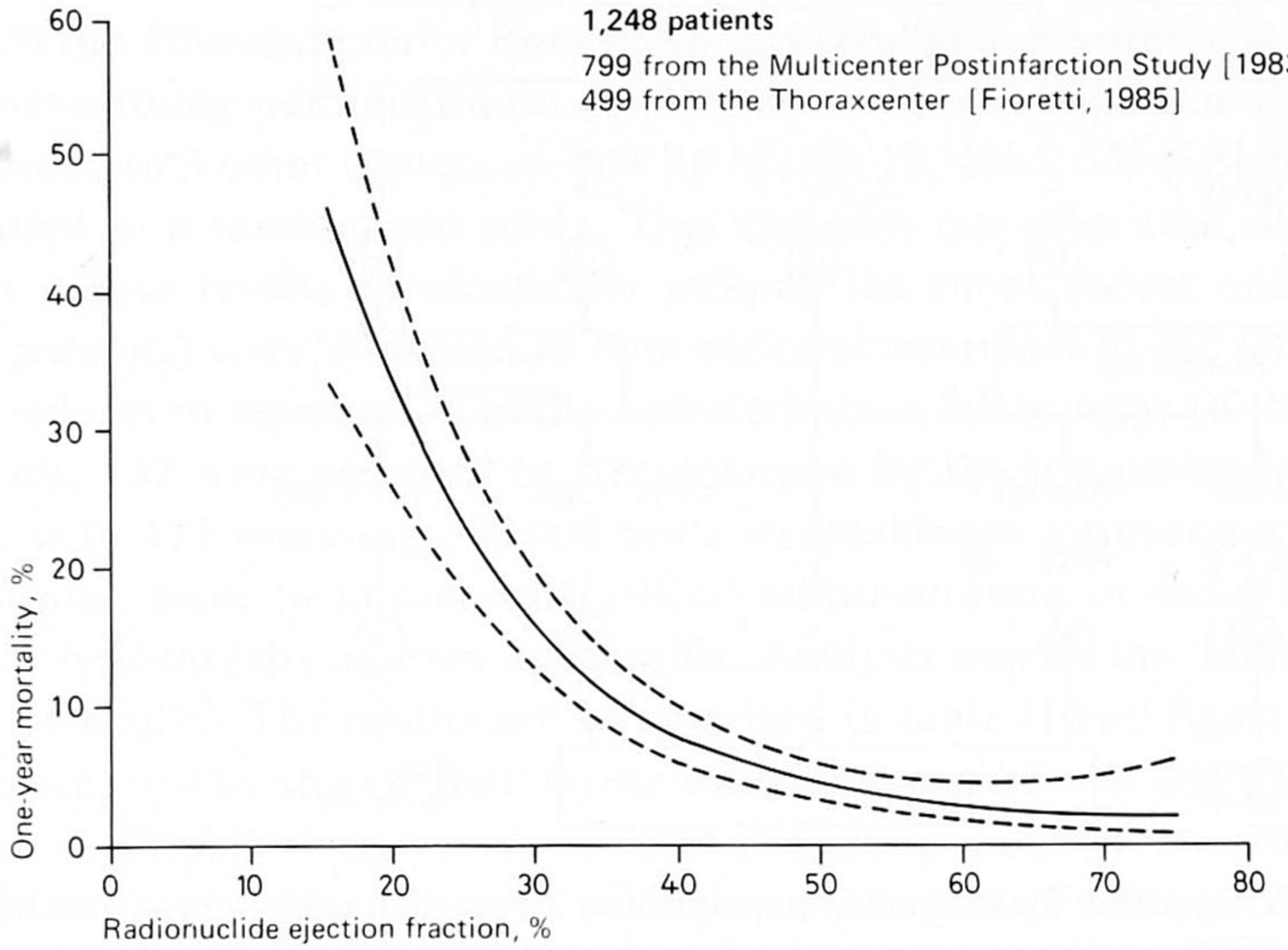



*Fig. 1.* One-year cardiac mortality as a function of radionuclide ejection fraction (%) measured at hospital discharge after acute myocardial infarction. The dotted lines indicate 95% confidence limits. The calculations are based on pooled data from the Multicenter Postinfarction Study [13] and the Thoraxcenter [14].

secondary prevention, have at best shown a 25% reduction in mortality. Even less significant results have been reported for calcium antagonists [10–12]. In both the early and late onset trials, up to 60% of the population at risk was excluded by contraindications of one kind or another and side effects have not been inconsiderable. Thus, at best in 20% of 40% at risk, i.e. in 8% of the total infarct population, can mortality reduction by this approach be anticipated and even then would be limited to 'low risk' cases.

This realization increased the attention to early reperfusion efforts with thrombolytic agents, the first of which dates from 1958 [15]. The relevance of attempts to improve left ventricular function and to reduce the amount of tissue lost to infarction is evident from figures 1 and 2, which depicts the relationship between residual left ventricular ejection fraction, a general expression of left ventricular function, and 1-year mortality after acute infarction with conventional therapy. Based on this relationship, it can be expected that by preserving or restoring the ejection fraction major increases in survival can be produced. Means to achieve this goal are now at hand.

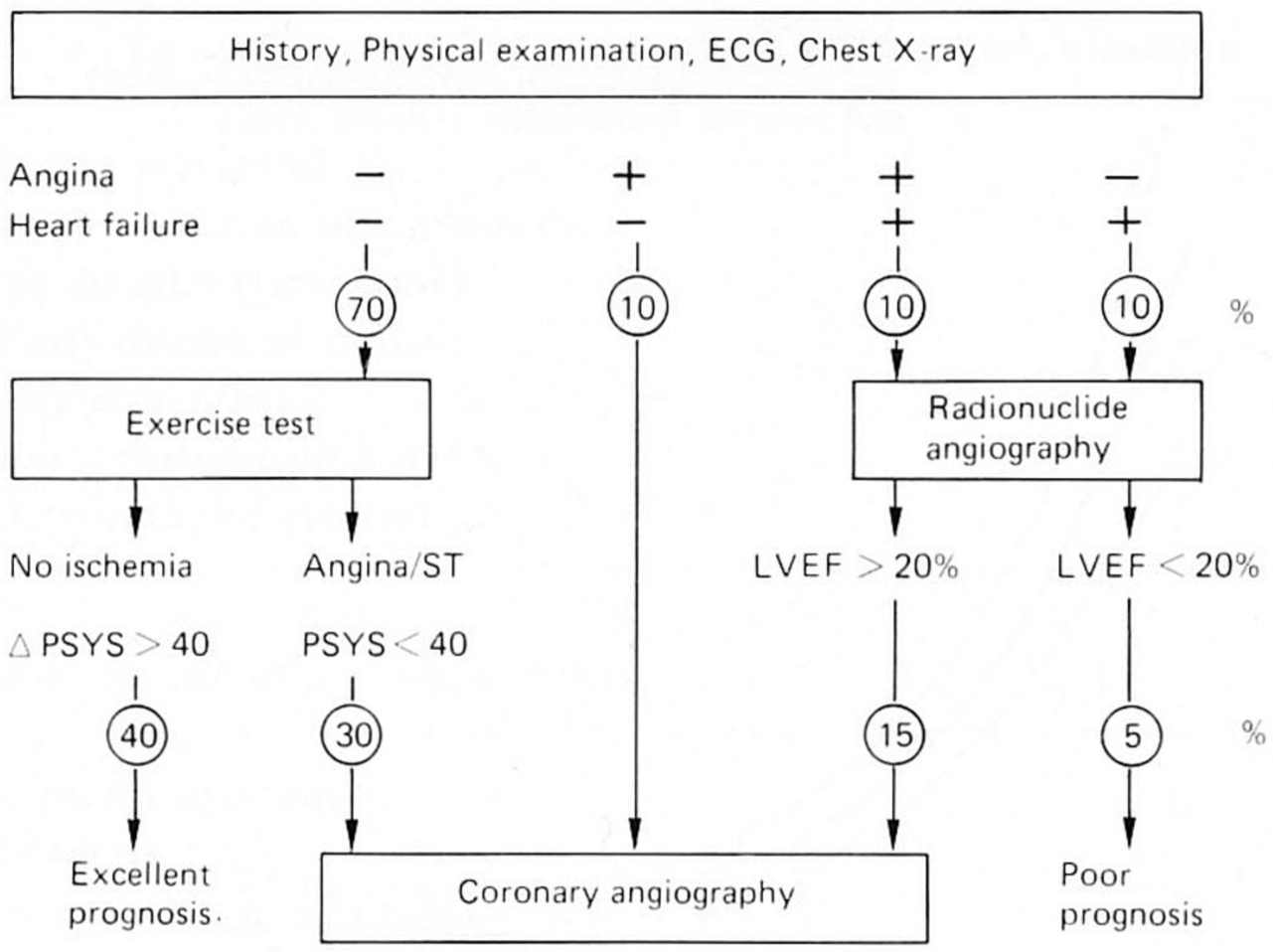



*Fig. 2.* Algorithm for evaluating patients after myocardial infarction at time of discharge. Δ PSYS = Rise in systolic blood pressure during stress testing; LVEF = left ventricular ejection fraction.

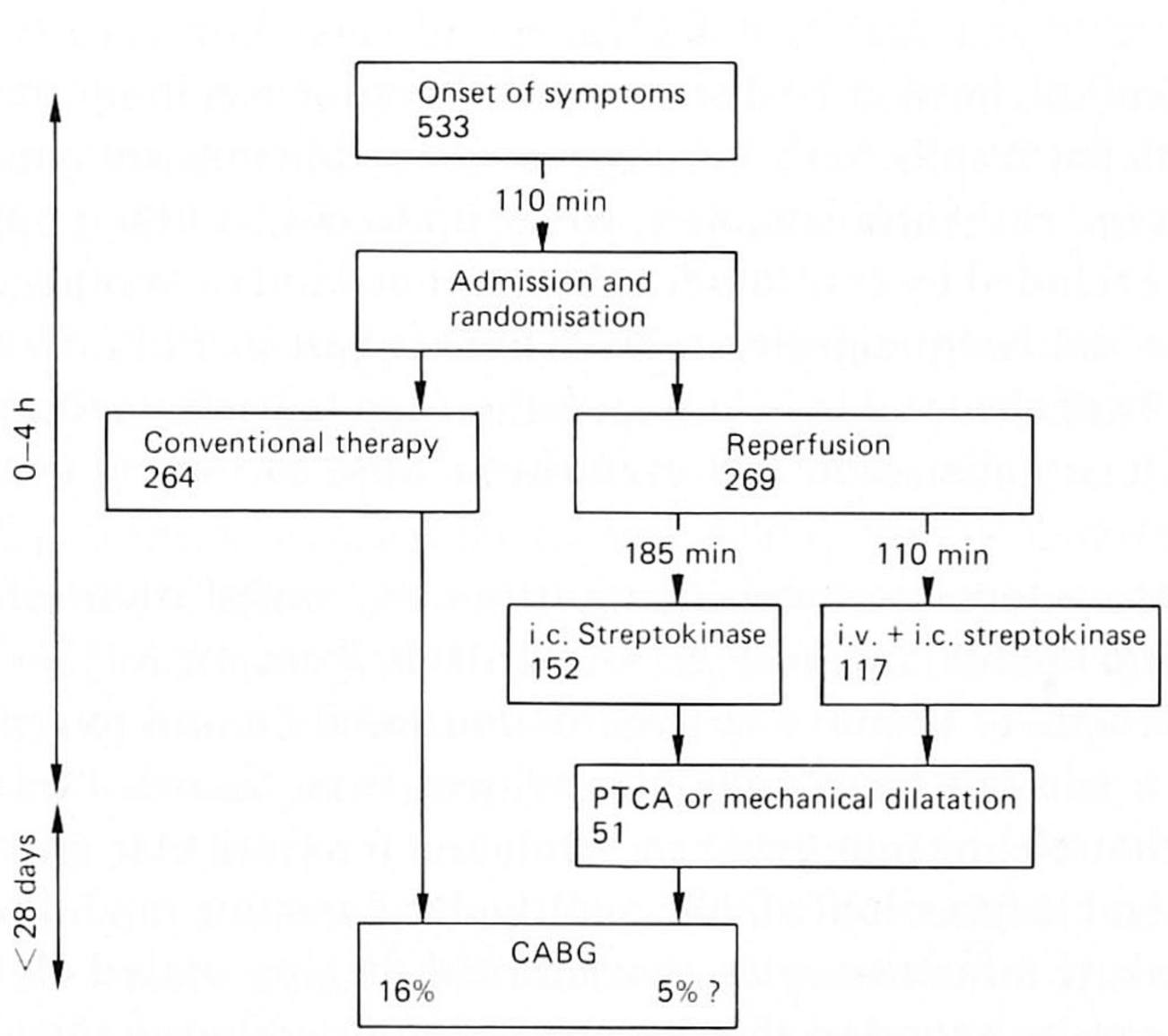



*Fig. 3.* Flowchart of intervals until final treatment in 533 patients of the Netherlands Interuniversity Cardiology Institute Trial.

At the Thoraxcenter in Rotterdam an investigation into the relevance of intracoronay administration of streptokinase was begun in 1981 and expanded to 3 other centers so that by March 15, 1985, 533 patients were included in a randomized study. This excludes our pilot trial of 82 patients whose results incidentically mimick the more recent ones. Half (264 patients) were allocated to conventional treatment in the CCU and 269 patients to reperfusion in the catheterization laboratory. Of these 269 patients, 152 were assigned to streptokinase by the intracoronary route only, with 117 receiving 500,000 units streptokinase intravenously (i.v.) ±100 min. prior to intracoronary (i.c.) administration in order to commence lytic therapy as soon as possible. Analysis was on the 'intention to treat principle'. The results are summarized in table II and figure 3. Furthermore, it was shown that 1-year mortality was 9% in the group assigned to thrombolysis versus 16% in the group assigned to usual therapy. Best results were achieved in the i.v. + i.c. group. Also, cardiogenic shock, ventricular fibrillation and pericarditis were considerably less in the group assigned to thrombolysis (table II). In addition, enzyme release (alpha-HBDH) was reduced by one-third on average, indicating a smaller size of the ultimate infarct in the treated group. This was also reflected in left ventricular ejection fraction which was significantly higher in the treated group than in the control group (table II, VII). the significance of this is shown in figure. Furthermore, throughout the study, experience was gained with percutaneous transluminal angioplasty (PTCA) which was carried out in those patients who, after initial reperfusion in the catheterisation laboratory, showed a significant residual coronary artery obstruction. In this subgroup of patients a 1-year mortality of $<2\%$ was observed. Similar results have been obtained in 135 recent patients with unstable angina, a condition which may be considered as a precursor of acute myocardial infarction. Again in this group, mortality is 1% with low recurrence rates of angina or infarction, when PTCA is carried out in the subset of patients refractory to pharmacological therapy [20]. Similar experiences are shown in patients who have undergone successful bypass grafting for stable angina (table VI).

The majority of the patients dying after myocardial infarction have extensive coronary artery disease. A recent very convincing study by Davies [16] indicated that in 100 subjects who died of ischemic heart disease in less than 6 h, coronary thrombi were present in 74, while plaque fissuring was seen in 21 of the remaining 26. Thus, only in 5 out of 100 cases could no acute arterial lesion be demonstrated. Their data also con-

*Table VI.* Mortality and causes of death at the Thoraxcenter, Rotterdam, The Netherlands after coronary artery surgery

| | Total | | Women | | Men | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| Number of patients | 1,041 | 100 | 126 | 100 | 915 | 100 |
| Operative mortality (28 days) | 12 | 1.2 | 3 | 2.4 | 9 | 1.0 |
| Late mortality | | | | | | |
| Cardiac, sudden | 15 | 1.4 | 2 | 1.6 | 13 | 1.4 |
| Cardiac, at reoperation | 2 | 0.2 | – | | 2 | 0.2 |
| Cardiac infarction | 17 | 1.6 | 4 | 3.2 | 13 | 1.4 |
| Chronic cardiac failure | 5 | 0.5 | – | | 5 | 0.6 |
| Noncardiac causes | 13 | 1.3 | 1 | 0.8 | 12 | 1.3 |
| Total mortality, all causes during 8 years follow-up | 64 | 6.2 | 10 | 8.0 | 54 | 5.9 |

*Table VII.* Comparison of first vs. repeat myocardial infarctions in 533 patients (Netherlands Interuniversity Cardiology Institute Trial)

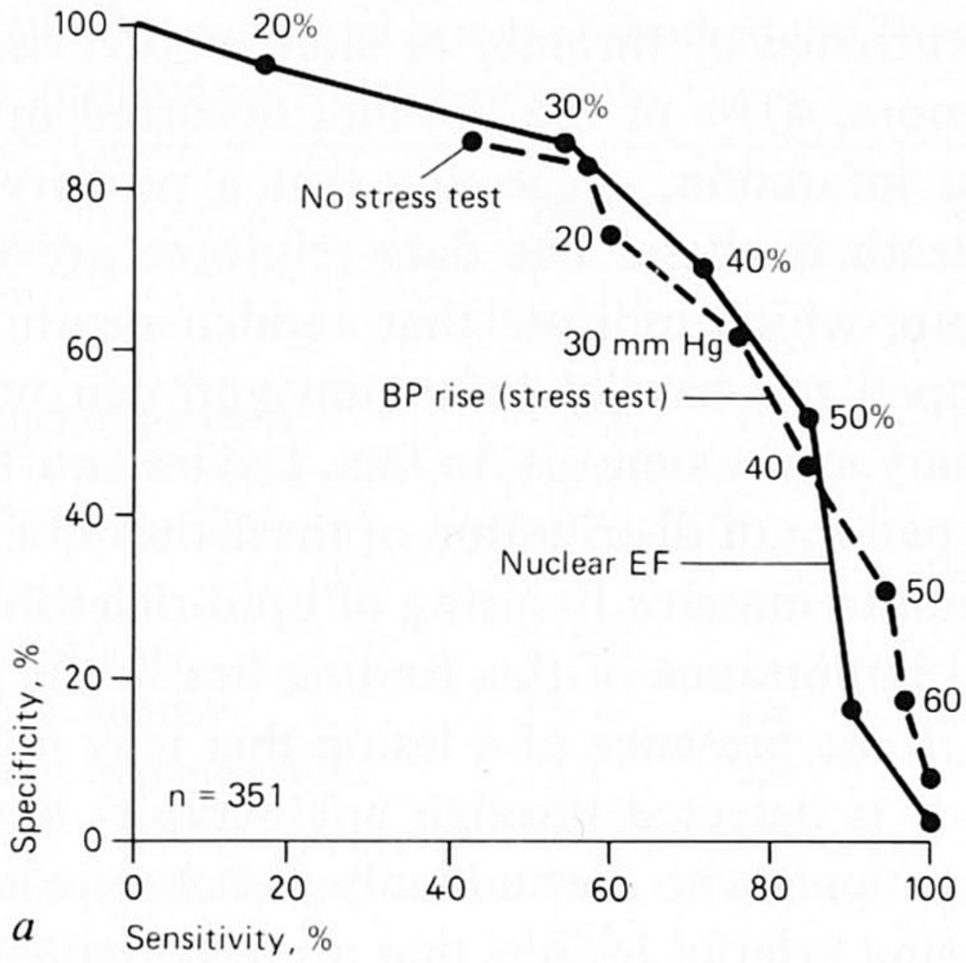
| | c̄ prior infarction | | | | s̄ prior infarction | | | |
|---|---|---|---|---|---|---|---|---|
| | streptokinase | | control | | streptokinase | | control | |
| | n | % | n | % | n | % | n | % |
| Number of patients | 56 | | 60 | | 213 | | 204 | |
| ⩾8 months † | 12 | 21 | 16 | 27 | 11 | 5 | 26 | 13 |
| ⩽14 days † | 7 | 12 | 11 | 18 | 7 | 3 | 15 | 7 |
| CABG/PTCA ⩽14 days | 17 | 30 | 12 | 20 | 27 | 13 | 18 | 9 |
| | 7 | 12 | 3 | 5 | 9 | 4 | 4 | 2 |
| Angio EF | 45% | | 40% | | 55% | | 49% | |
| Radionuclide EF ⩽14 days | 39% | | 35% | | 56% | | 50% | |
| Infarct size, u | 710 | | 1,090 | | 770 | | 1,140 | |

firm a high incidence of occurrence of thrombi at sites of preexisting high grade stenosis. Furthermore, 47% of the thrombi occurred in the right coronary artery without infarction, suggesting that a primary arrhythmia was the cause of death in these. The data reinforce previous studies from the Seattle group, which indicate that sudden death can strike without a fully developed myocardial infarction and can occur with varying severity of coronary artery stenosis. In fact, Davies and Thomas [17] state: 'No particular pattern of distribution of these thrombi was evident; all occurred in relation to massive fissuring of lipid-rich atheromatous plaques.' The clinical importance of this finding lies in the fact that thrombosis can develop in the presence of a lesion that may not be regarded as important when it is detected through angiography during the patient's life and that 'all patients who die suddenly of ischemic heart disease have actively progressing arterial lesions that are predominantly thrombotic. This fact has clear therapeutic implications for the reduction of the incidence of sudden death, and the recent demonstration that aspirin protects patients with unstable angina from acute myocardial infarction and death (better than any other therapy) illustrates this point. In our view the coronary-artery lesions in patients who die suddenly of ischemic heart disease are identical to those found in unstable angina.' Thus, the prognosis assessment of patients with acute myocardial infarction will require a thorough examination of the coronary artery system, at least in those in whom ischemic signs persist or are invokable after initial recovery.

### *Identification of Subsets Carrying High Risks*

#### *Patients with Angina pectoris/Unstable Angina*

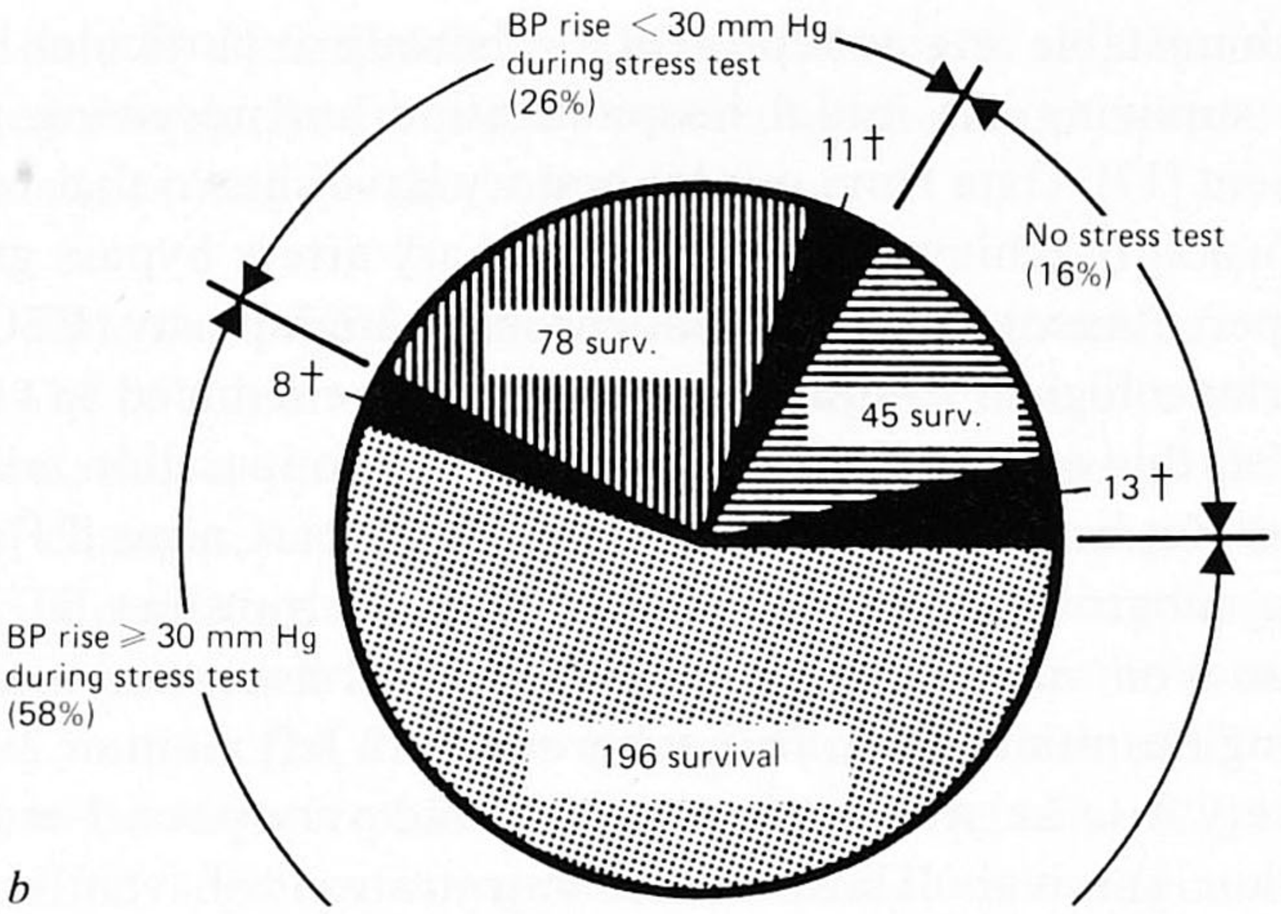
Angina pectoris is a potential precursor of acute myocardial infarction but its electrophysiologic and metabolic characteristics may create a different setting, which can lead to sudden death without infarction. The prevalence of angina pectoris is very high and its course is most often benign. Nonetheless, it is an antecedent of at least 50% of patients dying suddenly, as is clear from the earlier comments by Davies. Ruberman et al. [18] studied 400 patients with angina pectoris without prior myocardial infarction and compared their mortality over 5 years with that of more than 1700 men with prior myocardial infarction. From 1 h of ECG monitoring a multivariate analysis of survival parameters identified the



*a*

presence of PVCs and ST segment depression as the best independent predictors of death among those with angina, in addition to their age. Little difference in survival was found among the men with simple and those with complex PVCs but the mortality rate in both of these groups far exceeded those in whom PVCs were absent during the 1-hour monitoring period.

Because most patients with angina pectoris can undergo exercise testing, the presence of PVCs during exercise has been increasingly examined for its predictive value for the occurrence of myocardial infarction and death. In patients with coronary artery disease, the annual incidence of new coronary events was 1.7% in those without PVCs or ischemic ST segment changes, 6.4% in those with PVCs alone, 9.5% in those with only ischemic ST segment changes and 11.4% in those with both abnormalities. Thus, patients with coronary artery disease, exercise-induced PVCs and ST segment changes seem to be at higher risk. In our laboratory, it has become evident that the occurrence of exercise-induced arrhythmias has but limited value beyond ST segment changes. Fioretti et al. [19] have shown in a post-myocardial infarct population that the overall response to exercise, in particular the extent of blood pressure rise, has a much stronger predictive value. It is clear that the exercise test should be looked at in its entirety and that it is a very useful test in the physician's attempts at stratification of risk (fig. 2, 4).

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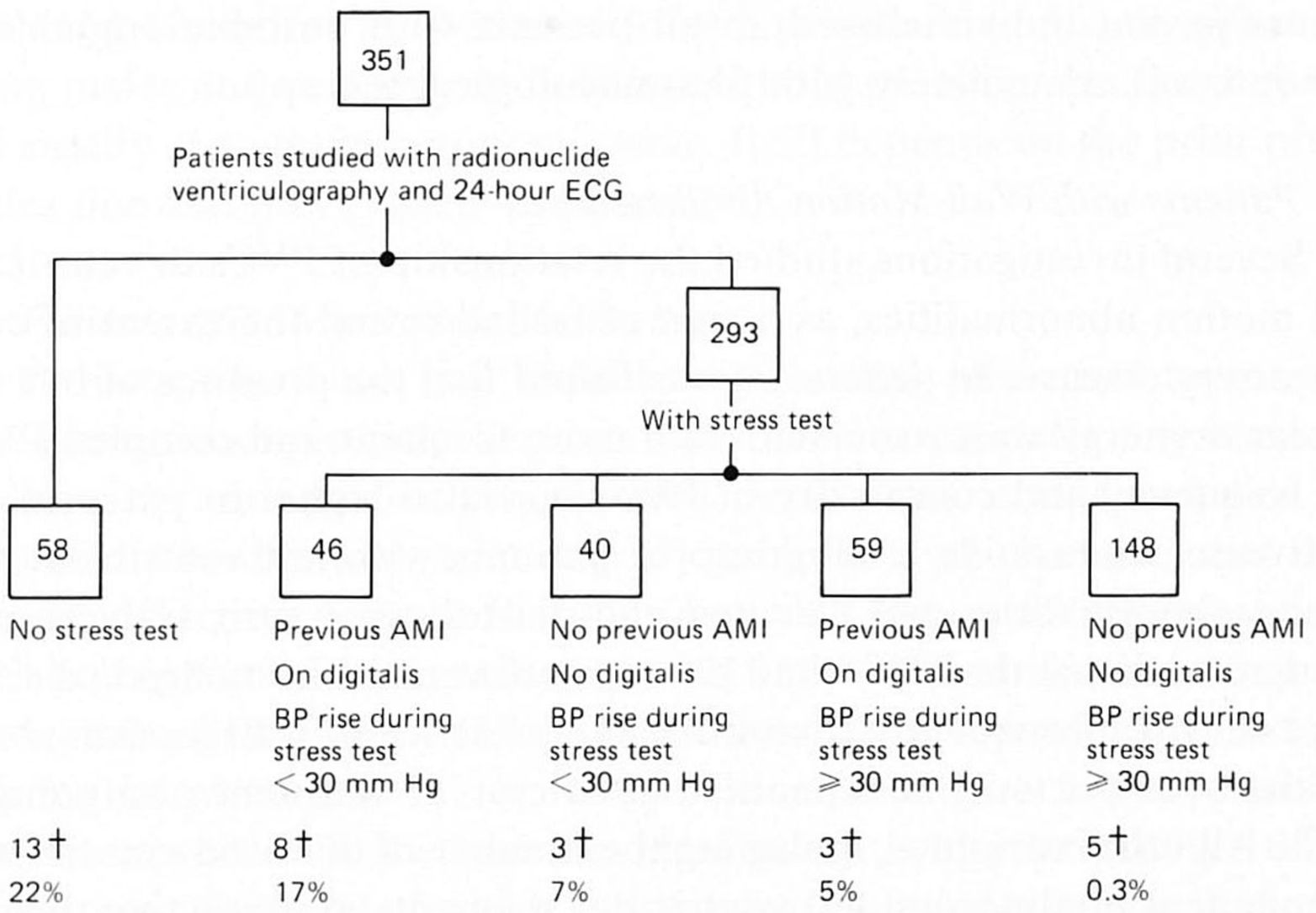


*c*

*Fig. 4a* Prediction of survival after myocardial infarction. Specificity versus sensitivity of exercise testing for late mortality (rise of systolic pressure during the test) and of radionuclide ventriculography. The superimposition of the 2 curves indicates that the 2 tests have a similar prognostic value. *b* One-year survival after hospital discharge in subsets of patients with myocardial infarction based on clinical and exercise test results (n = 351). *c* Prediction of 1-year mortality by exercise testing in hospital survivors of myocardial infarction. BP = Systolic blood pressure.

Patients with unstable angina represent a subgroup at particular high risk even when surviving the initial hospitalization and receiving prophylactic treatment [17]. Data from our laboratory have shown that when adequate reperfusion is achieved, either by coronary artery bypass grafting (CABG) or percutaneous transluminal coronary angioplasty (PTCA), after initial pharmacological cooling, mortalitity can be reduced to virtually zero [20]. Yet, this approach demands extensive cooperation within one unit and may not be extrapolated to all hospitals yet. Cairns [21] further identified a subgroup at 'high risk' who showed transient ST segment changes and/or ventricular tachycardia on 24-hour ambulatory monitoring during hospitalization. This subgroup with left main or 3-vessel coronary artery disease with variant angina had very poor 3-month posthospitalization survival. Thus, the demonstration of ventricular tachycardia and transient ST segment depression in patients with unstable angina allows the identification of a subgroup with severe coronary artery disease and risk of sudden death without need for exercise testing. These examples show that the therapeutic approach should be aggressive, but individualized, in all patients with unstable angina who do not 'cool' immediately with pharmacological therapy.

### *Patients with Wall Motion Abnormalities*

Several investigations studied the relationship of PVCs to ventricular wall motion abnormalities, as a sign of ischemia and the extent of coronary artery disease. In general, it was found that the presence of left ventricular asynergy was associated with more frequent and complex PVCs. The frequency and complexity of PVCs was also higher in patients with multivessel disease. In a subgroup of patients with left ventricular wall motion abnormalities and elevated end-diastolic pressure, Calvert et al. [22] demonstrated that 40% had paroxysmal ventricular tachycardia. Califf et al. [23] subsequently found that the presence of wall motion abnormalities was the only independent predictor of frequent and complex PVCs. All other variables, including the number of diseased vessels, were independent of abnormal left ventricular segments in predicting their association with PVCs. Only 11% of patients without an abnormal segment had 2 more PVCs/h whereas this percentage increased to 73% and 100% for patients with 4–6 and 7–9 abnormal segments, respectively. It therefore would seem advisable in all cases with significant PVCs to search by echocardiography and/or scintigraphy for such wall motion abnormalities as they may indicate ischemia, which is still treatable. Re-

cent data by Deanfield et al. [24] and Hirzel et al. [25] confirm the importance and relative frequency of silent ischemia in whom wall motion abnormalities may be the 'warning' sign.

*Patients with Silent Ischemia (The 'Silent Attack')*

The 'silent attack' conveys a concept of invisible submarine warfare. In fact, this is so. Modern technology has allowed us to measure ST segment changes, as well as rhythm disturbances during 24-hour ambulatory ECG monitoring while the patient was at rest or carrying out minimal activity – yet symptoms would be absent – hence the term 'silent attack'. Many authors [24, 28] have shown many episodes of ST segment depression to be without pain. However, Armstrong et al. [27] have argued that not all ST segment changes denote an ischemic origin. In their series of 44-year-old men with long-term follow-up, no increased death rate was observed. Thus, like with all tests, the interpretation depends on the substrate. In a patient with proven coronary artery disease, we would interpret ST segment depression of 1 mm and T wave changes or echocardiographic dysfunction as evidence for silent ischemia. In asymptomatic young males and certainly in females without previous disease they may well signify changes in autonomic tone. It all depends on the prior probabilities one assumes (Bayes' theorem).

*Patients after Myocardial Infarction*

Patients who have had definite myocardial infarction represent a large group among which there are those who are at definitely increased risk of early and, particularly, sudden death. This is evident when one looks at the Netherlands trial data [1] (table VI), which shows that those with an infarction prior to the one for which they were reperfused had much higher 8-month mortality rates (21%) than those in whom reperfusion was the first therapy (5%). The control groups, not reperfused, had even higher mortalities 27 vs. 13%. The mortality rate after discharge from the hospital for the entire control group is approximately 16% in the first year (table II) of which sudden death is most common in the first months, amounting to at least half of the total 1st year mortality.

Many investigators have studied the importance of premature ventricular complexes in the immediate and posthospitalization phase for mortality related to other risk factors such as impaired left ventricular function. Ventricular arrhythmias detected during the early phase of an acute myocardial infarction proved to be of no predictive value for sudden

death although the absence of complex PVCs did not preclude its occurrence later. One of the early findings of the Coronary Drug Project [28] was that the presence of one or more PVCs on a 12-lead ECG was associated with an increased risk of SCD during a variable 2- to 5-year follow-up. This finding has led to long-term ECG monitoring by many to evaluate the incidence and significance of PVCs in postinfarction patients and to develop specific risk stratification of patients who had survived a myocardial infarction on the basis of the results.

One of these, Ruberman et al. [29] prospectively studied 1,739 men with a history of myocardial infarction who were monitored for 1 h. After 4 years of follow-up they found that complex PVCs were associated with a threefold increased risk of sudden death when compared with patients without PVCs. When 16 other clinical variables were considered, multivariate analysis showed that complex PVCs were independent in their predictive power. For example, patients with congestive heart failure had a 3-year cumulative probability of sudden death of 21% when complex PVCs were present, as compared to only 8% when CHF occurred without such PVCs. These conclusions are somewhat at variance with those from Moss et al. [30] using a 6-hour monitoring period who concluded that complex PVCs were not an independent or sensitive indicator for subsequent sudden death as opposed to overall cardiac death. They indicated that the extent of coronary artery disease and the degree of left ventricular dysfunction after the first infarction were the most important factors contributing to the risk of cardiac death. Geltman et al. [31] found that PVCs were more frequent in patients with larger infarctions and that their combination was associated with increased early and late mortality after acute myocardial infarction. Schulze et al. [32] compared the predictive value of complex PVCs to that of a left ventricular ejection fraction of less than 40% determined prior to discharge after acute myocardial infarction. The combination of complex PVCs and reduced ejection fraction, identified all patients dying suddenly within 6 months. Davis et al. [33] studied 940 postinfarction patients. They found that the combination of an anterior myocardial infarction, clinical signs of left ventricular dysfunction and PVCs identified a high risk group with a 6-month mortality of 15% and a 3-year mortality of 30%. Thus, it is now generally accepted that the presence of left ventricular dysfunction after a myocardial infarction allows the identification of a high risk group for sudden death, while the occurrence of complex PVCs increases this risk even further.

In fact, these findings led us to investigate our own patients [34]. We summarized this recently as follows: 'The relative merits of resting ejection fraction measured by radionuclide angiography and predischarge exercise stress testing, were compared for predicting prognosis in hospital survivors of myocardial infarction. Two hundred and fourteen survivors of myocardial infarction were studied over a 14-month period. Hospital mortality was 13% (45 of 338) whereas 18 additional patients out of 214 died in the subsequent year (9%). High, intermediate and low risk groups could be identified by resting left ventricular ejection fraction measurement. Mortality was 33% in patients with an ejection fraction less than 20%, 19% for 58 patients with an ejection fraction between 20% and 39% and 3% for 147 patients with an ejection fraction higher than 40%. Mortality was high (23%) in 47 patients who were unable to perform the stress test because of heart failure or other limitations. The patients could be stratified further into intermediate and low risk groups according to the increase in systolic blood pressure during exercise: 6 deaths occurred in 46 patients with a blood pressure increase of $<30$ mm Hg while only 2 deaths occurred in 121 patients with an increase of $>30$ mm Hg. Maximum workload, angina, ST segment changes and ventricular arrhythmias were less predictive than were blood pressure changes. It was also concluded that the prognostic value of radionuclide angiography at rest and of symptom-limited exercise testing is similar. The latter investigation should be the method of choice since it provides more specific information for patient management. In a later study [35] we also showed that out of 298 patients, followed 1 year after a postinfarction predischarge exercise stress test, the mortality in 24 patients with ventricular couplets, ventricular tachycardia or ventricular fibrillation during the test was 12 vs. 6% in the 274 without such an arrhythmia. Since the occurrence of such severe arrhythmias during such exercise testing is rare, the significance of these arrhythmias is limited and the difference in mortality shown above has but little clinical relevance. If such an arrhythmia occurs during stress testing it has relevance but other information obtained during the test has more significance.'

During hospitalization for acute myocardial infarction there are important observations to be made regarding immediate and late outcome, as we have shown in earlier studies [36, 37]. We have compared these clinical data with the outcome of the discharge exercise test in the same 405 patients. These data were published in 1985 and can be summarized

as follows [19]: 'The predictive value of a predischarge symptom-limited stress test was studied in 405 consecutive survivors of acute myocardial infarction. Three hundred patients performed bicycle ergometry, 105 could not perform it. Among these latter 105 patients the stress test was contraindicated in 43 because of angina or heart failure and in 62 because of noncardiac limitations. One-year survival was 44% in the 'cardiac limited' group (19 of 43) and 92% in the 'noncardiac-limited' group (57 of 62). One-year survival among the patients who performed an exercise test at discharge was 93% (280 out of 300). The best stress test predictor of mortality by univariate analysis was the extent of blood pressure rise during exercise (BP increase: 42 ± 24 mm Hg in 280 survivors versus 21 ± 14 mm Hg in 20 nonsurvivors; $p<0.001$). Among the 212 patients in whom BP increased by 30 mm Hg or more, mortality was 3% (n=6), while it was 16% (n=14) among the 88 patients in whom BP increased by less than 30 mm Hg. Angina, ST changes and arrhythmias were not as predictive. Stepwise discriminant function analysis showed an inadequate BP increase to be an independent predictor of mortality. A high-risk group can be identified at discharge on clinical grounds in patients unable to perform a stress test, whereas intermediate and low risk groups can be identified by the extent of BP increase during exercise.'

A recent publication from our laboratory [Fioretti et al. accepted with revision by J. Am. Coll. Cardiol.] summarizes our current and, as far as possible, final thoughts on this matter. The data on the population from which the main conclusions are derived are shown in figure 4 and speak for themselves.

*Conclusion*

Our current opinion is that all patients, having come in contact with the medical profession, for one or more signs, symptoms or syndromes of coronary artery disease should be examined in a cost-effective manner, by beginning with their clinical variables. If acute ischemia is present early aggressive therapy must be considered. Those with lesser symptoms should be subjected to a symptom-limited exercise test or radionuclide evaluation if the former is impossible to be completed or equivocal. If these are normal, no therapy is needed and this may apply to 55- to 65% of all pre- or post-myocardial infarction patients.

If abnormal or contraindicated for cardiac reasons, patients should then be examined with angiography with the aim at and for the purpose of complete revascularization. If the latter is impossible, anticoagulant (aspirin, dipyridamol, coumadine), and anti-ischemic (beta-blockade with calcium antagonists) therapy should be provided. All patients must be followed monthly until free of ischemic signs and then at intervals of 6–12 months.

## *Summary*

Right after the first signs and symptoms of acute myocardial infarctions the prognosis is determined by the interventions which are carried out at that time. Preservation of as much myocardial tissue is the key element. Early desobstruction and reperfusion of the myocardium at jeopardy can lead to limitation of the ultimate infarct size, improved ventricular function and a halving of the 1-year mortality. Early supportive therapy with beta-blockade and calcium antagonists may enhance this effect. Data in 533 patients randomized to either a reperfusion strategy or to conventional therapy, combined with those from the recent literature on thrombolysis and early beta-blockade, provide the basis for this point of view. Once infarction is unavoidable and in the process of completion, probably 3–4 h after onset of symptoms, only supportive therapy is recommended, which will hardly change the outcome except for interventions during clinical care such as defibrillation.

In 351 other survivors of myocardial infarction the value of clinical variables, a sympton-limited bicycle stress test at discharge, radionuclide ventriculography and 24-hour ambulatory electrocardiogram was compared in predicting 1-year survival. A history of previous myocardial infarction and heart failure during the current episode proved to be the strongest clinical predictors of death. Similarly, a low ejection fraction (<40%) and an insufficient blood pressure rise during stress testing (<30 mm Hg) identified a high risk group. Stress-test-induced angina and ST depression as well as ventricular arrhythmias from 24-hour electrocardiography were less good as predictors. In these patiens treatment should be individualized and may require arteriography. Patients eligible for and completing a normal bicycle stress test after myocardial infarction proved to be a low risk group, which may constitute 65% of the total, seen in tertiary referral centers and even more in community hospitals. They neither require therapy nor further investigation. A subgroup with an intermediate risk can be identified when clinical variables, stress testing and/or resting radionuclide ventriculography are abnormal. This group requires 'tailored' therapy. Therefore, after infarction recovery, we recommend a pre-discharge stress test routinely to complement the clinical evaluation, since it also provides information on physical capacity, the indication of arrhythmias and the presence of myocardial ischemia. Thus, optimal management of acute myocardial infarction requires a stratified approach, which does not require expensive testing procedures.

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