Utility of conventional risk factors in evaluation of patients with coronary disease

William B. Kannel, M.D.
P. Sorlie, M.D.

Framingham, Massachusetts

We are uncertain about what remediable factors influence the future health and survival of coronary patients. It is clear that the amount of damage sustained by the heart and its circulation is an important determinant of survival. It is not established whether the major risk factors play an important role at this stage in the disease as they clearly do for the presymptomatic coronary candidate.

Once clinical coronary heart disease appears, the average prognosis is not encouraging.[1] Following a first-recognized myocardial infarction, a man's chances of dying within the first year (in the Framingham cohort) are about 20%. This is a mortality rate 14 times that of the cohort free of coronary heart disease. This proximate mortality includes hospital mortality and possibly some prehospital mortality, but not sudden death, which has been excluded. After surviving the first year, chances of dying in the ensuing 5 years is 23%, a rate four times that of the general population. Following this, the next 5 years carry a 25% mortality (three times the population free of coronary heart disease). Following the infarction, half will have angina, 30% developing it anew. Reinfarctions will occur at a rate of 4% per year and half the recurrences

Table 1. Premeasured characteristics and 30-day to 5-year mortality following myocardial infarction recognized men only; Mantel-Haenszel, observed and expected deaths

Variable	No. deaths		Ratio O/E
	Observed	Expected	
Systolic blood pressure			
<130	4	8.67	0.46
130–153	9	12.11	0.74
>153	19	11.22	1.69
Diastolic blood pressure			
<85	11	14.40	0.76
85–94	9	0.78	0.83
>94	12	6.82	1.76
Pulse rate			
<70	6	7.01	0.86
70–79	5	10.91	0.46
>79	16	9.08	1.76
Hypertension			
None	5	8.62	0.58
Borderline	11	13.89	0.79
Definite	16	9.49	1.69
Left ventricular hypertrophy—ECG			
None	27	29.39	0.92
Questionable	1	0.98	1.02
Definite	4	1.63	2.45

$p < 0.07$ for left ventricular hypertrophy.

will be fatal. Congestive failure will occur at ten times, and strokes at five times the rates for the general population. The prognosis for angina pectoris is not much better. Yet, as grim as these prospects for the future might seem, the risk is not uniform and for some the prognosis is good. We need to know how to identify those in greatest jeopardy and what factors are likely to determine the risk (*Tables 1 and 2*).

Persons with the transient myocardial ischemia of a positive exercise ECG, ischemic resting ECG aberrations, or a silent infarction have the same poor prognosis as a recovered myocardial infarction victim.[2–4] They warrant the same concern as those with symptomatic coronary heart disease. Awaiting symptoms before instituting prophylactic treatment would appear imprudent.

In persons with atypical symptoms and physical findings during the attack, the exercise ECG and the coronary risk profile may help to clarify the problem.[5] Although the absence of such corroboration does not exclude the possibility of coronary heart disease, in their absence the prognosis is little different from that of the general population the same age.

Once coronary heart disease has become clinically manifest, the utility of conventional risk factor assessments, either to gage the prognosis or to identify risk factors which need

Table 2. Premeasured characteristics and mortality between 5 and 10 years following myocardial infarction recognized men only; Mantel-Haenszel, observed and expected deaths

Variable	No. deaths		Ratio O/E
	Observed	Expected	
Diastolic blood pressure			
<85	4	8.58	0.46
85–94	9	6.86	1.31
>94	7	4.56	1.54
Vital capacity/height			
<45	7	3.87	1.81
45–54	7	7.11	0.98
>54	5	8.02	0.62
Intraventricular block			
No	18	19.49	0.92
Yes	2	0.51	3.92

Table 3. Postmeasured characteristics and 5-year mortality following angina pectoris uncomplicated men only; Mantel-Haenszel, observed and expected deaths

Variable	No. deaths		Ratio O/E
	Observed	Expected	
Systolic blood pressure			
<130	2	5.15	0.39
130–153	6	8.53	0.70
>153	12	6.32	1.90
Diastolic blood pressure			
<85	5	9.39	0.53
85–94	7	5.58	1.25
>94	8	5.02	1.59
Hypertension			
Normal	3	6.41	0.47
Borderline	7	8.52	0.82
Definite	10	5.06	1.98
Heart enlargement (x-ray)			
None	10	14.36	0.70
Questionable	1	2.87	0.35
Definite	9	2.77	1.25
Left ventricular hypertrophy—ECG			
None	15	17.75	0.85
Questionable	1	1.08	0.93
Definite	4	1.17	3.42
Glucose intolerance			
No	14	17.72	0.79
Yes	6	2.28	2.63

correction, is less clear. The severity and persistence of symptoms, response to submaximal exercise testing, and physical findings of congestive heart failure help to identify those with multiple vessel disease and a worse prognosis.[6,7]

Noninvasive clinical indicators of poor cardiac status also have value. Persons with angina or myocardial infarction in the Framingham cohort who also had ECG abnormalities or enlargement of the heart on x-ray had a high risk of mortality. Some of the conventional risk factors also appear to have some utility in identifying coronary heart disease patients who are more likely to have recurrences, congestive heart failure, strokes, and die prematurely. Thus, blood pressure, glucose tolerance, and ECG-LVH are related to recurrence rates and mortality. Others have shown that subsequent rates of morbidity and mortality can be expected to mount in relation to these and other risk factors.[6–9]

Interpretation of the influence of risk factors on subsequent mortality once coronary heart disease becomes overt is complicated by the fact that the coronary event may alter the risk attribute. Thus, blood pressure may fall after infarction. After infarction, mortality is related to the preinfarction blood pressure level, but not to the postinfarction blood pressure. Those who sustain a substantial fall in blood pressure after infarction

Table 4. Postmeasured characteristics and 5-year mortality following myocardial infarction recognized men only; Mantel-Haenszel, observed and expected deaths

Variable	Deaths		Ratio O/E
	Observed	Expected	
Heart enlargement (x-ray)			
None	12	18.54	0.65
Questionable	6	5.51	1.09
Definite	14	7.95	1.76
Left ventricular hypertrophy—ECG			
None	25	29.57	0.85
Questionable	1	0.75	1.33
Definite	6	1.68	3.57

have a doubled risk of mortality over 5 years (*Tables 3 and 4*).

The utility of correcting these remains to be demonstrated, but seems rational. Relief of hypertension has been shown to reduce the risk of strokes and congestive failure, if not coronary mortality, in the coronary patient. A number of studies have shown that quitting cigarettes reduces coronary mortality in coronary patients to half that of those who continue to smoke.[10, 11] Reduction of overweight, although rational, is as yet of unproven efficacy, as is treatment of diabetes or hypercholesterolemia.

Consideration of radical remedies for coronary heart disease must take cognizance of the high remission rate for angina pectoris. About 30% of those with established angina will have a sustained remission. Those with intermittent angina (transient) are likely to have an improved prognosis as to survival and risk of a myocardial infarction.

In summary, an assessment of conventional risk factors for coronary heart disease does have some utility in identifying coronary patients who are more likely to have recurrences, strokes, and congestive heart failure as well as a shortened life span. The utility of correcting most of these remains to be demonstrated, but seems rational and without great hazard or inconvenience to the potential benefactor.

References

1. Sorlie P: Cardiovascular diseases and death following myocardial infarction and angina pectoris: the Framingham study. Section 32, 20 year follow-up (Kannel WB, Gordon T, eds) DHEW Publication No. (NIH) 77-1247, 1977.
2. Robb GP, Marks HH: The postexercise electrocardiogram in the detection of coronary disease; a long-term evaluation. Trans Assoc Life Ins Med Dir Am **45:** 81–98, 1962.
3. Kannel WB, McNamara PM, Feinleib M, et al: The unrecognized myocardial infarction: fourteen-year follow-up experience in the Framingham study. Geriatrics **25:** 75–87, 1970.
4. Kannel WB, Gordon T, Castelli WP, et al: Electrocardiographic left ventricular hypertrophy and risk of coronary heart disease. The Framingham study. Ann Intern Med **72:** 813–822, 1970.
5. Friedberg CK: Some comments and reflections on changing interests and new developments in angina pectoris. Circulation **46:** 1037–1047, 1972.
6. Bruschke AV, Proudfit WL, Sones FM Jr: Progress study of 590 consecutive nonsurgical cases of coronary disease followed 5–9 years. II. Ventriculographic and other correlations. Circulation **47:** 1154–1163, 1973.
7. Oberman A, Jones WB, Riley CP: Natural history of coronary artery disease. Bull NY Acad Med **48:** 1109–1125, 1972.
8. Coronary Drug Project Research Group: Factors influencing long-term prognosis after recovery from myocardial infarction; three-year findings of the Coronary Drug Project. J Chronic Dis **27:** 267–285, 1974.
9. Weinblatt E, Frank CW, Shapiro S, et al: Prognostic factors in angina pectoris; a prospective study. J Chronic Dis **21:** 231–245, 1968.
10. Mulcahy R, Hickey N, Graham I, et al: Factors influencing long-term prognosis in male patients surviving a first coronary attack. Br Heart J **37:** 158–165, 1975.
11. Wilhelmsson C, Vedin JA, Elmfeldt D, et al: Smoking and myocardial infarction. Lancet **1:** 415–420, 1975.