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Predictability of sudden death from resting electrocardiogram

Effect of previous manifestations of coronary heart disease[1]

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SUMMARY The sudden death rate from coronary heart disease over a mean period of 4 years was related to the electrocardiographic findings in 3 groups of subjects, survivors of myocardial infarction, employed men, and employed men with no symptoms or history of coronary disease. Within each group the sudden death rate correlated with the number of electrocardiographic findings, particularly Q, ST, and T wave items. However, between groups there were large differences in sudden death rates in subjects with the same findings. These were greatest in the case of ventricular conduction disturbances and disturbances of rhythm and rate which appeared to be benign in those free of symptoms but ominous after infarction. Findings predictive of sudden death were also predictive of non-sudden coronary deaths. It is concluded that the electrocardiogram is only one of several aids to the diagnosis and assessment of severity of disease and not a substitute. Prognoses derived from clinical case series are inappropriate to symptomless individuals in whom isolated electrocardiographic findings denote little increase in risk.

The resting electrocardiogram is used not only in the diagnosis of disease but in the assessment of its severity, and there may, therefore, be a temptation to use electrocardiographic findings as the sole basis for estimating prognosis. Despite the widespread use of this method of investigation opportunities to validate electrocardiographic predictions seldom arise, as they depend on the standardised recording and analysis of the electrocardiogram in large numbers of subjects whose fate is followed over a number of years. Several such studies have been reported, either on known sufferers from coronary heart disease (Shanoff *et al.*, 1966; Leren *et al.*, 1970; Connolly *et al.*, 1971, 1972; Coronary Drug Project Research Group 1972, 1973a), on occupational groups (Fisher and Tyroler, 1973), on residents of defined communities (Chiang *et al.*, 1969, 1970), or in those free of symptomatic coronary heart disease (Blackburn *et al.*, 1970). Comparison of the results from these different studies suggests that the significance of particular electrocardiographic findings varies with the population under investigation. In this report two parallel studies, in which subjects of similar age had their electrocardiographic findings and deaths coded by the same methods and the same investigators, have been used to make a direct comparison of the prognostic value of certain electrocardiographic findings in predicting sudden death in three groups of subjects. These were survivors of myocardial infarction, employed men in industry, and employed men who, at the time of recording of the electrocardiogram, had no history or symptoms suggestive of coronary heart disease. By these means it was possible to estimate how much electrocardiographic findings alone determined prognosis and how much it depended on a history or symptoms of coronary heart disease. In addition sudden and non-sudden coronary deaths were compared to see if they had the same electrocardiographic predictors.

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Patients and methods

The definitions of the study groups were as follows.

(1) *Postinfarct* Men and women residents of the London borough of Tower Hamlets aged less than 65 who survived an episode of definite or possible acute myocardial infarction and at-

tended the Tower Hamlets Coronary Project office for a 3-month postinfarction electrocardiogram between July 1970 and March 1973 (World Health Organization, 1971, 1976; Tunstall Pedoe, 1975, 1977; Tunstall Pedoe *et al.*, 1975).

(2) *Employed* Men employed in industry aged 40 to 59 who took part in the cardiovascular screening programme of the United Kingdom Heart Disease Prevention Project between April 1971 and March 1973 (Rose, 1970; World Health Organisation European Collaborative Group, 1974).

(3) *Symptomless* The above employed men *less* those who were scored positive in a self administered questionnaire for possible angina pectoris of effort or possible previous myocardial infarction (Rose and Blackburn, 1968) or who gave a history of diagnosed or suspected heart disease.

The method of recording the electrocardiogram was not identical in the two studies. The postinfarct patients had a 12 lead single channel record lasting 20 to 30 seconds which was analysed by one reader using the Minnesota code (Rose and Blackburn, 1968). The employed (and symptomless) men had a 12 lead, 3 channel, record lasting 20 seconds of which the limb leads were coded by 2 readers.

In both studies mortality was followed through local sources. In addition the subjects' record cards were marked at the National Health Service Register Centre so that copies of any death certificates received there would be forwarded. (The few cases in which this proved impossible are excluded from this report.) Further information on circumstances of deaths was obtained from medical and forensic sources. All deaths notified up to April 1976 were included in the analysis. The electrocardiograms from both studies were coded by members of the same team and the deaths were coded by the same observer, without knowledge of the electrocardiographic findings.

For the purposes of the main analysis sudden death was defined as death attributed to coronary heart disease occurring definitely or possibly within 24 hours of the onset of acute symptoms. The sudden death rate was the number of such deaths over the period of follow-up per hundred at risk at the start; the prevalence of an electrocardiographic finding was the percentage of positive electrocardiograms for any item; and the percentage of sudden deaths predicted was that percentage of the total that occurred in subjects with positive electrocardiograms. The relative risk was the sudden death rate in those coded positive for an electrocardiographic item *divided* by the rate in those coded negative; the attributable sudden death rate was the *difference* between these rates. Subjects in whom the graph could not be coded for a particular item were omitted from consideration of that item only, so that the totals varied slightly.

Results

(1) *Postinfarct* Of 311 patients, 137 were below 55 years and 174 aged 55 to 64 years; 256 were men and 55 women. There were 61 deaths during a mean follow-up period of 4·3 years, 13 from non-coronary causes and 48 from coronary heart disease of which 40 were classified as sudden, yielding a sudden death rate during follow-up of 12·9 per cent.

(2) *Employed* The 9166 men were fairly evenly spread over the age span 40 to 59 years. There were 222 deaths during a mean follow-up period of 4·0 years, 114 from non-coronary causes and 108 from coronary heart disease, of which 87 were classified as sudden, yielding a sudden death rate of 0·95 per cent during follow-up.

(3) *Symptomless* The 8228 men remaining after the exclusion of those positive on the screening questionnaire were responsible for 160 deaths during a mean period of follow-up of 4·0 years, 96 from non-coronary causes and 64 from coronary heart disease, of which 51 were classified as sudden, yielding a sudden death rate of 0·62 per cent during follow-up.

The frequencies of grouped electrocardiographic findings using the major Minnesota code classes are shown in Table 1, with the sudden death rates associated with them. The proportion of positive graphs and of sudden deaths so predicted is highest in the postinfarct patients and least in the symptomless, with the employed men intermediate. The attributable death rate follows a similar gradient but in most cases the relative risk of sudden death between those with and without an electrocardiographic item is greatest in the employed men.

In Table 2 some more specific electrocardiographic findings are analysed. In each case there are gross differences in their associated sudden death rates between the three groups of subjects, the postinfarct patients suffering a high mortality in association with intraventricular block or frequent ectopic beats, the employed men are again intermediate, but symptomless men, despite reasonable numbers coded positive, suffered no losses.

In Table 3 a simple analysis is made of combinations of electrocardiographic findings and their associated outcomes. Each group of subjects shows a gradation of risk of sudden death with increasing

Table 1 *Sudden death rate according to grouped electrocardiographic findings*

Minnesota codes (Rose and Blackburn, 1968)	*Group studied*	*Item positive*			*Item negative*		
		No. coded a	*Sudden deaths b*	*Sudden death rate b/a %*	*No. coded c*	*Sudden deaths d*	*Sudden death rate d/c %*
Q+QS patterns 1-1 to 1-3	Postinfarct	168	28	16·7	143	12	8·4
	Employed	151	9	6·0	9005	78	0·9
	Symptomless	84	2	2·4	8135	49	0·6
QRS axis deviation 2-1 to 2-2	Postinfarct	43	7	16·3	268	33	12·3
	Employed	346	3	0·9	8813	84	1·0
	Symptomless	310	2	0·6	7911	49	0·6
High amplitude R waves 3-1 to 3-3	Postinfarct	21	2	9·5	290	38	13·1
	Employed	234	4	1·7	8916	82	0·9
	Symptomless	209	2	1·0	8003	48	0·6
ST junction (J) and segment depression 4-1 to 4-4	Postinfarct	107	18	16·8	204	22	10·8
	Employed	265	15	5·7	8842	71	0·8
	Symptomless	191	4	2·1	7986	47	0·6
T wave items 5-1 to 5-3	Postinfarct	187	26	13·9	124	14	11·3
	Employed	646	26	4·0	8465	59	0·7
	Symptomless	491	10	2·0	7689	41	0·5
AV conduction defects 6-1 to 6-5	Postinfarct	12	1	8·3	299	39	13·0
	Employed	232	1	0·4	8916	85	0·9
	Symptomless	206	1	0·5	8004	49	0·6
Ventricular conduction defects 7-1, 7-2, and 7-4	Postinfarct	32	9	28·2	279	31	11·1
	Employed	226	4	1·8	8926	82	0·9
	Symptomless	189	0	0·0	8025	50	0·6
Disturbances of rate and rhythm 8-1 to 8-8	Postinfarct	41	8	19·5	270	32	11·9
	Employed	573	5	0·9	8588	82	1·0
	Symptomless	502	1	0·2	7721	50	0·6
Any codeable item	Postinfarct	275	39	14·2	36	1	2·8
	Employed	2066	35	1·7	7100	52	0·7
	Symptomless	1760	13	0·7	6468	38	0·6

Table 2 *Sudden death rate according to specific electrocardiographic findings*

Minnesota code item	*Group studied*	*No. coded +ve*	*Prevalence of +ve finding %*	*No. of sudden deaths in +ves*	*Sudden death rate % in +ves*	*% age of sudden deaths predicted*
Left bundle-branch block 7-1	Postinfarct	7	2·3	2	29	5
	Employed	48	0·5	2	4	2
	Symptomless	39	0·5	0	0	0
Right bundle-branch block 7-2	Postinfarct	2	0·6	1	50	3
	Employed	86	0·9	0	0	0
	Symptomless	75	1·0	0	0	0
Intraventricular block 7-4	Postinfarct	23	7·4	6	26	15
	Employed	90	1·0	2	2	2
	Symptomless	73	0·9	0	0	0
Frequent ectopic beats 8-1	Postinfarct	18	5·8	4	22	10
	Employed	132	1·4	3	2	3
	Symptomless	103	1·3	0	0	0
Atrial fibrillation 8-3	Postinfarct	6	1·9	2	33	5
	Employed	31	0·3	1	3	1
	Symptomless	24	0·3	1	4	2
Tachycardia 8-7	Postinfarct	2	0·6	1	50	3
	Employed	315	3·4	1	<1	1
	Symptomless	290	3·5	0	0	0
Bradycardia 8-8	Postinfarct	15	4·8	1	7	3
	Employed	90	1·0	0	0	0
	Symptomless	82	1·0	0	0	0

numbers of positive items on the electrocardiogram, but each shows a different distribution of prevalences of abnormality at screening and of the sudden death rates associated with each level of abnormality.

In Table 4 the outcome in subjects showing specific pairs of electrocardiographic findings is analysed. Comparison with Table 1 shows that there is an inconstant increase in the specificity of prediction of sudden deaths at the cost of a re-

Table 1 *continued*

ercentage revalence /(a+c)	*Percentage of sudden deaths predicted b/(b+d)*	*Relative risk of sudden death b/a / d/c*	*Attributable sudden death rate (b/a–d/c)%*
4·0	70	2·0	8·3
1·6	10	6·9	5·1
1·0	4	4·0	1·8
3·8	18	1·3	4·0
3·8	3	0·9	–0·1
3·8	4	1·0	0·0
6·8	5	0·7	–3·6
2·5	5	1·9	0·8
2·5	4	1·6	0·4
4·4	45	1·6	6·0
2·9	17	7·1	4·9
2·3	8	3·5	1·5
0·1	65	1·2	2·6
7·0	30	5·8	3·3
6·0	20	3·8	1·5
3·5	3	0·6	–4·7
2·5	1	0·5	–0·5
2·5	2	0·8	–0·1
0·3	23	2·5	17·1
2·5	5	1·9	0·8
2·3	0	0·0	–0·6
3·2	20	1·6	7·7
6·3	6	0·9	–0·1
6·1	2	0·3	–0·4
8·4	98	5·1	11·4
2·5	40	2·3	1·0
1·4	25	1·3	0·2

duction in the proportion of deaths so predicted. Again there is a pronounced gradient in the prevalence of these paired abnormalities and in the sudden death rate in those coded positive between the three different groups studied.

The data were re-examined using differing definitions of sudden death and contrasting deaths occurring more and less than 24 hours from the onset of an attack. In each group studied there was some tendency for the proportion of deaths that were sudden to be related to the presence and number of electrocardiographic findings at screening. This effect was not very pronounced and the particular electrocardiographic findings associated predominantly with very rapid death in a fatal attack were not consistent from group to group. Overall, therefore, there appeared to be no specific predictors for sudden as distinct from all coronary deaths. Deaths from non-coronary causes did not appear to be preferentially associated with particular electrocardiographic findings.

In this series of postinfarction subjects the women had a higher prevalence of electrocardiographic abnormalities than the men and suffered a rather higher sudden death rate for the same degree of abnormality. The numbers were too small for the difference to be statistically significant. By contrast there seemed to be little effect of age. This was not true of the employed and symptomless men, in whom there was a tendency for most electrocardiographic findings to be more common in those over 50 at screening and for these findings to be associated with a relatively higher sudden death rate. However, the gradient of risk of death with age was similar to that in those without electrocardiographic findings, but disappeared in those with multiple findings, who resembled the postinfarct cases in this respect.

Discussion

It was expected that the sudden death rate and the proportion of deaths attributed to coronary heart disease would be higher in survivors of recent myocardial infarction than in a mixed population, and higher in the latter than in those with no symptoms. The question under investigation, however, was whether the prognosis associated with electrocardiographic findings varies in populations with different sudden death rates. These results suggest that the answer is yes. Though the numbers on which prognoses are based in some of the Tables

Table 3 *Sudden death rate according to number of electrocardiographic findings*

No. of electrocardiographic findings	*Group studied*	*No. so described*	*Percentage prevalence*	*No. of sudden deaths*	*Sudden death rate %*	*Percentage of sudden deaths predicted*
None	Postinfarct	36	11·6	1	2·8	3
	Employed	7099	77·4	52	0·7	60
	Symptomless	6468	78·6	38	0·6	75
Any one	Postinfarct	57	18·3	4	7·0	10
	Employed	1563	17·0	12	0·8	14
	Symptomless	1389	16·9	6	0·4	12
Any two	Postinfarct	72	23·2	12	16·7	30
	Employed	368	4·0	14	3·8	16
	Symptomless	281	3·4	5	1·8	10
Three or more	Postinfarct	146	47·0	23	15·8	58
	Employed	136	1·5	9	6·6	10
	Symptomless	90	1·1	2	2·2	4

Table 4 *Sudden death rate according to paired electrocardiographic findings*

Minnesota codes	*Group studied*	*No. +ve*	*Percentage prevalence*	*No. of sudden deaths in +ves*	*Sudden death rate %*	*Percentage of sudden deaths predicted*
Q/QS patterns and ST and J depression	Postinfarct	72	23·2	13	18·1	33
1–1 to 1–3 plus	Employed	32	0·3	3	9·4	3
4–1 to 4–4	Symptomless	12	0·1	1	8·3	2
Q/QS patterns and T wave items	Postinfarct	126	40·5	19	15·1	48
1–1 to 1–3 plus	Employed	60	0·7	7	11·7	8
5–1 to 5–3	Symptomless	22	0·3	1	4·5	2
ST/J depressions and T wave items	Postinfarct	107	34·4	18	16·8	45
4–1 to 4–4 plus	Employed	249	2·7	15	6·0	17
5–1 to 5–3	Symptomless	177	2·2	4	2·3	8
Q/QS patterns and ventricular conduction	Postinfarct	21	6·8	7	33·3	18
1–1 to 1–3 plus	Employed	11	0·1	1	9·1	1
7–1, 7–2, and 7–4	Symptomless	6	0	0	0	0

are small and the multiple comparisons make tests of significance inappropriate, the tendency towards a better prognosis in symptomless subjects, whatever the electrocardiographic finding, is too consistent to be ignored. The electrocardiogram should not, therefore, be used to generate a prognosis without other data on the subject in whom it was recorded. Most, but not all, of the prognostic sting of electrocardiographic findings can be removed if the subject proves to be symptomless. The electrocardiogram is only one of several risk factors related to prognosis (Coronary Drug Project Research Group, 1973b; Rose *et al.*, 1977). This study, therefore, confirms the results obtained by collating separate previous reports. It is of some importance when an increasing proportion of electrocardiograms is being recorded on those free of symptoms, when reports may be made in isolation from clinical data or with the assistance of computers, and when both clinicians and computers are largely trained on hospital patients, but may be asked to report on subjects whose only complaint is electrocardiographic (Caceres, 1976; Bertrand, 1976; *Lancet*, 1976).

That the prognostic significance of an electrocardiographic finding is not determined uniquely by that finding suggests that electrical diagnoses are inadequate substitutes for a full clinical assessment and pathological diagnosis. The electrocardiographic predictors of sudden death revealed here do not appear to be specific for sudden death as distinct from death from coronary heart disease. The primary predictors appear to be those commonly accepted as indicators of ischaemia (Q, ST, and T findings). The secondary predictors, which appear to operate only in those with established ischaemia (ventricular-conduction disturbances and disturbances of rate and rhythm), appear to be non-specific indicators of the degree of muscle damage. This suggests that preoccupation with electrocardiographic phenomena should not detract from consideration of the underlying pathological process or consideration of other coronary risk factors.

These findings can also be used to demonstrate the likely results of screening different populations by electrocardiogram. In symptomless individuals the yield of positives is low as is the attributable sudden death rate so that, for example, Q, ST, and T findings each occur in a small percentage and are associated with attributable death rates of 1·5 to 1·8 per cent over 4 years. Such screening might be justified by the existence of a safe and effective treatment that removed the excess risk. However, even if it existed it would involve some 200 man-years of treatment for each one gained. ST and T wave findings are not necessarily repeatable and may be induced in some symptomless subject with normal coronary arteriograms by adrenaline infusion (Taggart *et al.*, 1977). The low attributable sudden death rates and observations such as these suggest that ST and T wave findings are not specific for coronary heart disease and that the results of one resting electrocardiogram in a symptomless individual may be insufficient grounds for stigmatising him or her and embarking on treatment.

It might be argued that different methods of recording and analysing both the electrocardiograms and the deaths would lead to different conclusions. For example Minnesota code 8–1, for frequent ectopic beats, includes any form of ectopic beat that occurs in 10 per cent of complexes, but discounts rarer ones which might in any case be missed in a resting record lasting half a minute or less. While longer records or more specific analysis might have modified the conclusions somewhat, this code was adequate to delimit an abnormally high risk group in the post-infarction subjects but one in which there were no

sudden deaths in the symptomless. It seems unlikely that greater sophistication in technique would narrow this gap appreciably. Similarly, the definition of sudden death used in the main analysis was kept loose to include the bulk of coronary deaths. A rigid one-hour definition excluded many cases where death was probably sudden but unwitnessed, or detailed time intervals were not provided, so that the number of events was considerably reduced. If the effect of smaller numbers was allowed for, a change to the more rigid definition of sudden death appeared not to affect the overall findings.

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