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Role of the Electrocardiogram in Identifying the Patient at Increased Risk for Sudden Death

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Published data dealing with the electrocardiogram as a means of identifying individuals at increased risk for sudden death are meager. The available information suggests that the sensitivity of the electrocardiogram in association with other signs of heart disease is relatively good and that this may vary with the severity of the underlying disease. In contrast, its specificity for sudden death is poor; many patients with abnormal electrocardiograms do not die suddenly or of cardiac causes. The prognosis of any electrocardiographic abnormality is that of the underlying disease.

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Traditionally, the electrocardiogram is used to aid in the diagnosis of patients with suspected heart disease or to detect changes in patients with known heart disease. It is rarely an independent prognostic variable, the usual statement being that "the prognosis of a given abnormal electrocardiographic pattern is that of the underlying heart disease."

In epidemiologic surveys designed to detect patients with cardiovascular disease, the electrocardiogram is used as a possible independent predictor for future heart disease or as a prognostic marker in patients with known heart disease. Few studies, however, deal specifically with the role of the electrocardiogram in identifying patients at risk for sudden death. Even fewer studies are aimed specifically at the individual who dies suddenly without antecedent symptoms or findings of heart disease but whose electrocardiogram is abnormal. This brief review deals specifically with the electrocardiogram as a predictor of sudden death.

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Prognostic Value of the Electrocardiogram

An abnormal electrocardiogram in an adult is most likely an acquired abnormality and thus indicative of clinically evident or silent myocardial disease. This statement is supported by electrocardiographic findings in 776 patients aged 25 years or younger admitted to a psychiatric hospital without evidence of cardiovascular disease and by the prevalence of electrocardiographic abnormalities in 671 patients older than 65 years of age. The prevalence of electrocardiographic abnormalities was strikingly different between the two groups, few abnormalities being noted in the young age group (1).

There is convincing evidence that patients with certain electrocardiographic abnormalities ultimately develop clinically evident myocardial disease; most often the disease is coronary heart disease (2). The electrocardiographic abnormalities include primarily intraventricular conduction defects, especially left bundle branch block, ST segment and T wave changes and left ventricular hypertrophy when both voltage and ST-T changes are present (2–6).

In the Framingham study (2), electrocardiographic evidence of left ventricular hypertrophy was present in 157 patients, and this abnormality preceded clinical evidence of coronary heart disease in 97 patients (61.7%). In general, the pattern of left ventricular hypertrophy was associated with a "very grave" prognosis. Patients with an unequivocal pattern of left ventricular hypertrophy, even when not associated with clinically evident coronary heart disease,

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congestive heart failure or rheumatic heart disease, had a 12 year mortality rate of 59%. In fact, it was suggested that the mortality rate in individuals with definite left ventricular hypertrophy is greater than that in patients surviving myocardial infarction. Of the 264 deaths, 44% were preceded by a definite or "possible" finding of left ventricular hypertrophy. There was no difference in prognosis between patients whose abnormal electrocardiogram was present on the first examination or those in whom it became abnormal during subsequent examinations (2).

Electrocardiogram as Predictor of Sudden Death

Of the 234 deaths attributed to coronary heart disease in the Framingham study (3), 109 were sudden. Among the latter, 60% of the patients had prior evidence of coronary heart disease. It was suggested that the appearance of an electrocardiographic pattern of left ventricular hypertrophy with both voltage and ST-T changes in a patient with one or more conventional risk factors for coronary heart disease suggests the presence of ischemic heart disease. Such patients have a fivefold greater risk of sudden death than do those without such an electrocardiographic pattern.

In the Tecumseh study (7), there were 98 deaths from coronary heart disease, of which 45 were sudden. Among the latter, 38 of the patients (84.4%) had an abnormal electrocardiogram. Intraventricular conduction defects, left ventricular hypertrophy and old myocardial infarction were particularly frequent in patients who died suddenly. Only 7 of 2,456 patients without electrocardiographic abnormalities died suddenly during a 6 year follow-up period (7).

In another 4 year follow-up study from England (5), designed specifically to correlate incidence of sudden death with evidence of heart disease, 18,705 men were classified into three groups: 1) 311 patients who recovered from myocardial infarction, 2) 9,166 employed men, and 3), 8,228 men who had no evidence of heart disease on screening. The total number of deaths caused by coronary heart disease and, specifically, the sudden deaths in each of the three groups were as follows: group 1 had 48 deaths, of which 40 were sudden; group 2 had 108 deaths, of which 87 were sudden and group 3 had 64 deaths, of which 51 were sudden. Within each of the three groups, sudden death correlated with electrocardiographic findings, particularly with significant Q waves, ST segment and T wave changes. The electrocardiographic findings were similar to those from other studies. However, the prognosis for subjects with the same electrocardiographic abnormalities varied among the three groups, being much better in the group without evident heart disease. This was true for total deaths as well as sudden deaths. The authors concluded that "the tendency toward a better prognosis in asymptomatic subjects, whatever the electrocardiographic findings, 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 is recorded."

The Canadian Air Force study (8) was designed to evaluate the predictive value of the electrocardiogram for sudden death in the absence of preexisting manifestation of heart disease; it included 3,983 men, all former Air Force pilots, pilots in training or licensed pilots who were followed up for 30 years. Of this group, 70 individuals died suddenly without antecedent evidence of clinical heart disease. Fifty (71.4%) of the 70 had electrocardiographic abnormalities; these included 22 (31.4%) with ST segment and T wave changes, 11 (15%) with ventricular ectopic impulses, 9 (12.9%) with left ventricular hypertrophy with voltage criteria only, 5 (7.1%) with left bundle branch block and 4 (5.7%) with abnormal left axis deviation. None had right bundle branch block. All electrocardiographic abnormalities except left axis deviation and right bundle branch block were significant predictors of sudden death.

In the vast majority of patients dying suddenly, the cause is severe coronary heart disease (9–11). This is true for both those with known coronary heart disease and those considered clinically "normal." In a large autopsy study, Spain et al. (11) reported that 90% of sudden fatalities resulted from coronary heart disease. In the Tecumseh study (7), only 1 of the 45 patients who died suddenly was free of hypertension, diabetes, coronary heart disease or electrocardiographic abnormalities. Necropsies performed in 25 of 70 deaths reported in the Canadian Air Force study (8) disclosed that 24 (96%) of the 25 men who died had "severe coronary atherosclerosis" as evidenced by lesions reducing coronary artery lumen by greater than 75% and the other had moderate (50 to 75%) narrowing by atherosclerosis.

The issue of intraventricular conduction defects and sudden death deserves a brief comment. The significance of bundle branch block as an independent marker for sudden death is controversial. Its prevalence in the general population is relatively low and, thus, numerically it is not a major determinant of sudden death. However, an extensive and detailed review of bundle branch block and sudden death suggests that the presence of chronic bundle branch block, with or without an accompanying fascicular block, is associated with a higher mortality rate and incidence of sudden death than are other electrocardiographic abnormalities, and this is especially true in the presence of coronary heart disease (12). In a follow-up study (mean 20 months) of 189 patients with chronic bundle branch block, sudden deaths occurred in 21 patients, 19 of whom had coronary heart disease. More often, however, the bundle branch block is associated with heart failure and cardiomegaly, and the electrocardiogram may be only a marker of the severity of the underlying disease. The prognostic value of bundle branch block in survivors of acute myocardial infarction differs from that in patients with other forms of heart disease. There

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is considerable evidence that bundle branch block appearing during acute myocardial ischemia, either transient or persistent is accompanied by a significantly increased incidence of late sudden deaths (12).

Comment

Longitudinal large scale studies have inherent limitations. The electrocardiograms are usually recorded annually or perhaps less often. Thus, there is no assurance that the electrocardiogram was normal before sudden death. The assumption of a "clinically" normal heart is often made on the basis of a questionnaire and is another compounding variable. The diagnosis of ischemic heart disease may be difficult to make when an experienced physician is personally examining the patient, and diagnoses arrived at from questionnaires are sometimes erroneous.

Summary

From the relatively meager published data it can be concluded that:

1) Although the sensitivity of the electrocardiogram alone or in association with other signs of coronary heart disease for sudden death is reasonably good, the specificity for sudden death is poor since many patients with an abnormal electrocardiogram do not die suddenly—or otherwise—of cardiac causes. There is also evidence to suggest that the sensitivity of an abnormal electrocardiogram for sudden death varies with the severity of the underlying disease.
2) More than 90% of sudden deaths are associated with severe coronary heart disease. Thus, the electrocardiogram acts as a marker for sudden death by identifying the patient with coronary heart disease.

References

1. Fisch C. The electrocardiogram in the aged. Cardiovasc Clin 1981;12:65–74.
2. Kannel WB, Gordon T, Offutt D. Left ventricular hypertrophy by electrocardiogram. Prevalence, incidence, and mortality in the Framingham study. Ann Intern Med 1969;71:89–105.
3. Kannel WB, Doyle JT, McNamara PM, Quickenton P, Gordon T. Precursors of sudden coronary death. Factors related to the incidence of sudden death. Circulation 1975;51:606–13.
4. Schneider JF, Thomas HE Jr, Kreger BE, McNamara PM, Kannel WB. Newly acquired left bundle-branch block: the Framingham study. Ann Intern Med 1979;90:303–10.
5. Pedoe HDT. Predictability of sudden death from resting electrocardiogram. Effect of previous manifestations of coronary heart disease. Br Heart J 1978;40:630–5.
6. Blackburn H, Taylor HL, Keys A. XVI. The electrocardiogram in prediction of five-year coronary heart disease incidence among men aged forty through fifty-nine. Circulation 1970;41/42(suppl I):I-154–61.
7. Chiang BN, Perlman LV, Fulton M, Ostrander LD, Epstein FH. Predisposing factors in sudden cardiac death in Tecumseh, Michigan. A prospective study. Circulation 1970;41:31–7.
8. Rose G, Baxter PJ, Reid DD, McCartney P. Prevalence and prognosis of electrocardiographic findings in middle-aged men. Br Heart J 1978;40:636–43.
9. Rabkin SW, Mathewson FAL, Tate RB. The electrocardiogram in apparently healthy men and the risk of sudden death. Br Heart J 1982;47:546–52.
10. Peters RW, Scheinman MM, Modin GM, et al. Prophylactic permanent pacemakers for patients with chronic bundle branch block. Am J Med 1979;66:978–85.
11. Spain DM, Bradess VA, Mohr C. Coronary atherosclerosis as a cause of unexpected and unexplained death: an autopsy study from 1949-1959. JAMA 1960;174:384–8.
12. Fisch GR, Zipes DP, Fisch C. Bundle branch block and sudden death. Prog Cardiovasc Dis 1980;23:187–224.