

Regular Review

Lessons from ambulatory electrocardiography

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In the 1950s Norman "Jeff" Holter, the American physicist, developed and gave his name to the technique of recording physiological signals from ambulant patients.¹ Holter monitoring has now come to be synonymous with ambulatory electrocardiography. In hospital practice the term ECG (electrocardiogram) taping is used, often with the prefix 24 hours to indicate the usual duration of the record. Ambulatory electrocardiography is the preferable term, however, because it covers variations in the way electrocardiographic signals are recorded (for example, by radio-telemetry or through the telephone rather than, as is usual, on to a tape cassette) and those monitors that are geared to record only when the patient experiences symptoms and then triggers the "event recording" button on the machine.

The commonest problem in interpreting ambulatory electrocardiographic records is recognising the signals that are due to artefact generated from poor contact between the skin and the recording electrode. Most artefacts are easily identified by their high frequency but others mimic arrhythmias such as ventricular tachycardia. Machine artefacts may also mislead—for example, slowing of the cassette may erroneously suggest bradycardia. Thus the first lesson of interpreting the results of ambulatory electrocardiography is to be wary of findings which do not fit with the clinical picture. It is also important to appreciate the extent to which electrocardiographic recordings vary in otherwise normal people, the poor correlation between the symptoms and arrhythmias, the changes that may lead to sudden death, and the signs of occult ischaemia.

Normal variation

A normal person may be defined as someone who has not been adequately investigated. Apply this aphorism to ambulatory electrocardiography and it becomes apparent that if anyone is monitored carefully sooner or later an extrasystole or pause is recorded. So what is normality? Clarke and colleagues examined 101 volunteers. Fifteen were excluded because of probable medical abnormalities but on the ambulatory electrocardiograms of the remaining 86, 10 had disturbances of rhythm which at the time were widely believed to be of "serious prognostic significance."²

Many hundreds of apparently normal people have been studied since then,³ and our ideas of normality have changed. Bradycardias are common in young people, especially at night, and in a study of 50 medical students 12 had sinus rates of 40 a minute or less; three had second degree atrioventricular block.⁴ Sinus pauses, first degree atrioventricular block, and nodal escape rhythms are not unusual and may be regarded as normal in youth. Sinus pauses should excite

suspicion in older people, however, and should never exceed 2.5 seconds at any age.³ Complete heart block as an unexpected finding has been found only once in a study of 722 people.³

Atrial extrasystoles are common and are not related to age. But runs of atrial extrasystoles become increasingly prevalent with age, and paroxysmal atrial fibrillation is an occasional finding in normal people over the age of 50. Ventricular extrasystoles are rare in schoolchildren,⁵ but their prevalence increases with age to reach 75% in people aged 60-79.⁶ They are also more frequent and complex in older people and in one study five out of 260 asymptomatic people over the age of 40 had episodes of ventricular tachycardia.⁶

Do these findings merely indicate occult heart disease? Common sense suggests not, and this view is supported by studies such as that of Kennedy and colleagues, who investigated 25 people with complex ventricular extrasystoles and found that only six had coronary artery disease.⁷ Long term follow up of asymptomatic people with ventricular extrasystoles has confirmed that such individuals have a good prognosis.⁸ Thus ventricular extrasystoles may merely be an indicator of aging in the way that high vagal tone may result in bradycardia in younger people.

Symptoms and arrhythmias

Some people tolerate serious arrhythmias without comment, others report every extrasystole, and it is because a person's awareness of his heart beat is so unpredictable and arrhythmias are so common that only those that give rise to symptoms should be considered to be the cause of the patient's complaint. A diary is, therefore, an essential adjunct to ambulatory electrocardiography. Without it misinterpretation is easy. More than one cardiac pacemaker has been mistakenly implanted for the treatment of dizzy spells in a patient with a physiological bradycardia. Conversely, a normal electrocardiogram and a blank diary cannot be taken to exclude paroxysmal tachycardia.

Sadly only a minority of ambulatory electrocardiograms give clear cut answers. In 165 patients complaining of palpitation a correlation between symptoms and arrhythmia was obtained in only 43.⁹ This does not suggest that ambulatory electrocardiography is a waste of time, but in many patients, especially those described as having transient non-focal neurological abnormalities, it is a question of balancing probabilities and frequently undertaking more than one day's monitoring.¹⁰ In the final analysis diagnosis may rest primarily on the history rather than the results of the test.¹¹

Repeated monitoring may uncover another lesson: the spontaneous variation of arrhythmias. The incidence of

ventricular extrasystoles may vary by fivefold or sixfold in two 24 hour electrocardiograms. One important implication of this variability is that an antiarrhythmic drug may only be said to be effective if the number of extrasystoles is reduced by about 80% in 24 hours.

Sudden death

Several people have died while undergoing ambulatory electrocardiography. Most were suffering from ischaemic heart disease and, as we already knew from resuscitation attempts in similar patients, most collapsed with ventricular fibrillation. The electrocardiogram rarely showed signs of acute infarction and thus corroborated the pathological and other evidence that suggests that sudden death is due to a primary electrical disturbance. Ventricular fibrillation is usually preceded by ventricular tachycardia, and this is often heralded by increasingly frequent and complex ventricular extrasystoles.¹²⁻¹³ The trigger factor may be an early coupled beat.¹⁴⁻¹⁵ These antecedent arrhythmias differ from those observed in asymptomatic patients with ischaemic heart disease. The ventricular tachycardia is faster and lasts longer, and the pattern of extrasystoles suggests considerable electrical instability in the hours before death, which in the reported cases tended to occur in the afternoon and evening.

One alarming feature of these deaths is their association with treatment with antiarrhythmic drugs—particularly digoxin and quinidine.¹²⁻¹⁶ The association may not be necessarily causal, however, for sinister arrhythmias tend to occur in patients with severely damaged ventricles, who are already at risk. Nevertheless, it does suggest that it is unwise to prescribe antiarrhythmic drugs for patients with poor left ventricular function, especially if the arrhythmia is not causing symptoms.

Asystole is obviously the ultimate arrhythmia but a progressive bradycardia is less common than ventricular fibrillation. This may be because most ambulatory electrocardiographic studies of patients who have collapsed have been in those with ischaemic heart disease and abnormal ventricular rhythms. Bradycardia may precede asystole

when the cause of death is non-cardiac, as in the one reported case of sudden death during ambulatory electrocardiography when necropsy showed pulmonary embolism.¹⁷

Occult ischaemia

Modern ambulatory electrocardiographic recorders can faithfully reproduce not only the QRS complex but also the low frequency components of the electrocardiogram such as the ST segment. Shift of the ST segment often but not invariably indicates cardiac ischaemia. Episodes of raising of the ST segment have been recorded during "normal" nocturnal bradycardia in younger men and are unlikely to be of pathological importance.¹⁸⁻¹⁹ Intermittent ST segment depression is different: its frequency, magnitude, and duration are greater in patients with severe coronary disease²⁰; it correlates well with reduced myocardial perfusion and with ST segment depression induced by exercise testing²⁰⁻²¹; it is reduced by antianginal medication²²; in unstable angina it predicts prognosis; and it is abolished by coronary artery bypass surgery.²³ Finally, it is rare in normal people, although there is some disagreement on this point.¹⁸⁻²⁰⁻²⁴

What does emerge is that many patients with ischaemic heart disease and angina have ST segment depression in their ambulatory electrocardiogram not only during episodes of pain but at other times as well. This discovery provokes a number of questions. Do the painless episodes represent ischaemia of small segments of myocardium²⁵ or an increase in the patient's threshold to pain?²⁶ And do they matter, given that myocardial infarction is silent in over a quarter of cases²⁷ and that the outlook in these patients is as bad as in those who are symptomatic? Should treatment of patients with angina aim at abolishing all ischaemic episodes or just relieve symptoms?²⁸ These questions are fundamental but more work is needed before they may be answered.

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