Clinical Topics

Role of 24-hour ambulatory electrocardiographic monitoring in a general hospital

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Summary and conclusions

During 1976, 24-hour ambulatory electrocardiographic (ECG) monitoring was available to all physicians at this hospital, and 281 patients were investigated by 322 recordings. Cardiac arrhythmias requiring treatment were detected in 100 patients (36%). Some presented after symptoms such as faintness, giddiness, palpitations, collapse, or fits, but ominous arrhythmias were also found in asymptomatic patients. A demand pacemaker was implanted for episodic sinoatrial or atrioventricular conduction disorder in 30, while 70 patients (25%) required antiarrhythmic drug treatment for ventricular or atrial tachyarrhythmias.

Facilities for ambulatory 24-hour ECG monitoring are necessary in any large hospital, and precise diagnosis in most of our patients studied could not have been achieved by any other investigation.

Introduction

The development of methods for long-term electrocardiographic (ECG) monitoring[1] has led to the use of ambulatory techniques for detecting transient cardiac arrhythmias. A wide range of symptoms may result from such transient arrhythmias.[2-5] Application of these methods to epidemiological studies has shown that ventricular ectopic beats are associated with sudden death and coronary artery disease.[6-9]

We tried to evaluate the diagnostic yield produced by routine ambulatory 24-hour ECG recordings when applied to patients in a general hospital. Our preliminary report[10] suggested that 30% of 420 patients investigated in the cardiac clinic over a two-year period required antiarrhythmic drug treatment or a permanent pacemaker to alleviate or abolish important cardiac arrhythmias. During 1976 investigation by ambulatory 24-hour ECG was freely available to all physicians in this hospital. We analysed recordings from all patients investigated during this period to establish the exact proportion requiring treatment and the range of arrhythmias responsible for their symptoms.

Patients and methods

Patients were drawn from those attending clinics at this hospital during 1976. Most of the 281 patients studied were outpatients, and the remainder were ambulant inpatients. Ages ranged from 13 to 89 years. Indications for 24-hour ECG were generally those shown in table I. Antiarrhythmic agents were withdrawn for a minimum of five days before recording when practicable. Tape-recording was carried out for at least one 24-hour period, and 322 tapes were obtained. Two systems were used. Most of the tapes were recorded as two simultaneous ECG channels on Avionics reel-to-reel Electrocardiocorders (model 425) and analysed on the Avionics 660 Dynamic Cardioscanner. Some were made as single ECG channels on the Medilog 4-24 cassette tape recorder (Oxford Instruments) and analysed on the Neilson Pathfinder rapid analysis system (P J Reynolds Ltd) using a PD-2 playback unit (Oxford Instruments). To this system a purpose-built electronic counter was added for assessing and measuring the number of ventricular arrhythmias. The accuracy of this system has been described.[10]

TABLE I—*Indications for 24-hour ambulant ECG in various conditions*

	Indications for 24-hour ambulant ECG
Symptomatic conditions:	
Collapse attacks	Particularly when clinical or ECG findings suggest cardiac disease—for example, ventricular aneurysm, inappropriate sinus bradycardia
Palpitations	
Dizziness	
Asymptomatic conditions:	
Abnormal rhythm on routine ECG	For example, sinus rate <50/min; ventricular ectopic beats
Known arrhythmogenic condition	For example, mitral valve prolapse; Wolff-Parkinson-White syndrome
Implanted pacemaker	For example, possible pacemaker failure or resumption of stable rhythm
Known coronary artery disease:	
Myocardial infarction	(*a*) To detect late ventricular arrhythmias in patients believed to be at risk; (*b*) to assess reliance on temporary pervenous pacing system
Angina	When arrhythmia has been suspected of precipitating angina
Ischaemia	As supplement to exercise testing
Controlled evaluation of antiarrhythmic drug efficacy	See ref [21]

Ventricular arrhythmias were graded as follows according to Lown's classification[11]: grades 0-1, unifocal ventricular ectopic beats absent or of low frequency (<30/h); grade 2, frequent unifocal ventricular beats (>30/h); grade 3, multifocal ectopic beats; grade 4 (*a*), ventricular ectopic pairs; grade 4 (*b*), salvoes of ventricular tachycardia comprising three or more consecutive ectopic beats (rate >100/min); and grade 5, early-cycle (R-on-T) beats.

Results

Of the 281 patients, 248 showed cardiac arrhythmias that could be grouped as being predominantly ventricular or supraventricular or as examples of sinoatrial disorder or atrioventricular nodal conduction disorder. Thirty-three patients had arrhythmia-free recordings.

Ventricular arrhythmias—Ventricular ectopic activity was found in 163 patients (table II). Ectopic beats were frequent (Lown grade 2) or showed bigeminy in 40 patients, and were multifocal or paired (grades 3 and 4 (*a*)) in 68; 22 patients had episodes of ventricular

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TABLE II—*Ventricular arrhythmias graded according to Lown's classification*[11] *detected by 24-hour ambulant ECG in 163 patients*

	No of patients
Grade 1—Infrequent unifocal ventricular ectopic beats (<30/h)	57
Grade 2—Frequent unifocal ventricular beats (>30/h) including bigeminy	40*
Grades 3 and 4 (a)—Multifocal or paired ventricular ectopic beats, or both	68*
Grade 4 (b)—Ventricular tachycardia; three or more consecutive ventricular ectopic beats (>100/min)	22

*Grade 2 and grades 3 and 4 (*a*) not mutually exclusive.

FIG 1—Episode of ventricular tachycardia of torsade-de-pointes morphology in girl aged 16.

tachycardia (grade 4 (*b*)) (fig 1). Fifty-seven patients had infrequent ventricular ectopic beats (grade 1). Twelve of the 22 patients with ventricular tachycardia presented as the result of dizziness, syncope, or a fit. Six of the patients had associated sinoatrial disorder. The routine ECG was normal in 10 patients but gave a clue to the presence of underlying cardiac disease in the shape of ventricular ectopic beats, ischaemia, previous infarction, cardiac hypertrophy, or sinus bradycardia in the remaining 12. Of the total number with ventricular arrhythmias, 58 patients received beta-blocking drugs, mexiletine, disopyramide, or procainamide as antiarrhythmic treatment in direct consequence of the findings.

Supraventricular arrhythmias—Eighty patients had isolated supraventricular ectopic activity. A further 82 patients had both ventricular and supraventricular ectopic beats. Complex atrial tachyarrhythmias were found in 35 patients (fig 2). Twenty of these patients had intermittent atrial fibrillation, 13 supraventricular tachycardia, one atrial flutter, and one paroxysmal atrial tachycardia with block. In this group with tachyarrhythmias only, symptoms were not limited to palpitations but included breathlessness, angina, dizziness, faintness, and syncope. Treatment was instituted with various drugs, including digoxin, beta-blocking agents, disopyramide, quinidine, verapamil, and amiodarone.

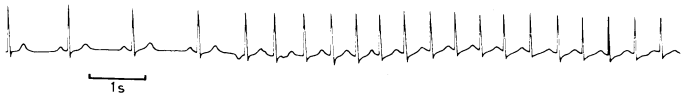

FIG 2—Episode of supraventricular tachycardia initiated by atrial extrasystole in man aged 68.

Sinoatrial disorder—In 43 patients 24-hour tape-recordings suggested sinoatrial disorder. At least two of the three recognised criteria for the disorder were present—namely, sinoatrial block, inappropriate sinus bradycardia, or supraventricular arrhythmias (fig 3). Syncope was by far the commonest presentation (22 patients), but a further four had fits of grand-mal type. One patient with syncope also complained of associated chest pain, and one had angina alone. Seven complained of palpitations alone. One patient was investigated because of recurrent cerebral emboli and bradycardia. Only four were asymptomatic, investigations being undertaken because of abnormal routine ECGs in three (two with left bundle-branch block) and recent myocardial infarction associated with heart block and sinus arrest in the other. All ages from the second to ninth decades were represented, with a mean of 50 years. Many young patients were seen in this group, which contrasts with the 72 patients in our pacemaker clinic who have received a pacemaker for sinoatrial disorder (mean age 63 years).

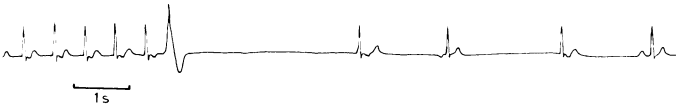

FIG 3—Termination of nodal tachycardia in same patient as fig 2. Fortuitous ventricular premature beat interrupts re-entry, and nodal and atrial escape beats precede first sinus beat (right side of tracing).

Sinoatrial disorder was associated with ventricular arrhythmias. Twenty-one patients had ventricular ectopic activity that was not associated with prolonged sinus arrest and subsequent ischaemia[12] but was characterised by frequent, premature ectopic beats. Patients with ventricular arrhythmias tended to be older. Thus 13 out of 25 patients aged 55 or over had Lown grades 2 or 4 (*a*) arrhythmia, while five had episodes of ventricular tachycardia. Of the 18 aged under 55, only one had grade 2 arrhythmia, and one had episodes of ventricular tachycardia. Twenty-two of the patients received permanent demand pacemakers. In the remaining 21 the main feature was tachyarrhythmias, particularly in younger patients, or the cases were still under review.

Atrioventricular block—Eight patients received pacemakers after intermittent atrioventricular block (fig 4) had been detected. Two patients had Mobitz type I (Wenckebach) second-degree atrioventricular block, and three had Mobitz type II block. In two further patients the pattern of right bundle-branch block with left axis deviation was seen in their routine ECG, while their 24-hour tape showed intermittent complete heart block. One patient complained of loss of balance, and the tape disclosed episodes of complete heart block interrupting stable atrial fibrillation. The two patients with the Wenckebach pattern and two of the three with Mobitz type II block also had syncope. The other patients were investigated during convalescence after myocardial infarction.

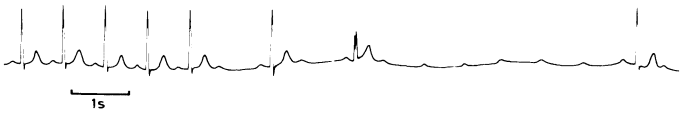

FIG 4—Episode of Mobitz type II atrioventricular block in woman aged 56.

Failure to achieve diagnosis—It is impossible to assess the number of false-negative recordings, but these obviously occur. One patient[5] had six 24-hour tape-recordings before intermittent Wenckebach atrioventricular block was discovered. A further patient's condition remained undiagnosed despite a history of rapid palpitations that precipitated anginal pain and were sometimes followed by sudden syncope. Three 24-hour tape-recordings showed infrequent ventricular ectopic beats only. Her symptoms were controlled by empirical treatment with beta-adrenergic blocking agents. In neither of these instances, however, nor in other undiagnosed cases were routine ECGs or other investigations helpful in diagnosis.

Pacemaker response—The incidence of false-positive abnormalities shown by 24-hour ECG monitoring may partly be assessed by the response to pacemaker implantation. No patient whose rhythm disturbances were detected by 24-hour ECG during the study who received a pacemaker continued to have syncopal attacks.

Recording quality—Recording quality with the Avionics system was good, and despite its weight and reel-to-reel recording system, two ECG channels could be analysed simultaneously. The standard Oxford Instruments system provides one ECG channel and is also more liable to artefact. Advantages of this system include its smaller size, simple cassette recording, and lower cost. First-class recording quality is essential for accurate diagnosis of sinoatrial disorder because of the importance of P-wave position and morphology, and the Avionics system was superior in this respect. The Pathfinder analyser is probably the most accurate device for quantifying ventricular arrhythmias that is currently available, and can be used with either of the above systems.

Discussion

These findings suggest that many of our outpatients required treatment for undetected arrhythmias. Symptomatology is protean,[2–5] and close co-operation with neurological colleagues is essential for investigating difficult cases. Ventricular ectopic activity was common. Patients might present with collapse or a fit precipitated by ventricular tachycardia and yet be unaware of palpitations.

Epidemiological studies have shown the prognostic importance of ventricular arrhythmias in patients with coronary artery disease and also in the apparently healthy population.[7 13–16] In addition, the presence of ventricular arrhythmias may be

independent of other risk factors as a cause of sudden death,[13] and the more severe grades of such arrhythmias are probably more ominous.[6] There is dispute over the incidence and importance of arrhythmias in the normal population,[17] but our findings show that severe ventricular tachyarrhythmias may often be found in outpatients. It is these patients, many of whom are already known to have coronary artery disease, for whom treatment is appropriate. Patients who have been routinely investigated after myocardial infarction were not included in our study, but such a policy might prove valuable in identifying a group with a high risk for convalescent mortality.[15 16]

The role of 24-hour ambulatory ECG monitoring in detecting myocardial ischaemia has been controversial.[18–20] The frequency-response of the Avionics system is comparable with direct-writing ECG recorders, and it has been successfully used in our department for objectively monitoring anginal attacks and their response to beta-adrenergic blocking treatment.[21]

Supraventricular arrhythmias were common in our patients. Thirty-five patients (12·5%) were found to have complex arrhythmias with attendant symptoms such as breathlessness, angina, or distressing palpitations. Patients with intermittent atrial fibrillation also risk developing emboli, and anticoagulation should be considered in these cases. The expected association of supraventricular arrhythmias with age and ventricular ectopic activity was seen. Fewer of these patients required treatment than those with ventricular arrhythmias because the indications for treatment were usually based on symptoms, since there is no enhanced risk of sudden death.[7] Supraventricular rhythm disturbances often merged with the constellation of arrhythmias that comprise sinoatrial disorder (sick sinus syndrome). In addition to many obvious examples of multiple conduction-system disease the age-related association with ventricular arrhythmias is interesting, and antiarrhythmic treatment after pacemaker implantation may have to be directed against ventricular as well as supraventricular tachyarrhythmia.

We seem to have uncovered a younger population of patients with sinoatrial disorder than expected from our overall experience of this condition. In 1976 the mean age of patients was 50 compared with one of 63 in patients who had had pacemakers implanted for sinoatrial disorder in 1970-7. It is our policy to institute permanent pacing for patients with symptoms and two of the three main features of the disorder—namely, inappropriate sinus bradycardia, supraventricular tachyarrhythmias, and sinoatrial block. No pharmacological or electrophysiological test reliably predicts sinoatrial disorder,[22] and symptoms are the important determinant for treatment. Accurate diagnosis by 24-hour ambulatory ECG monitoring may well lead to more insertions of permanent pacemakers in the UK, where the implantation rate is low compared with other countries.[23]

Our results establish that routine use of 24-hour ECG recordings in a general hospital will show that over a third of patients with tachyarrhythmias or conduction disturbances require treatment with antiarrhythmic drugs or permanent pacing systems. Precise diagnosis of the cardiac rhythm disturbance allows specific treatment to be chosen and undoubtedly reduces morbidity and mortality in selected patients. We conclude that 24-hour ambulant ECG recordings are an essential investigation in a modern general hospital.

We thank Miss F Weaver for help with the tape-recording in most of these patients.

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ONE HUNDRED YEARS AGO Elizabeth P, thirty-four years of age, was admitted into the East Suffolk Hospital, under the care of Mr Branford Edwards, on the 8th instant. She was suffering from fistula in ano, and was stated to have been of very intemperate habits. Two days after admission, she was brought into the theatre for the purpose of having the fistula operated upon. She was very nervous and greatly dreaded the operation, which she was strongly advised to undergo without anaesthesia; but this she positively refused to do. Accordingly, chloroform was administered, in quantities of half-a-drachm at a time, on a few folds of lint. Her pulse was rather feeble and quick, conditions which, in the absence of other symptoms, were attributed to her fear of the knife. The chloroform was slow in its effect; but, beyond considerable struggling and excitement at the end of the first stage, there were no unusual symptoms. As soon as anaesthesia was barely produced, not more than three drachms having been inhaled, the administration was discontinued. The character of the pulse had not materially altered, and the breathing, though somewhat rapid, was fair. The patient was now turned on her left side for the convenience of the operator, the fistula was examined, and a director inserted. Just as the division of the fistula was being performed, about two minutes after the withdrawal of the anaesthetic, the patient gave a sharp scream, the pupils dilated, urine was passed involuntarily, the face became pale, and, simultaneously, the heart's action and the pulse suddenly ceased, and, after a few moments, the breathing stopped also. Artificial respiration upon Howard's plan was immediately commenced, fresh air in abundance was admitted into the room, interrupted shocks from a coil were applied over the heart, brandy was injected *per rectum*, and the right median basilic vein was opened. A few feeble respiratory movements occurred once or twice, but no heart-sounds could be detected. After more than an hour had elapsed, the attempt was abandoned as hopeless. At the *post mortem* examination, nineteen hours after death, the heart, as seen *in situ*, was firmly collapsed and very pale, and covered with a thick layer of fat. On removal, the organ weighed nine ounces and three-quarters, and its cavities were quite empty. The walls were remarkably thin and very pallid. Microscopical examination revealed well-marked fatty degeneration of the muscular fibres. There was a distinct patch, apparently atheromatous, on the mitral valve, and the base and transverse part of the arch of the aorta were atheromatous over a considerable extent. The other organs were very congested, but otherwise healthy. At the inquest, a verdict of "death from the action of chloroform on a diseased heart" was returned. (*British Medical Journal*, 1878.)