

CARDIAC ARRHYTHMIAS

CARDIAC SYNCOPES IN PATIENTS WITH SINUS RHYTHM

By E. Sandoe, B. Sigurd, G. Jensen, and J. Meibom

In 1827 and 19 years later in 1846 Adams and Stokes respectively reported on—I quote from the paper of Stokes—"cases of permanent slow pulse in which the patient suffered from repeated cerebral attacks of an apoplectic character though not followed by paralysis", observations which according to Stokes "were published with view of drawing the attention of the Profession to a combination of cerebral and cardiac phenomena, of which our knowledge are still imperfect".

In the light of our present knowledge—and expressed in the terminology of cardiology to-day—the patient of Adams and the patients of Stokes had chronic third degree a-v block and cerebral attacks caused by paroxysms of ventricular stand still, extreme bradycardia or—may be—ventricular fibrillation.

Adams Stokes syndrome is now widely known, and the cardiac nature of cerebral attacks which occur in combination with 3° a-v block will—and should—easily be recognized by most clinicians. But what is the incidence of chronic 3° a-v block in the total population of patients with cerebral attacks of dysrhythmic origin? How often does the clinician have this easily understood and easily remembered diagnostic guidance in form of a slow arterial pulse at his disposal in a case of cardiac syncope or black out episodes?

According to the information included in Fig. 1 chronic 3° a-v block is present in only one third of all cases with cardiac syncope or black out.

The figure indicates prevalence of chronic 3° a-v block in a consecutive series of 182 patients. The patients are cases with chronic or intermittent tendency to cardiac syncope or black out or intermittent tendency to cardiac syncope or blackout episodes seen during a 9 year period from 1963 to 1972 in medical department B, University Clinic of Copenhagen. The series does not include cases of acute myocardial infarction, electrolyte disturbances or drug intoxication.

It appears from the figure, that 65% of the patients, 118 out of the 182, had preserved a-v conduction between attacks. My lecture today is based on an analysis of these 118 patients, who in spite of predominantly sinus rhythm or supraventricular rhythm had paroxysms of dysrhythmias causing fainting and/or black-out.

ECG monitoring revealed that the cerebral symptoms were due to paroxysms of ventricular standstill secondary either to attacks of 3° a-v block as shown in the upper tracing of Fig. 2 or to attacks of 3° s-a block as shown in the tracing in the center of the figure, or the cerebral symptoms were caused by paroxysms of ventricular fibrillation as shown in the tracing in the lower part of the figure.

67 patients (57%) fainted due to paroxysms of 3° a-v block, 46 (39%) fainted or had blackouts due to paroxysms of 3° s-a block, and 5 patients only, fainted due to paroxysms of ventricular fibrillation. Most of the recorded dysrhythmias causing blackout or fainting lasted from 4-20 seconds, a few for a longer period of time. Most cerebral attacks started suddenly without preceding sensations of disturbed cardiac function due to paroxysms of 3° s-a block. Finally, 5 patients fainted due to paroxysms of ventricular fibrillation as indicated in the lower part of the figure. Most of the recorded dysrhythmias causing fainting or black out, lasted from 4-20 seconds, a few for a longer period of time. 3 patients with paroxysmal ventricular fibrillation had to be resuscitated by DC countershock once or twice.

Most cerebral attacks started suddenly without preceding sensations of disturbed cardiac function, dyspnoea or anginal pain. An exception was the patients with paroxysmal 3° s-a block in whom the black out episodes in about half the cases were preceded by a period of palpitations.

The following part of my lecture focuses on the two groups of patients, the patients with paroxysmal 3° a-v block and the patients with paroxysmal 3° s-a block, but I shall return to a brief account of the patients with paroxysmal ventricular fibrillation at the end of my lecture.

Sex ratio and age distribution of the two groups differed significantly (Fig. 3).

The group of patients with paroxysmal 3° a-v block (upper part of the figure) showed conditions comparable to what have been described in series of patients with chronic 3° a-v block, i.e. a male preponderance with a male/female ratio of 2.5/1, and a mean age for onset of symptoms about 65 years in men, and 75 years in women.

The patients with paroxysmal 3° s-a block (lower part of the figure) presented an equal sex distribution and a mean age for onset of symptoms of about 60 years in both sexes.

The length of the upper bar in the two diagrams (Fig. 4) indicates an equally high prevalence—it is about 30%—of arteriosclerotic heart disease in the two groups. The diagnosis of arteriosclerotic heart disease was based on a history of typical stress-induced anginal pain or on a former episode of acute myocardial infarction.

The first uncoloured part of the lower bar in each diagram indicates patients without cardiac complaints, and without cardiac enlargement or other clinical manifestations of cardiac disease except for their cerebral attacks and some ecg changes, which will be described later. Most remarkably, these cases of what may be called "normal hearts" constitute about 1/4 of the patients in each group.

Some type of interventricular conduction disturbances was consistently present in a major part of the patients with paroxysmal 3° a-v block, more exactly in 56 of the 67 patients, or in 83%. Altogether, interventricular conduction disturbances were observed in 91% of the patients.

Right bundle branch block, RBBB in the figure (Fig. 5) was the most frequent finding. It often occurred in combination with left anterior hemiblock, LAH, more rarely with left posterior hemiblock, LPH. Left bundle branch block, LBBB, was observed in altogether 25% of the patients.

A-v conduction disturbances were also frequent findings as shown in (Fig. 6). However, they were not observed as frequently or consistently as the just mentioned interventricular conduction disturbances and, completely normal a-v conduction was recorded at one or several occasions in 70% of the patients. It might be added that paroxysms of 3° a-v block followed by an immediate establishment of an escape rhythm—and consequently not leading to fainting—have been recorded episodically during long term ECG-monitoring in 54 of the 67 patients.

More than half the patients with paroxysmal 3° s-a block had normal ECGs at most recordings. About 25% only, showed interventricular or a-v conduction disturbances. The most frequently observed ecg abnormalities were atrial flutter, fibrillation or tachycar-

**CARDIAC SYNCOPE AND/OR BLACK OUT
PREVALENCE OF CHRONIC 3° A-V BLOCK
(182 Patients)**

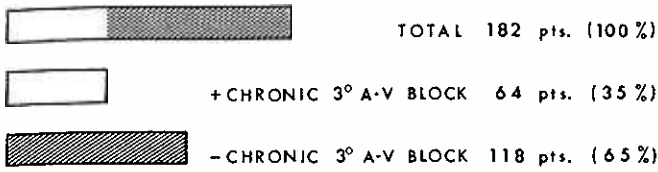


Fig. 1.

**CARDIAC SYNCOPE AND/OR BLACK OUT
PREDOMINANTLY SINUS RHYTHM* BETWEEN ATTACKS
(118 patients)**

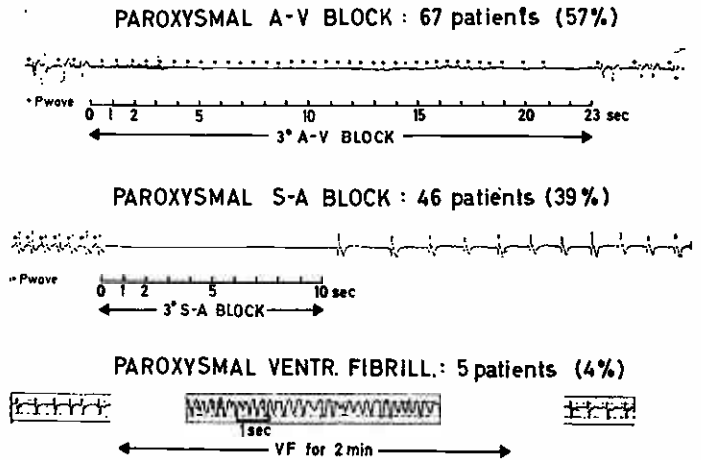


Fig. 2.

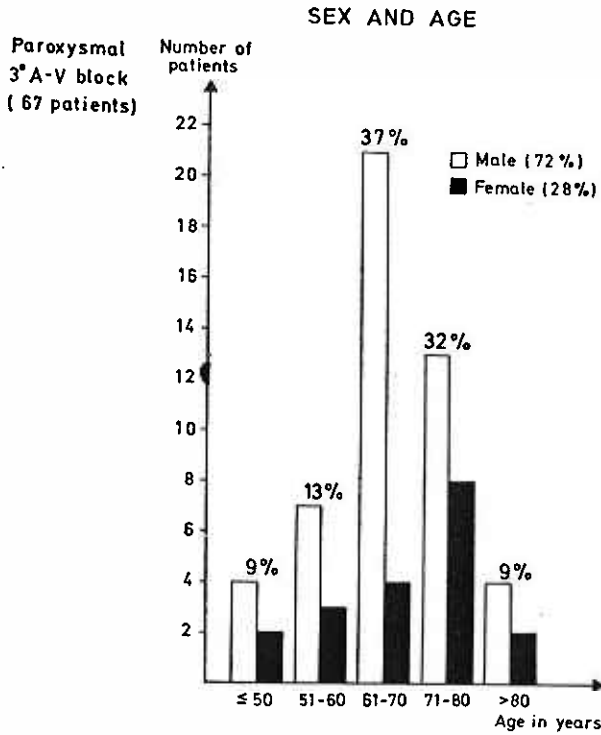


Fig. 3.

ASSOCIATED HEART DISEASE - HEARTSIZE - C.H.F.

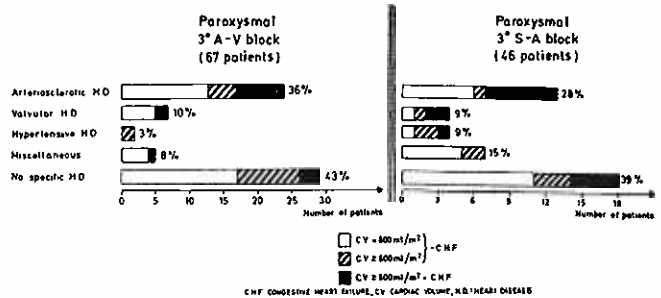


Fig. 4.

**PAROXYSMAL 3° A-V BLOCK
INTERVENTRICULAR CONDUCTION IN SINUSRHYTHM
(67 patients)**

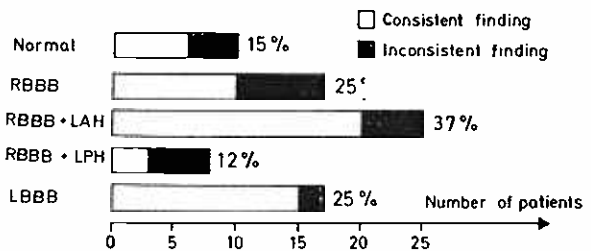
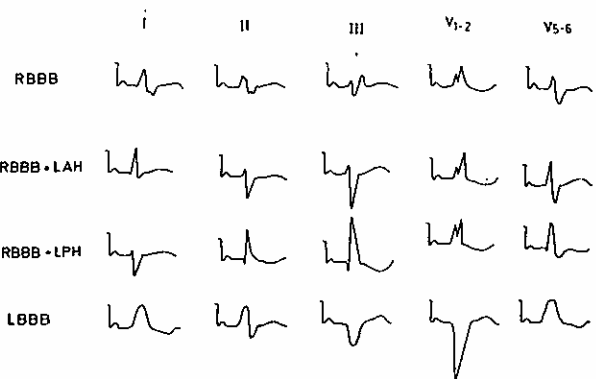
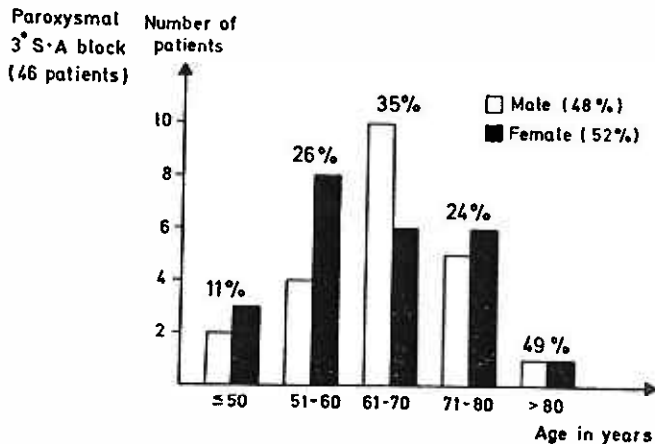


Fig. 5.



PAROXYSMAL 3° A-V BLOCK
A-V CONDUCTION IN SINUSRHYTHM
(67 patients)

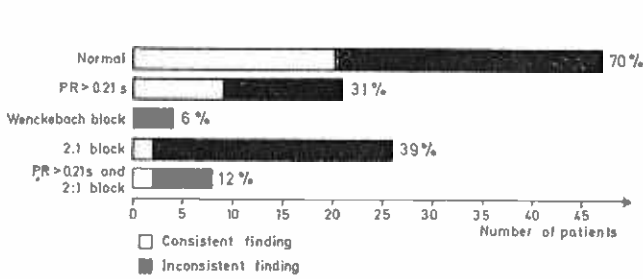


Fig. 6.

PAROXYSMAL 3° S-A BLOCK
ECG between attacks
(46 patients)

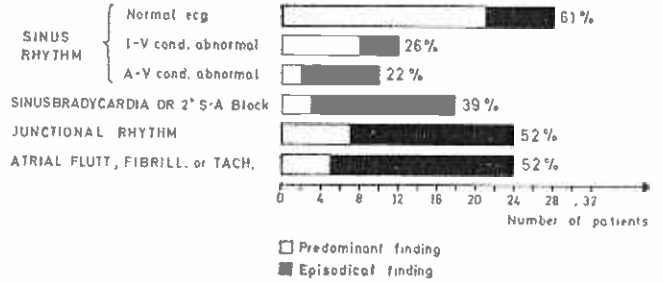


Fig. 7.

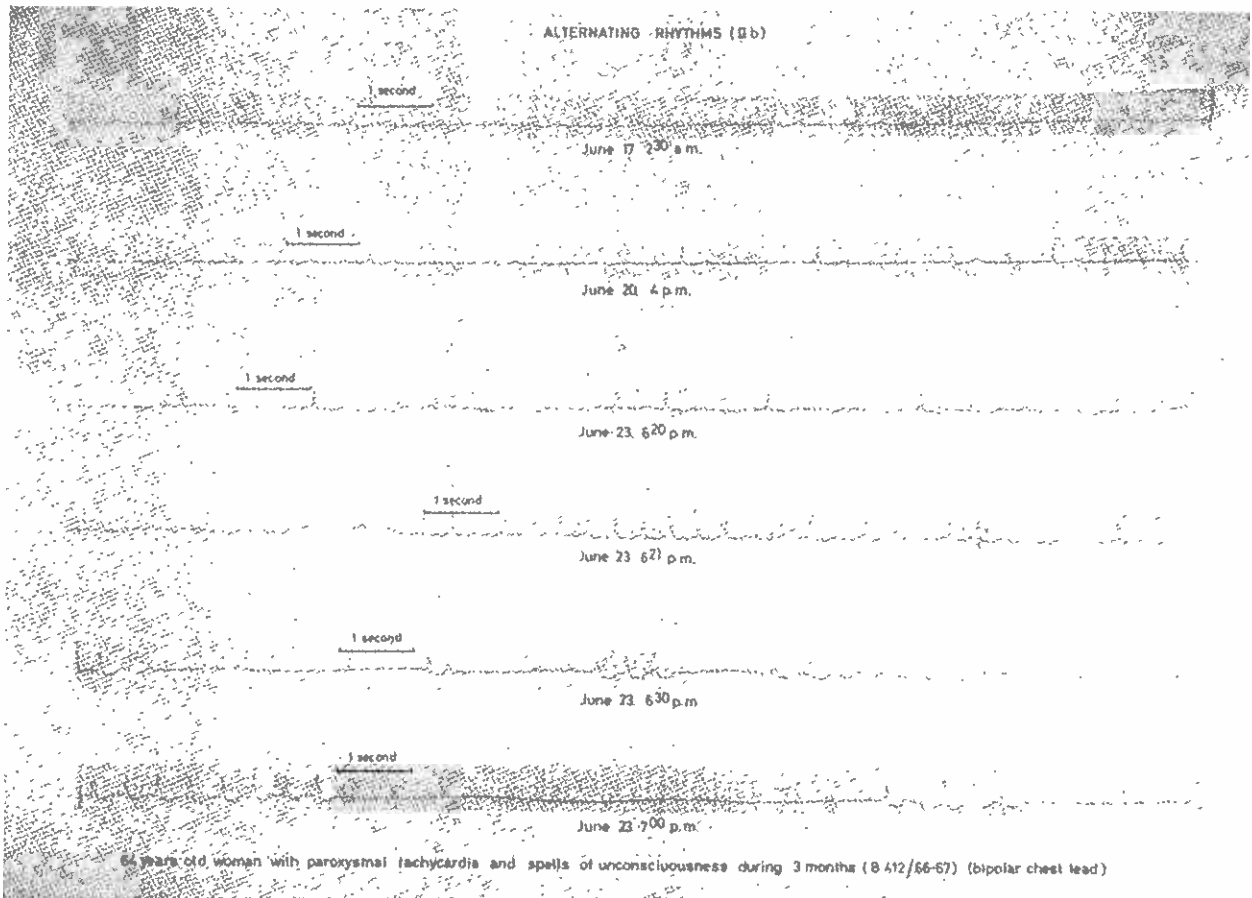


Fig. 8.

dia or, junctional rhythm or, sinusbradycardia which were found consistently in 12 patients (26%) and episodically, in more than half the patients (Fig. 7).

About half the patients with paroxysmal 3° s-a block and ventricular stand-still of seconds' duration, preceded by sensations of palpitations and of rapid pulse. The ECG in (Fig. 8) reveals the reason for their complaints. It appears that the patient had frequent bouts of atrial flutter often followed by s-a block and ventricular stand-still of seconds' duration.

The patients with paroxysmal ventricular fibrillation formed an inhomogeneous group which included two young adults, one case of aortic stenosis and one of non obstructive cardiomyopathy and, three children, two girls without definite signs of cardiac disease, and one boy with non-obstructive cardiomyopathy.

The three children all presented histories of stress-induced faintings. Their ECG's at rest revealed no signs of ectopic activity, but heavy work on a bicycle ergometer resulted in short episodes of ventricular tachycardia or bursts of multifocal ventricular ectopics as shown in the upper two tracings of (Fig. 9). The stress-induced tachycardia usually changed to normal sinus rhythm in a few minutes or less and, most im-

portant from a diagnostic point of view, the ectopic activity was never felt by the patients. Occasionally, the activity might rapidly convert to ventricular fibrillation as shown in the lower part of the figure.

In conclusion, it has been shown that about 2/3 of patients with cerebral attacks due to paroxysms of malignant dysrhythmias have sinus rhythm or at least supraventricular rhythm in between attacks and, that the dysrhythmias which provoke attacks are usually not felt by the patient and, that the cerebral attacks in one fourth of the cases may remain the only clinical manifestation of cardiac disease. Consequently, a diagnosis of paroxysmal malignant dysrhythmia ought to be considered in any obscure case of repeated fainting or blackout not followed by neurological drop out symptoms.

Differential diagnosis may be difficult, but may be facilitated by one or, preferably, repeated ECG-recording(s).

80-90% of patients with paroxysmal 3° a-v block show interventricular conduction disturbances of the bundle branch type, and a fair number of them a-v conduction disturbances in addition. Normal eeg is recorded in about half the patients with paroxysmal 3° s-a block, but repeated recordings will usually reveal episodes of atrial flutter, tachycardia or fibrillation, or of sinus bradycardia and 2° s-a block or of junctional rhythm. In patients with paroxysmal ventricular fibrillation, diagnosis may be extremely difficult, but ECG-monitoring performed during strenuous exercise may reveal multifocal ventricular ectopic activity, and this test should be considered obligatory in any patient with a history of stress-induced syncope of unknown etiology. However, the exercise test may provoke life-threatening attacks of ventricular fibrillation and should be performed only with a DC defibrillator standing by, ready for immediate use.

Continuous ECG-recording for a period of time, hours, days or weeks depending on attack rate, may be necessary to record the dysrhythmic paroxysms causing the cerebral attack, as this usually lasts for 5-20 seconds only. Long term ECG-monitoring may also reveal episodes of 3° a-v block with escaperhythm, shorter lasting paroxysms of s-a block or of multifocal ventricular activity etc., all of them dysrhythmias which not by themselves lead to cerebral symptoms, but which nevertheless provide evidence—although not confirmative—for a diagnosis of cardiac syncope or blackout.

Finally, I shall give a brief preliminary report of the therapeutical results achieved in the 118 patients with fainting or blackout episodes due to paroxysmal malignant dysrhythmias.

Implantation of demand pacemakers was performed in all 67 patients with paroxysmal 3° a-v block and in 38 of the 46 patients with paroxysmal 3° s-a block. A part from periods of technical problems due to electrode displacement, battery failure etc., pacemaker treatment brought cerebral attacks to an end in all but one patient. In four out of the five patients with paroxysmal ventricular fibrillation, beta-blocking agents reduced the incidence of attacks significantly. In one case finally, a patient with aortic stenosis, attacks did not stop on treatment with antiarrhythmic drugs but an operation with insertion of a Starr Edward valve combined with continued quinidine treatment proved successful.

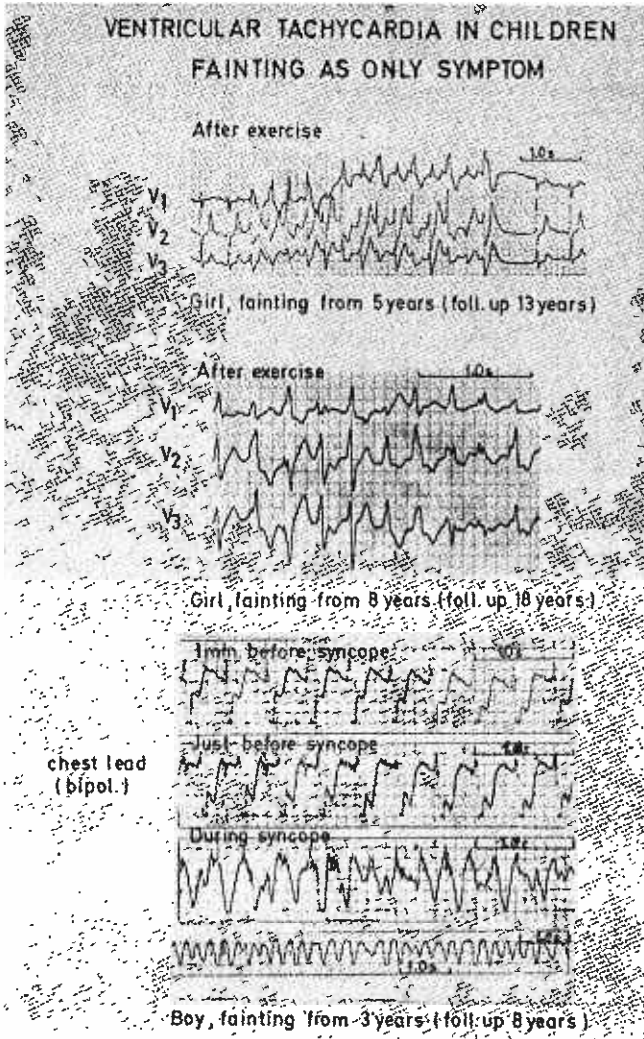


Fig. 9.