Angina pectoris with syncope due to paroxysmal atrioventricular block: role of ischaemia

Report of two cases

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Two patients who experienced angina pectoris resulting in syncope, caused by paroxysmal atrioventricular block, are reported. In both cases circulatory arrest occurred at the peak of the anginal attack. It was related in one case to ventricular standstill and in the other to runs of ventricular tachycardia (wave bursts 'torsades de pointe'). In both cases abolition of the syncopal attacks was achieved by insertion of a permanent demand pacemaker. These observations illustrate one of the possible mechanisms of sudden death in patients with coronary disease. They stress the primary role of acute ischaemia in the genesis of arrhythmias both during an attack of angina and the course of asymptomatic atherosclerotic heart disease.

The occurrence of syncope caused by paroxysmal atrioventricular (AV) block during attacks of angina pectoris in patients with ischaemic heart disease is rare. To our knowledge only 5 documented reports have been published so far. We thought it relevant to describe two new cases, which demonstrate one of the possible mechanisms of sudden death in patients with atherosclerotic heart disease.

Case reports

Case 1

A 78-year-old man presented with a past history of hypertension and mild renal insufficiency. In 1969 he experienced for the first time an attack of angina pectoris. Since 1972 syncope frequently occurred at the peak of anginal pain, at rest or on exercise. The patient complained of typical constrictive pain, then fainted. Syncope lasted less than one minute, and was associated with urinary incontinence. Neurological examination and two encephalograms revealed no abnormality. The patient was referred to us in January 1973 because of the recent recurrence of syncopal attacks. Physical examination was non-contributory except for a grade 2/6 systolic murmur at the apex and a moderate cardiomegaly on the chest x-ray film. The electrocardiogram showed sinus rhythm with right bundle-branch block and left anterosuperior hemiblock (Fig. 1). Paroxysmal AV block was suspected and the patient was placed under continuous electrocardiographic monitoring. The next day at 5.45 a.m. he was awakened by a severe retrosternal constrictive pain similar to the previous ones,

then lost consciousness for 45 seconds. The electrocardiogram recorded during syncope (Fig. 2) showed complete AV block with prolonged ventricular standstill (3.5 sec). A permanent demand pacemaker (Stanium Monopolar) was implanted using the jugular vein. During a nine-month follow-up period the patient is doing well: he is free of any syncope though he does experience episodic anginal pain.

Summary A 78-year-old man with coronary insufficiency and bifascicular block (right bundle-branch block and left anterosuperior hemiblock) experienced, at the peak of anginal pain, syncope caused by paroxysmal AV block and ventricular standstill. Relief from syncopal attacks was obtained after insertion of a permanent demand pacemaker.

Case 2

An 80-year-old woman presented with a past history of hypertension and mild cardiac failure. Since 1971 she repeatedly experienced short attacks of thoracic constrictive pain immediately followed by dizziness or syncope. The patient was referred to us in May 1973 for cardiac evaluation. Physical examination was non-contributory except for a grade 1/6 midsystolic murmur at the apex. The basic electrocardiogram showed sinus rhythm with normal PR interval and left bundle-branch block. The patient was placed under continuous electrocardiographic monitoring. On several occasions the tracing showed asymptomatic episodes of high degree AV block (Fig. 3). The day after referral the patient had thoracic constrictive pain identical with the previous ones and then lost consciousness. The electrocardiogram

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FIG. 1 Case 2. 9 January 1973. Sinus rhythm is present with normal PR interval and right bundle-branch block, with left anterosuperior hemiblock.

FIG. 2 Case 1. 10 January 1973. 5.45 a.m. During a syncopal episode which occurred at the height of an anginal attack complete atrioventricular block with prolonged ventricular asystole (3.5 sec) is recorded from the electrocardiogram.

FIG. 3 Case 2. 11 May 1973. In the absence of anginal pain an asymptomatic high grade atrioventricular block with occasional capture is recorded.

FIG. 4 Case 2. 12 May 1973. During an attack of angina pectoris followed by syncope, wave bursts ('torsades de pointe') appear.

recorded during the syncopal attack (Fig. 4) showed a high grade AV block, and, for the first time, runs of ventricular tachycardia (wave bursts: 'torsades de pointe' (Dessertenne, 1966)). The next day a permanent demand pacemaker (Stanium Monopolar) was implanted using the jugular vein. During a six-month follow-up the patient is doing well: she is free of any syncope though does experience a few attacks of angina pectoris.

Summary An 80-year-old woman with coronary insufficiency and left bundle-branch block experienced syncopal attacks at the peak of anginal pain. Between the attacks of angina continuous electrocardiographic monitoring showed episodes of high degree AV block which remained asymptomatic. During an anginal attack syncope occurred and simultaneously wave bursts ('torsades de pointe') were recorded on the electrocardiogram. Relief from syncopal attacks was obtained after insertion of a permanent demand pacemaker.

Discussion

Asymptomatic atrioventricular (AV) conduction disturbances are known to occur in about 20 per cent of cases in the acute phase of myocardial infarction. They are more frequent in posteroinferior (15%) than anterior (5%) infarctions (Himbert and Lenègre, 1967). As a rule, they are due to reversible ischaemia of the AV node and are transient. Asymptomatic AV conduction disturbances are probably caused by a similar mechanism, but are quite unusual during an attack of angina. Auzépy *et al.* (1962) reported one case out of 195. A few other reports (Prinzmetal *et al.*, 1959; Bouvrain, Fortin, and Coumel, 1963; Nadal-Ginard and Cardenas, 1973; Oliva, Potts, and Pluss, 1973) include cases seen during variant angina pectoris described by Prinzmetal *et al.* (1959); in these cases, the degree of block varied from a second-degree block (Mobitz I (Nadal-Ginard and Cardenas, 1973) or 2:1 (Bouvrain *et al.*, 1963) to a complete heart block (Nadal-Ginard and Cardenas, 1973).

The occurrence of a syncopal attack during an episode of angina is rare (Gallavardin, 1922; Golden, 1944). Pathogenesis of the syncope include (Chiche, 1972): increase in vagal tone; cerebral ischaemia resulting from the reduction in myocardial performance due to acute ischaemia (Chiche, 1972); arrhythmias as especially observed in Prinzmetal's angina: sinoatrial block (Dorra *et al.*, 1968); ventricular fibrillation (Raynaud *et al.*, 1969; Schwartz, Schwedel, and Schwartz, 1966), and paroxysmal AV block which is discussed here.

Syncopal attacks caused by paroxysmal AV block during angina, to our knowledge, so far have been mentioned in only 5 documented reports (Gallavardin, 1922; Botti, 1966; Schwartz *et al.*, 1966; Raynaud *et al.*, 1969; Faivre *et al.*, 1972). Gallavardin (1922) was the first to report these in what is now a classical description: 'Every time the patients have violent attacks of angina they are subject to fits of dizziness or actual syncope. Slowing of the pulse cannot always be recorded because once the attack ends everything returns to normal. Undoubtedly it is only during an attack that one can notice the slowing or maybe the disappearance of the pulse... The linking of the fits of dizziness or syncope to conduction disturbances is still more likely to be true with the finding of an unusually prolonged a-c interval in the venous pulse tracing'. More recent cases (Botti, 1966; Schwartz *et al.*, 1966; Raynaud *et al.*, 1969) have made it possible to state precisely the mechanism of the circulatory arrest: ventricular pause (Case 1), or 'wave bursts' ('torsades de pointe') (Case 2). This last term refers to runs of ventricular tachycardia which have been individualized according to their particular morphology (Dessertenne, 1966). When recorded on three simultaneous leads the electrocardiogram evidences remarkable periodical changes in QRS amplitude (gradual decrease then increase) and orientation (the complexes seem to 'twist' them-

selves around the baseline). In American reports 'torsades de pointes' are usually referred as 'transient ventricular fibrillation' (Schwartz, Orloff, and Fox, 1949) or 'chaotic ventricular tachycardia' (Scherf, Cohen, and Shafiiha, 1967).

In our patients, syncope caused by paroxysmal AV block occurred at the peak of typical anginal attacks which did not have the features of Prinzmetal's angina. Anginal attacks were unlikely to have been caused by AV block itself, as they preceded the occurrence of syncope (Case 1) or were not always followed by syncope (Case 2). They imply both a failure in the sinus impulse conduction and a delay in the junctional escape resulting in a ventricular pause, or allowing wave bursts to appear. The arrhythmia is likely to result from a simultaneous decrease of the blood supply in the atherosclerotic arteries of sinus and AV nodes (James, 1969). In such cases the frequent onset of atrial arrhythmias (atrial fibrillation (Bouvrain *et al.*, 1963), sinus bradycardia (Schwartz *et al.*, 1966), or tachycardia (Botti, 1966)) together with AV block may be explained on this basis. AV conduction disturbances occurring during the anginal pain emphasize the preponderant role of myocardial ischaemia in their genesis. Gallavardin (1922) already stated, 'We can postulate that the transient ischaemic disturbance which is the cause of the attack of angina spreads to the His bundle.' In a recent report Nadal-Ginard and Cardenas (1973) referred to it as 'angina of the AV node or of the His bundle'. Ischaemia is known to result in severe arrhythmias as it may result in a transient decrease of left ventricular performance (Chiche *et al.*, 1973; Amsterdam, 1973; Haiat *et al.*, 1973): in the very first hours of the acute phase of myocardial infarction, arrhythmias occur at a time when ischaemia is far more important than necrosis; in many cases of sudden death (Lenègre, 1958; Killip, 1971) myocardial lesions are discrete or moderate.

The reason why syncopal attacks occurring as a result of paroxysmal AV block are relatively rare during anginal episodes remains unclear. It is likely, however, that in many cases the conduction disturbances result in sudden death.

In our two cases, the syncope no longer occurred after insertion of a permanent demand pacemaker. The decision to use a pacemaker was even clearer as an underlying intraventricular conduction disturbance (bifascicular block (Case 1), left bundle-branch block (Case 2)) was present. It is assumed that prevention of sudden death was so realized.

These two unusual case reports emphasize the primary role of acute or subacute ischaemia in the course of atherosclerotic heart disease with or with-

out angina (as anginal pain is a late and/or inconstant sign of myocardial ischaemia). As clearly shown, ischaemia may lead to severe arrhythmias and must be treated so in an endeavour to prevent sudden death (Chiche, 1973).

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