Syncope after effort

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Summary
A 29-year-old man developed recurrent syncope following exertion. Cardiac investigations revealed no evidence of structural heart disease, but during exercise testing, in the recovery phase, he sustained a bradycardia and then asystole for a prolonged period. Before cardiac massage could be instituted a tonic-clonic fit occurred, and this initiated a return to sinus rhythm. His symptoms were abolished following the implantation of a dual-chamber pacemaker.

Keywords: syncope; asystole; convulsion; pacemaker

Effort syncope is a well-recognised complication of any pathology which obstructs central blood flow, eg, aortic stenosis. Syncope occurring after effort is also recognised but more unusual. We report here a case with the unique characteristic that convulsions restarted the patient’s heart.

Case report
A 29-year-old man first lost consciousness after unpacking his hang-glider, which he had just carried up a mountain. He had two subsequent less severe episodes following sexual intercourse. The fourth episode occurred after swimming six lengths of an Olympic-sized swimming pool, trying to go faster with each length. He sat on the side of the pool and lost consciousness. He was ‘revived’ but collapsed again on attempting to stand and was incontinent of urine. No jerking movements were noted. He was otherwise very well with no relevant family or past history. He was taking no medication.

Examination revealed a very fit young man weighing 79 kg. There were no untoward cardiovascular signs apart from a soft ejection murmur and an even softer immediate diastolic murmur. Neurological examination was normal. His resting electrocardiogram (ECG) and electroencephalogram were normal, and a cranial magnetic resonance imaging scan showed no focal abnormality. Echocardiography revealed a mildly eccentric aortic valve with a normal aortic root, normal left ventricular dimensions and function, and a normal mitral valve.

Exercise testing confirmed the clinical impression of physical fitness. He was able to reach 17 minutes, 16 seconds of the Bruce protocol with a normal haemodynamic response; his heart rate increased from 51 to 163 beats/min and his blood pressure from 145/61 to 195/67 mmHg. There was minor downsloping ST segment depression at peak exercise and a few extrasystoles were observed. However, 75 seconds into the recovery phase he developed a bradycardia of 37 beats/min and shortly after that his heart stopped for a prolonged period (figure). He was placed in a supine position, and whilst preparations were being made to institute cardiac massage he developed a tonic-clonic fit. There seemed no point in attempting massage during the tonic phase and with the first clonic convulsion a sinus beat was noted which initiated a return of the sinus bradycardia and a rapid recovery. Shortly afterwards, however, cardiac asystole

Figure Three-lead ECG (II, V3, V5) recorded during the recovery phase of a standard exercise test, revealing a sinus bradycardia followed by prolonged (>20 s) episode of asystole (non-continuous recording)
was again observed and the sequence of events was repeated. Following the administration of 1 mg of atropine intravenously he made an uneventful recovery.

A dual-chamber pacemaker system was implanted and repeat exercise testing to similar workload was uneventful. During the recovery phase the pacemaker was seen to activate his atria. The patient experienced minor symptoms but there was no recordable drop in blood pressure.

Discussion

Syncope occurring after vigorous effort is rare but has been documented previously. The cause has been thought to be a variant of neurocardiogenic syncope with an afferent parasympathetic response to decreased venous return that is greater than normal, and reduced sympathetic tone following exercise. Patients in previous reports have been treated conservatively with either guidance on avoiding strenuous exercise, drug treatment such as beta blockers, or anticholinergics, including disopyramide. Such an approach in our patient was undesirable in view of his lifestyle.

The implantation of a pacemaker resulted in abolition of his symptoms, as might be expected, as this treatment is often effective in the cardio-inhibitory form of vasovagal syncope. A single chamber atrial system was considered, but in view of the severity of his symptoms and the small but definite risk of atrial lead displacement, a dual chamber system was felt to be most desirable.

The ability of a convulsion to restart the heart does not appear to have been documented previously. The relationship between a clonic convulsion and the onset of a sinus beat on two occasions was remarkable. In this way, the convulsion mimicked a praecordial thump and initiated the heart beat in exactly the same fashion as bystander resuscitation can do.

Summary points

- Syncope occurring during effort is well recognised, but syncope after effort may occur in the absence of identifiable cardiac or neurological disease.
- Exercise testing with prolonged observation in the recovery phase may be useful to establish the diagnosis.
- Convulsive movements during asystole may inaugurate a return to sinus rhythm.
- The definitive treatment in patients with syncope secondary to bradycardia/asystole is pacemaker implantation.