He continued to have dizzy spells and collapses, culminating in a severe episode at school, three months after his mother had Brugada syndrome diagnosed. The episode was witnessed by his teacher, who reported that his pulse rate was extremely rapid during the attack. Subsequent investigations confirmed that he had Brugada syndrome, and he has remained free of symptoms since the insertion of a cardioverter defibrillator.

Discussion

The family history of patients with syncope should be carefully evaluated for evidence of familial causes of sudden cardiac death. The commonest of these is hypertrophic cardiomyopathy, which has an estimated prevalence of 1:500 to 1:5000.[4] Other causes include hereditary arrhythmias (such as long QT syndromes and Brugada syndrome) and hereditary structural defects (such as arrhythmogenic right ventricular dysplasia). These conditions are rare compared with other causes of syncope. Careful history taking and simple investigations are therefore needed to identify patients who warrant referral for specialist assessment.

Diagnosing Brugada syndrome is not straightforward as it exists in many forms. The syndrome is inherited as an autosomal dominant trait, but expression varies—for example, the typical electrocardiographic pattern is often absent or transitory.[5] The syndrome is most common in men of South East Asian origin, but it has been described in many different age groups and ethnic origins.[5]

Our report shows the difficulty in diagnosing Brugada syndrome in atypical cases. Case 2 had symptoms but normal resting electrocardiographic appearances. Diagnosis in such patients depends on unmasking concealed conduction abnormalities by giving intravenous antiarrhythmic drugs such as ajmaline. This test is useful because symptomatic patients are at risk of sudden death if left untreated.[6]

Case 2 also illustrates the difficulty in distinguishing cardiac causes of loss of consciousness from neurological causes. Investigations in all cases of unexplained syncope should include resting and 24 hour electrocardiography. However, normal results do not exclude Brugada syndrome, and if the patient is at risk an ajmaline test should be considered.

Diagnosis is further complicated by unrelated conditions such as right ventricular dysplasia, in which the electrocardiogram may show the typical Brugada-like pattern and the patient is prone to sudden arrhythmic death.[7] Structural heart disease must therefore be excluded before diagnosing Brugada syndrome.

Identification of a genetic abnormality is helpful in distinguishing Brugada syndrome. Furthermore, identification of a recognised mutation will facilitate screening of relatives. The genetic defect is a mutated cardiac sodium channel gene (SCN5A) on chromosome 3.[8] The mutant sodium channel shortens the cardiac action potential, making parts of the cardiac tissue vulnerable to re-entry circuits. There are several recognised mutations causing Brugada syndrome, but in many cases a recognised mutation is not found. Such genetic heterogeneity may explain the heterogeneity of expression between individuals.

The prognosis for patients with Brugada syndrome is poor unless they are treated. Mortality is thought to be up to 10% a year.[5] Drug treatment is not effective, but an implantable cardioverter defibrillator has been shown to prevent sudden death.[3]

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Corrections and clarifications

Libraries face higher costs for academic journals
In this news article by Susan Mayor (19 April, p 840), we said that an article in the *Wall Street Journal* (2003 Feb 21) cited a 43% net profit by Reed Elsevier. This is wrong. The 43% was, in fact, the increase in Elsevier's profits from last year.

Minerva
At the end of her page in the issue of 5 April (p 772), Minerva reported a follow up study of men taking part in atmospheric tests of nuclear weapons in Australia and the Pacific in the 1950s and 1960s (*Occupational and Environmental Medicine* 2003;60:165-72). Unfortunately, she overstated the total number of UK test participants by a factor of 10: just over 21 000 men took part, not "some 200 000." The authors estimate that the cohort they studied represented about 85% of all UK test participants.

Afghanistan struggles to build post-conflict health care
In this news article by Tessa Richards we wrongly attributed the report "Collateral damage: the health and environmental costs of war on Iraq" to the World Health Organization (19 April, p 837). As we stated, Jane Salvage wrote the report, but it was in fact commissioned by Medact, a UK organisation of health professionals working for global health. The report can be viewed on Medact's website (www.medact.org) and is also available by email (info@medact.org).

Interactive case report

A 42 year old man with acute chest pain

This man's case was described on 26 April and 3 May (*BMJ* 2003; 326:920 and 974). Debate on his management continues on bmj.com (http://bmj.com/misc/interactive_case_report2.shtml). On 24 May we will publish the outcome of the case together with commentaries on the issues raised by the management and online discussion.

We welcome contributions of interactive case reports. Cases should raise interesting clinical, investigative, diagnostic, and management issues but not be so rare that they appeal to only a minority of readers. Full details of the criteria are available at: bmj.com/cgi/content/full/326/7389/564/DC1