development of CHD in postmenopausal women. Such studies are planned but until the results are known we can only remark that if a therapy that seemed to reduce CHD incidence by 50% were available to men they would be encouraged to take it.

Royal Alexandra Hospital,
Paisley
Royal Brompton and
National Heart Hospital, London
Western Infirmary, Glasgow

IAIN FINDLAY

DAVID CUNNINGHAM

HENRY J DARGIE

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COMMENTARY

Arrhythmogenic right ventricular dysplasia: the phenotypic range defined

In the fully expressed form seen by pathologists the diagnosis of arrhythmogenic right ventricular dysplasia is not difficult. The heart shown here (figure) is typical and it came from a 23 year old man who died suddenly while playing football. There was a history of previous palpitation. The pathology is unmistakable. The right ventricle is dilated

and there is transmural replacement of the myocardium with fat particularly in the anterior wall. It is a truism that the pathologist sees the worst of every disease but the disease is fully expressed and easily recognised. Studies of large families show a wide clinical expression of arrhythmogenic right ventricular dysplasia in living subjects, many of whom do not have symptoms.

A classic method for defining the gene responsible for any disease is to determine its chromosomal site by linkage with other known chromosomal markers through several generations. For this to be successful, however, it needs to be certain who has the disease and who does not. Laying down a set of criteria for the diagnosis of arrhythmogenic right ventricular dysplasia is a valuable advance. The criteria will also help to establish how common arrhythmogenic right ventricular disease is in any particular population. In the United Kingdom the impression among pathologists is that arrhythmogenic right ventricular dysplasia is much less common than hypertrophic cardiomyopathy as a cause of sudden natural death in young adults. The ratio is thought to be lower than 1:10 but may not reflect the true frequency of the disease in living subjects.

Sudden death on exercise in a man of 23. The right ventricle is dilated and in areas the wall is totally replaced by fat.

MJ DAVIES