It must be emphasized that there are great difficulties in the certain assignment of specific gene loci to particular chromosomes, and J. H. Renwick[10] has reviewed the pitfalls in using aberrations of chromosomes to place gene loci on these bodies. Some of the problems, such as the inheritance of silent alleles or doubtful paternity, are eliminated in the patient described by Dr. Callender and her colleagues, and this is an advantage of such prospective studies in single individuals.

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A Cause of Sudden Death

Obstructive cardiomyopathy has become well recognized in the last 12 years. There is marked hypertrophy of the left ventricle, especially of the interventricular septum and outflow tract with consequent narrowing in systole. It presents in two guises: as a cause of sudden death in the previously healthy and as a disease whose manifestations include dyspnoea, angina, dizziness and syncope, and congestive heart failure. The importance of obstructive cardiomyopathy as a cause of sudden death is underlined by a report by T. K. Marshall[1] of 16 examples occurring in Northern Ireland between 1959 and 1967. It accounted for one in every 200 sudden cardiac deaths, and though occurring mostly in the under 30 age group there were instances in every decade up to the eighth. The mechanism of death was thought to be a sudden arrhythmia, and two deaths were apparently precipitated by precordial trauma. With such a frequency as a cause of sudden death it is not surprising that the first comprehensive pathological description was made by a forensic pathologist.[2]

Since D. Teare's description the disease has gained many synonyms and initials (but no eponyms) which include asymmetrical hypertrophy, idiopathic hypertrophic subaortic stenosis (I.H.S.S.), hypertrophic obstructive cardiomyopathy (H.O.C.M.), and hereditary cardiac dysplasia. Two forms are recognized, the familial and the sporadic, and in a series of 126 patients at Bethesda[3, 4] 40 fell into the familial group and 86 into the sporadic. There was a male preponderance in both groups, greater in the sporadic. Sudden death was commoner in the familial, as was severe disablement in the non-fatal cases. Inheritance is apparently by a dominant gene with variable expressivity and incomplete penetrance. The condition has been described in the newborn and has been reported with concomitant congenital cardiac anomalies.[5] The exact pathogenesis of the hypertrophy and even the mechanism of its effect on cardiac function are in doubt. Electron microscopy of the abnormal region has shown mainly non-specific changes, although E. H. Sonnenblick[6] has demonstrated great variations in sarcomere length in operative biopsy specimens, and it seems possible that these abnormal fibres are unable to contract.

The suggestion that there is an increased amount of myocardial noradrenaline[7] has encouraged the study of the effect of β-adrenergic blocking agents in treatment, and some good results have been claimed. Surgical treatment has mostly consisted of removal or division of hypertrophied muscle, either via the left or the right ventricle. In a series of 42 patients treated at the Hammersmith Hospital[8] with oral propranolol for four years dyspnoea was relieved in no more than half, but angina was relieved in the majority. The main place of surgery appears to be in those patients with severe outflow tract obstruction, and in 22 individuals so treated 14 showed symptomatic improvement.

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Aortic Aneurysm and Peptic Ulcer

Aneurysms of the abdominal aorta are being detected more often than formerly, and the great majority are atheromatous in origin.[1] Atheromatous aneurysms generally affect the aorta below the origin of the renal arteries, and they may extend to its bifurcation. They enlarge progressively and usually show themselves as a pulsatile abdominal swelling. Sometimes they cause upper abdominal pain, which may simulate a peptic ulcer, though it is usually not related to food. Untreated aneurysms tend to rupture if the patient does not succumb beforehand to other cardiovascular diseases. About 10% rupture into some part of the gastrointestinal tract, the third part of the duodenum being the commonest site.[2] The resulting haematemesis may be difficult to distinguish from that due to a peptic ulcer.

A. W. Jones and his colleagues have recently investigated the incidence of peptic ulcer in patients with an abdominal aortic aneurysm.[3] They examined the necropsy records of a Manchester teaching hospital over a 13-year period and found 99 cases of aneurysm and 523 cases of peptic ulcer. The incidence of peptic ulcer in the general necropsy population was 7·2%, while in cases with aneurysm it was 22·6%. Of the 22 cases of aneurysm with peptic ulcer only two occurred in women; these had gastric ulcers and both died of ruptured aneurysm. Of the 20 men with the combined lesions, 14 had duodenal ulcers. There was a significant statistical association in males between duodenal ulceration and abdominal aortic aneurysm.

The explanation of this association is obscure. A. Elkeles[4] noted an association between radiological calcification of the aorta and its branches and gastric ulcer in people over the age of 50, and suggested that the ulcers were caused by ischaemia. Gastric ulceration of the elderly is generally held to differ in certain respects from the more common duodenal ulceration that occurs throughout adult life. It is seen predominantly in the labouring classes in Britain and particularly in poor, malnourished people.[5] Perhaps devitalization of the