instance, is nearly £15 a week and East Anglia's is £14 12s. 4d. Conversely, at £12 8s. 4d. the Liverpool region's is less than the national average of £13 13s. 4d.

But the cost per inpatient week is only one aspect of the financial picture. A better measurement of efficiency for acute hospitals—and all the figures given here have been for this type of hospital—is the cost per inpatient case, calculated by dividing inpatient expenditure by the total number of discharges and deaths. Hospitals that, by reducing the length of stay, can treat more patients show a lower cost per case. To some extent, therefore, hospital regions with high staff costs show a low cost per case because they are able to discharge patients more quickly. Oxford's cost per case, for instance, is the lowest, at just below £69 compared with a national average of £82 10s. 2d., and the length of stay in that region is only eight days compared with a national average of 10·4 days. Similarly, the region with the highest cost per case (nearly £93) is the South-west Metropolitan. It not only has a comparatively high weekly cost for maintaining inpatients, because its staff bill is higher than the average, but also keeps them in hospital for just over 11 days.

The average length of stay in hospital over the country as a whole was lower by a day and a half in 1969-70 than five years previously, a trend that means that the increase in the cost per case rose by just under 8% last year, compared with the rise of 10½% in weekly maintenance costs. Here is an aspect of financial management that directly concerns doctors. But it is not the only aspect. Both at the Cogwheel-Salmon conference held at Church House recently[2] and at a conference on management accounting sponsored by the King's Fund at the Hospital Centre last week hospital administrators and treasurers urged that doctors—not only senior doctors—should take a greater increase in management, for which costing is one tool. The new costing returns are for the year 1969-70, and are therefore published ten months after the event, after budgets have been fixed for the coming financial year, 1971-2, and after treasurers have begun to frame their estimates for 1972-3. Last week hospital treasurers, a few of whom at the suggestion of the King's Fund have been experimenting with departmental costing[3] throughout the year, strongly recommended that monthly costing returns should be adopted in all hospitals and that the monthly returns of expenditure under subjective headings (manpower, food, drugs, heating, lighting, etc.), unrelated to departments, should be dropped. The present financial returns, it was pointed out, merely show whether hospitals are keeping within their allocations, not whether the money is being spent on the right things; nor do they show changes in work load and the consequent need to transfer resources from one department to another.

The present form of costing is not, of course, the last word in management accounting. Some hospitals are going much farther and have adopted functional budgeting. They provide budgets for medical and surgical services, nursing services, laboratories, engineering, supplies, and catering for a whole group and give the heads of departments responsibility for spending them. The Department of Health is apparently sympathetic towards the new outlook on management, and, provided the Treasury agrees, the old system of subjective accounting may be dropped. In any case doctors, who after all initiate expenditure, cannot continue to remain aloof from management.

[1] *Hospital Costing Returns for the Year Ended 31 March 1970.* London, H.M.S.O., 1971 (30s. net).
[2] *British Medical Journal,* 1970, **4**, 635.
[3] Tagg, A. J., Baddeley, S., Hall, E. A., *The Hospital,* 1970, **66**, 341.

Chromosome Mapping

Human chromosomes can be observed directly in dividing cells, whereas genes can be observed only indirectly by studying known inherited characters. Genes, or more correctly gene loci, are said to be "linked" when the characters for which they are responsible have a strong tendency to be passed on together from parent to offspring. But, though linked gene loci are on the same chromosome, the particular one is not often identified. Thus the demonstration of linkage between two gene loci and the assignment of a gene locus to a particular chromosome are different though complementary methods of mapping the human chromosomes. Studies based on the segregation of two inherited characters within families have established with some certainty linkage between particular gene loci. Examples are the ABO blood group locus and the nail-patella syndrome,[1] and Rh and one form of elliptocytosis.[2 3] In addition certain stable gene complexes (closely linked genes) such as those controlling the Rhesus and HL-A tissue antigen systems are known.

The paucity of good evidence for assigning genes to particular chromosomes makes the case reported in this week's *B.M.J.* (p. 131) by Dr. Sheila Callender and her colleagues specially important. They describe a man with polycythaemia who initially had bone marrow cells with normal chromosomes and who was of Rh blood group CDe/cDE. Five years later he had a major abnormal clone of marrow cells with a missing chromosome from the C (6–12) group and also deletion in the long arm of a chromosome in the B (4–5) group. He also had a minor clone of cells in which the only abnormality was a missing C group chromosome. Only a few cells with normal chromosomes were seen. At this time he had two populations of red cells, one with Rh group CDe/cDE (as before) and the other with CDe/CDe. One interpretation of the findings is that the Rh locus is on the missing C group chromosome, and another that it is on the deleted portion of the B group chromosome. The authors consider the latter interpretation the more likely.

This location of a clinically important blood group locus is tentative, because our knowledge of the factors, genetic or otherwise, which influence the phenotypic expression of the blood group genes is still very limited. For example, perhaps the chromosomal deletion observed modifies the expression of the genes concerned rather than that the genes themselves are lost with the deletion. A modifying gene was invoked by W. L. Jenkins and colleagues[4] to explain their findings in a blood donor with two cell populations differing in both the Rh and Duffy blood groups.

The firmly assigned gene loci so far are those for Duffy,[5 6] the enzyme thymidine kinase,[7] and the serum protein alpha haptoglobin,[8] and the chromosomes concerned are numbers 1, 17 (?18), and 16 respectively. The gene locus for the Duffy blood group is linked to that for congenital zonular pulverulent cataract,[9] and this therefore is presumably also on chromosome 1.

The method of assigning control of the Duffy blood group to a gene locus was to establish the existence of a linkage between the blood group locus and a variant of chromosome 1 itself—an elongation of the long arm in one member of the pair. In the location of alpha haptoglobin a pedigree was examined in which there was a translocation between one arm of a No. 2 chromosome and one arm of a No. 16 chromosome. The thymidine kinase locus was established as a result of culturing hybrids between human and mouse somatic cells.

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