

specific receptors for low density lipoprotein. He might have added that there are at least three mutant alleles² giving rise to absence of receptors, to receptors that are present but unable to bind low density lipoprotein adequately, or to receptors which having bound low density lipoprotein fail to internalise the bound particles. The importance of these defects is not only in the very slow removal of low density lipoprotein and cholesterol from the circulation but also in the associated enhancement of endogenous cholesterol synthesis which impeded or absent entry of low density lipoprotein into the hepatic cells, in particular, encourages.³

Miller's persuasive paper on the rise in serum cholesterol concentration with age points firmly to a decline in the activity of these receptors with increasing age.⁴ The accumulating evidence, especially from the several research establishments studying Watanabe's heritable hyperlipidaemic rabbits, makes it clear that low density lipoprotein particles do not normally carry any cholesterol of exogenous or dietary origin and that a raised serum low density lipoprotein cholesterol occurs only in subjects with one or more of several genetic defects.⁵ Hypercholesterolaemia in man is never simply a question of dietary excess.

Perhaps we should number among the disservices done us by the dietary evangelists, who prefer the lay media to the scientific journals for their pulpit, the fact that so much research into the origins of atherosclerosis has centred on diet when it might more profitably have addressed the questions of receptor function and elimination pathways for cholesterol. With luck now as the pendulum begins to swing the other way we will hear a lot less about a diet of muesli and low fat yoghurt for the nation and more about screening a selected population for the genetic markers for a predisposition to premature coronary heart disease. The signs are encouraging that we will know what to do for them when we find them other than handing out the standard and ineffectual diet sheet.

ALEXANDER MACNAIR

London W1M 7AD

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- 2 Fredrickson DS, et al. The familial hyperlipoproteinaemias. In: Stanbury JB, et al (eds). *The Metabolic Basis of Inherited Disease*, 4th ed. New York: McGraw-Hill, 1978:30.
- 3 Brown MS, Kovanen PT, Goldstein JL. Regulation of plasma cholesterol by lipoprotein receptors. *Science* 1981;212:628-35.
- 4 Miller NE. Why does plasma low-density-lipoprotein concentration increase with age? *Lancet* 1984;i:263-7.
- 5 Goldstein JL, Kita T, Brown MS. Defective lipoprotein receptors and atherosclerosis. *N Engl J Med* 1983;309:288-97.

"First vacant, first cut"

SIR,—May I use your columns to correct some misconceptions about this matter? In their letter "First vacant, first cut" Professor C G Clark and Professor N L Browse (4 August, p 322) refer to closure of certain posts in general medicine and general surgery—the only specialties affected by the new procedures. The power to do this has been invested in the Central Manpower Committee by a joint working party set up between the Joint Consultants Committee and the Department of Health and Social Security. The Central Manpower Committee does not decide policy.

When posts fall vacant I, as chairman of the Central Manpower Committee, and the chairman of the senior registrars subcommittee of the Central Manpower Committee are told and receive submissions from the interested parties in the district and region. If it is thought that a post might be closed we confer with the chairman of the appropriate college manpower panel and usually with members of the departments likely to be affected. Following a decision of the last Joint Consultants Committee meeting we were asked to tell the college presidents as well. In the event of appeals against closure (the rule so far) we reconsider the matter. In one case permission has been granted to readvertise because incomplete information was given in the first place.

We are fully aware of the difficulties that arise when a senior registrar post is closed. What must be borne in mind, however, is the present regrettable surplus of senior registrars in certain specialties. Even allowing for a 2% to 3% annual expansion in consultant posts, we estimate that there are roughly 30 too many senior registrar posts in general medicine and 40 too many in general surgery. One indication of this excess is the increase by 10 of senior registrars in posts for more than four years between 1982 and 1983. Time is not the only thing that expires for senior registrars—hope does as well. Many dare not express their despair in case this militates against their chance of promotion to a consultant post. It is wrong to train people for posts that do not exist. A failure to agree on the methods by which career prospects could be improved must not be allowed any longer to defer approaching the objective. We agree that it is regrettable that there has been delay in convening the Joint Planning Advisory Committee which will, if it is created, recommend target quotas for each region. The DHSS has been made aware by the Central Manpower Committee of how much easier and fairer closure procedures would be using this approach. Pressures from all quarters would be welcome.

It has to be said that the prospects for consultant expansion are not good enough to absorb all senior registrars at present in training without an unacceptable increase in the years spent in the senior registrar grade. Opportunities for part time retirement at the age of 60 would greatly help to remedy the present position, and I hope that progress will be made by the joint negotiating committee in the next session. Every encouragement should be given to this initiative by all concerned with manpower problems. Experience over the past five years has led me to observe that while everybody agrees collectively that "something should be done," when it comes down to individual cases compelling reasons are always proposed why the cuts should not occur here. The strength of the Central Manpower Committee is that it has no vested interests to protect.

Professor M D Vickers (25 August, p 501) has already replied to the protest letter from the junior surgeons at St Thomas's Hospital. Steps are being taken by the Joint Consultants Committee to attempt to match the number of career registrars to senior registrar vacancies, which should improve prospects somewhat. Meanwhile, we must face the difficult and painful process of turning the career pyramid into a cube.

G H HALL
Chairman, Central
Manpower Committee

Exeter EX2 4NT

Family history of congenital hydrocephalus

SIR,—Professor John L Lorber (4 August, p 281) in his longitudinal study of uncomplicated congenital hydrocephalus tried to eliminate all cases which could have resulted from prenatal or perinatal haemorrhage. He comments: "Perinatal bleeding was unlikely to have caused the hydrocephalus in any of the present series because the proportion of babies weighing less than 2500 g was similar to that in the general population."

With the routine use of ultrasound scanning in the preterm baby intraventricular haemorrhage has been recognised as a common problem. It has been reported in mature babies in whom there have been no perinatal predisposing factors and in whom presentation has been after discharge from the neonatal nursery.¹

We have recently noted intraventricular haemorrhage in three babies, two presenting after discharge from the maternity unit. Ventricular dilatation was present at the time of diagnosis in all babies. Two required ventriculoperitoneal shunts in the neonatal period for control of the hydrocephalus; repeat scans showed resolution of the intraventricular haemorrhage.

With increased ultrasound scanning of mature newborn babies, reports of intraventricular haemorrhage in babies born at term have increased.² Congenital hydrocephalus has been recognised in relation to intraventricular haemorrhage in these babies.³ Intraventricular haemorrhage as an aetiological factor for congenital hydrocephalus cannot be excluded unless the infant has been screened by ultrasound soon after birth.

A E HILL
M E I MORGAN

Child Development Centre,
Alder Hey Children's Hospital,
Liverpool L12 2AP

- 1 Andreoli S, Josephson DA, Schreiner RL. Late presentation of intraventricular haemorrhage in term infants without predisposing factors. *J Indiana State Med Assoc* 1982;75:460-2.
- 2 Lacey DW, Terplan K. Intraventricular haemorrhage in full-term neonates. *Dev Med Child Neurol* 1982;24:332-7.
- 3 Scher MS, Wright FS, Lockman LA, Thompson TR. Intraventricular haemorrhage in the full-term neonate. *Arch Neurol* 1982;39:769-72.

"I have a bone stuck in my throat"

SIR,—Unfortunately, the management of patients with suspected upper digestive tract bony foreign bodies is not as straightforward as Dr N Kirkham and Dr Ruth English imply (18 August, p 424). Accurate clinical assessment of the patient is vital, but the authors failed to mention important signs such as the presence of localised tenderness in the neck, loss of laryngeal crepitus, and pooling of saliva in the hypopharynx on mirror examination. We would also like to point out that indirect laryngoscopy is unsatisfactory in many patients due to gagging; even if a good view is obtained the cervical oesophagus cannot be assessed. The statement that indirect laryngoscopy and radiology can conclusively exclude the presence of a foreign body is untrue. Definitive exclusion requires direct laryngoscopy and pharyngo-oesophagoscopy—a procedure fraught with difficulties, especially in the kyphotic patient, and one carrying a small but significant mortality.

We believe, therefore, that the management of this relatively common ear, nose, and throat