suggestion[16] that it resembles halothane hepatitis has been denied.[17] Liver problems have not been documented after isoflurane, but, none the less, four of 27 cases reported to a subcommittee of the Food and Drug Administration in the United States over four years could not be attributed to other factors.

The incidence of reports of hepatotoxicity related to enflurane or isoflurane has not increased in the United States despite these agents having almost supplanted halothane. If that experience is translated to Britain the incidence of liver failure due to enflurane is likely to be one case a year if all 3 million surgical patients given anaesthesia received enflurane. Early results from Oxford of comparative studies of enflurane or isoflurane in a population likely to develop hepatitis related to halothane show that neither agent results in any substantial changes in liver function values.[18]

What, then, should the anaesthetist do to protect his patient from the remote possibility of liver damage and himself from the threat of litigation? Are there now any absolute indications for the repeated use of halothane within six months? The only problem comes where inappropriate concern about the added cost of using enflurane or isoflurane (or failure to provide the means to deliver these agents) may force the anaesthetist to put the patient unnecessarily at risk. Surely it is reasonable for the added cost of more expensive agents to be borne by the health authorities rather than their having to join the medical insurers in paying potentially huge settlements for avoidable halothane hepatitis?

Halothane hepatitis is as rare as mortality related to anaesthesia itself, and it is avoidable given adequate equipment, records, and forethought. The revolution that resulted from the introduction of halothane is likely to be replaced by another—the use of the more expensive but less hazardous enflurane and isoflurane.

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# Obese deceivers?

Without doubt obese people owe their excess body weight to the consumption of food energy in excess of their expenditure,[1] yet many studies of food intake by the obese have shown—paradoxically—that their energy intakes are not excessive. In addition, a multitude of largely anecdotal, but occasionally well documented, examples have been reported of individuals who maintain excessive weight despite caloric restriction.[2] The paradox has led many workers to propose and seek for metabolic causes, either in lower basal metabolic rates or lower expenditure in activities. In recent years a biochemical basis has been postulated for this apparent metabolic efficiency,[3] leading to considerable interest in thermogenesis induced by diet and to elegant studies of brown adipose tissue.

The study of energy balance with the precision needed to test these various hypotheses has to overcome two main difficulties: firstly, the measurement of energy expenditure in people leading a normal life, and, secondly, the measurement of food intake and its translation into nutrient intake, the major challenge to the quantitative study of human nutrition.[4] The first of these problems seems to have been solved by using water double labelled with deuterium and oxygen-18 together with improved instruments for measuring isotope mass ratio. We now have a procedure for measuring energy expenditure in free living people that is both ethical and non-restrictive.[5] The method provides a measure of carbon dioxide production, and thus provides data equivalent to those generated by well established indirect calorimetric procedures.

A recent study by Prentice and colleagues (12 April, p 983) attempted to tackle various elements of the energy balance paradox in a group of women who became obese during or after pregnancy and compared them with a group of lean women. Their conclusion was that in this type of obesity (and possibly other types of maturity onset obesity) the cause of the paradox lies in the underestimation of food intake by the individuals being studied. They found that the basal metabolic rates of the obese women were higher than those of the lean ones—in proportion to their lean body mass. Their 24 hour energy expenditures, measured by classic indirect calorimetry, were also higher, as were their energy expenditures in their normal life, as measured by the use of double labelled water. In both cases total energy expenditures were related to their increased body weight.

The recorded food intake of the obese people was, however, lower than that of the lean—and some 800 kcal less than energy expenditure. The obese group had lost weight during the study but even when this was taken into account their intakes were less than their expenditures by at least 500 kcal a day. Since the authors could find no evidence of lower

metabolic needs or energy sparing mechanisms they were left with only one other possibility: the underestimation of food intake. They concluded that the obese people must have been consuming an average of 530 kcal a day more than the lean controls in order to sustain their obesity. The cause of the obesity must, therefore, be sought in the control of food intake and eating behaviour.

This raises several questions. Is the deception deliberate? In this study did the obese misread the scales when they weighed their food or did they "forget" to weigh and record some foods? The size of the postulated underestimation appears too great to countenance accidental omission—it is equivalent to nearly a third of all the food they did record. Apparently these obese women were unable to admit to the experimenters (and probably to themselves) their actual food intake. Furthermore, the data from the lean group also included one substantial underestimation of intake; it might be difficult to prove that the lean were more consistent than the obese.

The paper also adds to the growing evidence that energy expenditures in normal life are lower than the currently accepted estimates of energy requirements.[6] Nutritionists will need to bear this in mind if obesity is not to become more common than it is. The central finding, however, is that this type of obese patient should no longer be able to claim inherently lower metabolic requirements. We should treat with scepticism all claims from obese patients that their dietary intakes are modest. In such people, possibly even more than in the lean ones, habitual intake is impossible to measure accurately, at least with existing techniques—a message that needs to be appreciated much more widely. The measurement of food intake is so crucial that it might be argued that research on these techniques is more important than other more prestigious topics.

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# *Regular Review*

# Psoriasis

R H CHAMPION

Psoriasis was last reviewed in the *BMJ* five years ago,[1] and, though no major breakthroughs have been made in our understanding or treatment of the disease, many small but useful advances have been made and have influenced clinical practice. The ramifications of the disease make it of interest to both scientists and clinicians and some 1500 papers have been written about psoriasis in these five years, including some good reviews and an excellent 600 page monograph.[2-6] Psoriasis remains one of the most common skin complaints, affecting about 1·5% of the population—or even up to 5% at some stage of their lives.[7] Once patients have developed psoriasis only 40% have prolonged remissions on long follow-up.[8] The severity may range from a minimal occasional cosmetic problem to a life threatening emergency.

The aetiology of psoriasis is multifactorial. Most of the clinical and pathological changes may be explained on the basis of the rapid turnover of the epidermis, but this does not immediately explain the other main ingredient of psoriasis—inflammation. We have come a long way from the observations of Van Scott and Ekel in 1963 that there is 27 times the normal rate of mitosis in the epidermis.[9] Normal skin produces 1246 new cells cm$^2$ a day from a total 27 000 cells; but psoriatic skin produces 35 000 new cells cm$^2$ a day from 52 000.[10 11] To achieve this dramatic increase the cell cycle is reduced from 311 to 36 hours; the proliferative cell population is doubled; and the proportion of basal cells actually concerned in division is also roughly doubled.

What causes this overactivity of the epidermis is still a vexed question. A genetic factor is undisputed but variable, probably with polygenic inheritance. Our usual concept of a powerful genetic influence has to be stretched to accommodate a disease which may appear for the first time at the age of 108.[12] The genetic influence seems less important in patients whose psoriasis starts later in adult life. Other trigger factors include trauma (a useful model for studying the disease), infection, drugs (notably lithium, chloroquine, and rarely β blockers), and rather more nebulously climate and hormonal factors, and perhaps even smoking.[13] The contribution of psychological stress is disputed. Most clinicians would agree that stress may modify the course of the disease, though seldom to the extent that some patients are led to believe, and there is only meagre scientific evidence for this.[6 14 15]

The whole skin is potentially abnormal in patients with