numbered 30 916, of whom 5119 were first admissions.[21] Over the country as a whole only about 10% of first admissions remain in hospital continuously for two years or more. Whatever the reasons, this group make neither a full recovery nor a social recovery; pursue neither a remitting nor relapsing course[19]; and remain in hospital for years—but they are comparatively few. They now total 500 to 600 a year—only about three people in an average London borough; but they are certainly part of that shadowy group of uncertain definition, the new long-stay patients.

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Can cockroaches cause asthma?

Though allergy to insects has been recognised for many years,[1-3] only recently has Kang shown that cockroaches may act as a provoking allergen in asthmatics.[4] He tested 22 patients with severe asthma by bronchial provocation with an extract of cockroach; 14 gave immediate positive reactions, and 13 of these also showed late reactions. Sodium cromoglycate given by inhalation before the tests inhibited these positive reactions. Other reports[5 6] have described a high incidence of skin sensitivity to cockroaches in allergic persons, and specific skin-sensitising antibodies have been found in the plasma. A study in New York[6] showed that 44% of 589 patients with allergic disease had positive skin tests. The incidence was highest in Puerto Ricans—the lowest socioeconomic ethnic group, who also had the most domestic infestations with cockroaches. Bernton and Brown[7] showed that skin reactions may appear at an early age, with positive results in 40% of a group of allergic children. As yet, however, the clinical relevance of these provocation tests is uncertain, since many patients also have other known sensitivities.

A contact skin sensitivity may occur from exposure to cockroaches, but their role as an inhalant respiratory allergen is probably more important. Attacks of asthma have been described in laboratory workers[8] exposed to these insects. The exoskeleton, faeces, and secretions are all allergenic and may form an important component of house dust, especially in poorer homes. Cockroaches may also contaminate food with thermostable allergens,[8] which can resist denaturation by normal cooking, and their high prevalence in the kitchens of restaurants and institutions may prove to be an unrecognised source of ingested allergen. Hospitals also commonly attract these unwelcome guests. The level and distribution of domestic infestation in Britain are unknown, but outbreaks have occurred in blocks of flats in some London boroughs. Thus further studies may lead to the identification of allergens, currently unsuspected, that prove to be of clinical importance.

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Perinatal epidemiology

One of the most difficult exercises in medicine is evaluating changes in clinical practice, unless this is done as part of a planned prospective study. This principle is particularly true of the care of pregnant women, for obstetricians are individualists who cling tenaciously to their particular style of practice. In any one unit there may be three or four techniques in use for achieving the same objective.

A great recent change in obstetric practice throughout the Western world has been an increasing reliance on tests of fetal wellbeing, which are replacing "risk factors" as a guide to the obstetrician on management. A simple example is that nowadays a primigravida aged 38 may have a normal vaginal delivery provided the condition of the fetus is satisfactory, whereas ten years ago she would probably have had an elective caesarean section because of the "potential" risk of labour to the baby. Few would dispute that such a change is for the better, but we need objective evidence that the tests for fetal wellbeing are as reliable as we think they are.

Most obstetric units use serial measurements of urinary or plasma oestrogens and of fetal biparietal diameter by ultrasound as their main indices of fetal wellbeing. Any decline in oestrogen excretion or fall-off in the growth of the head is considered to be evidence of failing placental function, necessitating delivery. The extent to which these tests are used generally depends on the incidence of risk factors in the obstetric population and the assiduity of the obstetrician, but there is a universal tendency for them to be used increasingly as reliance on them grows. The cost in terms of manpower and equipment must be considerable and can be justified only by improved clinical results.

In 1965 one of the two obstetric teams in Cardiff Maternity Hospital introduced several changes in practice—the use of tests of fetoplacental function and a more active approach to management in labour—intended to improve the prognosis for the fetus. The other team continued to practise as it had done over preceding years. Detailed information was collected about the patients, their management, and the outcome of their pregnancies. What is so exceptional about the Cardiff

Births Survey is not so much the results but the fact that obstetricians with major differences in their management of patients should have agreed to co-operate in such a venture.[1 2]

The results of this natural experiment provide us with an important evaluation of the effectiveness of what (with the exception of intrapartum monitoring) may be termed modern obstetric practice. Any benefit to the fetus and newborn of antenatal monitoring should be reflected in some improvement in perinatal outcome—at the very least that seems a reasonable hypothesis. The Cardiff figures do not provide such evidence of the value of currently accepted methods of antenatal monitoring. There was no difference in perinatal mortality or morbidity between the two units, even though most high risk patients on the active unit had been screened by oestrogen and ultrasound measurements. These results suggest that the more general application of these tests is unlikely to result in any major improvement in outcome. On the other hand, it would be wrong to condemn these widely used tests out of hand simply on the basis of a lack of demonstrable improvement in perinatal mortality. The experience of obstetricians generally is that they are valuable in detecting fetal growth retardation, mistaken dates, and so on. What the Cardiff results do show is that in these times of financial stringency we cannot afford to allow an increasing load to be placed on our laboratories by introducing new tests unless we are certain of their efficacy. The same was true of intrapartum monitoring by continuous fetal heart rate recording and pH measurements, though it seems that the evidence of the value of these techniques is so strong that the opportunity to evaluate them prospectively has been lost.[3] The probable consequence is that the DHSS will have to equip all labour wards throughout the country with fetal heart monitors without unimpeachable evidence of their value.

How can expensive mistakes be avoided in the future? It seems certain that obstetrics will continue to develop as a more precise and scientific branch of medicine with a resulting increase in demand for expensive services. Until now the policy within the NHS has been to allow local demand from consultants to determine whether or not a particular service was made available in a particular area. The cost might not appear to be very much—but it must have been multiplied many times if, as seems possible, a change in practice similar to the one in Cardiff has taken place in most other British obstetric units. With a centrally directed Health Service we should be in an ideal position to carry out well-conceived prospective trials on sufficient patients to answer any questions. Two perinatal mortality surveys[4 5] have shown the valuable influence that studies of clinical outcome can have on clinical practice. Perhaps now is the time to consider establishing a permanent unit of perinatal epidemiology for England and Wales. Scotland has already accepted the importance of the subject by making it an integral part of the community health service. Such a unit would make several important contributions, quite apart from the organisation of trials. By collecting data on perinatal mortality, and eventually morbidity, it could provide information on geographical variations that could be used to improve clinical practice. Once this basis of national data collection had been established questions that have obstinately remained unanswered, such as the influence of birth asphyxia on development, would be amenable to investigation. Undoubtedly the relatively small annual investment in such a unit would bring valuable dividends by making the best use of limited resources, quite apart from improving clinical care. We cannot afford to overlook the lessons that Cardiff has taught us.

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Liver disease and vitamin C

In company with guinea pigs, the red-vented bulbul, the Indian fruit bat, and other primates man cannot synthesise ascorbic acid (vitamin C). We have to rely on an adequate dietary intake, and deficiency readily occurs in diseases such as cirrhosis. Vitamin C deficiency in cirrhotics must be seen, however, in the wider context of deficiencies of many of the water- and fat-soluble vitamins. One survey of 140 patients with alcoholic cirrhosis in the United States showed that they all had low blood concentrations of at least two of the 11 vitamins measured.[1] Vitamin deficiencies were found less frequently in a recent survey of patients with non-alcoholic liver disease in Leeds.[2] Vitamin requirements are increased in liver disease to allow tissue repair and to compensate for diminished storage capacity,[1] but the dietary intake is often reduced because of anorexia or vomiting, while malabsorption,[3] pancreatic disease, and abnormalities of vitamin metabolism and storage[1] also contribute to the overall deficiency.

A recent survey has focused on the vitamin C status of 138 patients with liver disease.[4] Low concentrations of leucocyte ascorbic acid (a measure of tissue stores) were found only in the patients with alcoholic cirrhosis or primary biliary cirrhosis. Deficiency of vitamin C occurs in alcoholics with or without liver disease, and it can be correlated with dietary vitamin intake[5]; but the findings in primary biliary cirrhosis were unexpected, and various factors which might be responsible were considered. There was no correlation with dietary intake nor with the raised serum concentrations of the copper-binding protein caeruloplasmin, which has ascorbate reductase activity. Patients taking cholestyramine to control their itching had low vitamin C concentrations, and this may be a causal relationship—though binding to cholestyramine could not be detected in laboratory experiments. Nevertheless, many other factors influence vitamin C concentrations, including age, sex, and smoking.[6]

Vitamin C is concerned in many metabolic processes, such as collagen biosynthesis, corticosteroid and cholesterol metabolism, and electron transport processes. The conversion of folic acid to folinic acid is facilitated by vitamin C, so that a macrocytic anaemia may occur in deficiency states,[7] and there are theoretical reasons why gallstone formation might be enhanced.[8] Delayed drug metabolism is perhaps more immediately practically important. In guinea-pigs vitamin C deficiency results in decreased metabolism of various pharmacological agents, as shown by prolonged sleeping time after pentobarbitone and a prolonged half life for antipyrine. Individual liver microsomal electron transport components such as cytochrome P-450 are decreased.[9] All these abnormalities are reversed by the administration of ascorbic acid. In man there is little direct evidence that ascorbic acid deficiency interferes with drug metabolism, but one recent study showed that antipyrine half lives were significantly longer in those patients with liver disease who had the lowest