reduction in blood pressure in patients in the treatment group, the systolic pressure falling by 25 mm Hg and the diastolic by 10 mm Hg. This fall has been maintained during the period of follow-up, which in some cases has been for as long as five years. The trial protocol requires that data on morbidity and mortality should not be released during the study but that if treatment were shown to confer significant benefit or disadvantage the trial would be terminated. We can take it, therefore, that the substantial fall in blood pressure has not as yet been accompanied by a statistically significant reduction in stroke, myocardial infarction, and death.

But what of the adverse effects of treatment? No major disturbances have been reported in the concentrations of serum electrolytes, but there have been significant changes in the serum concentrations of creatinine and urate and in the fasting blood glucose. A rise in the creatinine concentration occurred in both the placebo and the treatment groups, but the initial rise was greater in those having treatment. The increase in urate also correlated with the rise in creatinine concentration. At the end of one year's medication the concentration of urate remained on average 0·06 mmol/l (1 mg/100 ml) higher than in the placebo group. Perhaps the most disturbing biochemical abnormality has been a rise in fasting blood glucose concentration in patients having treatment. Diabetes is a major risk factor for cardiovascular disease, and any benefits from reduction in blood pressure may turn out to be counterbalanced by thiazide-induced glucose intolerance.

Two points are worth making on the basis of these interim results. As yet no evidence has emerged to show that reduction in blood pressure by antihypertensive drugs alters the prognosis of hypertension in the elderly; and though the European trial may in time give valuable information on the benefit or harm of treatment, its conclusions will apply only to the particular treatments chosen.

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Alcoholic heart muscle disease

The damaging effect of alcohol on the heart muscle has been shown very clearly in studies both on animals and in man.[1-3] More recently, it has been found to have an adverse effect on skeletal muscle too.[4] Biopsy specimens were taken from non-alcoholic volunteers maintained on a high-protein diet enriched with vitamins and repeated after one month of alcohol intake.[4] No changes were found in the microscopic appearances of the muscle before and after alcohol consumption—but ultrastructural examination showed intracellular oedema, lipid droplets, excessive glycogen, deranged elements of sarcoplasmic reticulum, and abnormal mitochondria in the specimens after alcohol consumption. These are similar to those found in patients with heart disease due to alcoholism.[4 5] (Since the term cardiomyopathy is now reserved for diseases of heart muscle in which no cause can be established,[6] cardiac damage from alcohol is best termed[7] "alcoholic heart muscle disease" rather than "alcoholic cardiomyopathy.")

The results of attempts to detect early myocardial damage from alcohol by biochemical tests have so far been disappointing.[8] Estimations of the serum concentrations of creatine kinase and lactic dehydrogenase isoenzymes in 73 patients known to have alcoholism showed no examples of raised concentrations of the cardiac-specific isoenzymes.

Once the asymptomatic stage has passed, both acute and chronic forms of alcoholic heart disease may be recognised on clinical grounds and if alcoholic abuse is continued these may culminate in recurrent acute episodes of myoglobinuria and irreversible congestive heart failure.[4] Even at the early stage the condition may be diagnosed with reasonable certainty if the patient admits to regular, heavy consumption of alcohol. In practice, however, alcoholism is seldom admitted, and the clinician is faced with the difficulty of distinguishing heart failure due to alcohol from that due to congestive cardiomyopathy. No specific morphological criteria distinguishing the two conditions have been defined in the heart,[2 9 10] but advances have been made since the introduction of the bioptome,[11 12] which has permitted small samples of endomyocardial tissue to be taken for analysis.

Patients with heart failure associated with alcoholism have been found to have negative indirect fluorescence and raised serum concentrations of IgA when compared with those whose heart failure was due to congestive cardiomyopathy. Biopsy specimens from patients with congestive cardiomyopathy showed preferential binding of IgG and IgA.[13] In another study[14] the content of lactate dehydrogenase isoenzymes (LDH) was analysed in biopsy specimens from patients with congestive cardiomyopathy and alcoholic heart disease. The patients were then grouped on haemodynamic criteria, when a higher proportion of LDH_1 (H subunits) was found in those with alcoholic heart disease. This may prove a useful test for distinguishing the two clinical groups. Analysis of endomyocardial tissue samples has shown raised activity of enzymes such as creatinine phosphokinase, lactic dehydrogenase, and α-hydroxybutyric dehydrogenase in patients with alcoholic heart disease when compared with those with established congestive cardiomyopathy.[15]

These investigations are important, since accurate diagnosis has great relevance to prognosis. In alcoholic patients the outcome depends on the degree and stage of the damage to the heart (or skeletal muscle). Both preclinical and acute varieties of muscle damage are usually reversible if the patient stops drinking alcohol. Death may, however, occur in cases of permanent congestive failure or acute rhabdomyolysis and fatal myoglobinuria.[4]

Probably between 1% and 2% of chronic alcoholics reach the phase of heart failure.[8] If they can be persuaded to abstain from further drinking the progression of muscle damage can be arrested or improved. Continuing the high alcoholic intake, however, will eventually lead to irreversible myocardial damage, when the prognosis may then become poor. In contrast, patients with congestive cardiomyopathy almost invariably have a poor prognosis, with a 10-year mortality of 70%,[16] though a few patients may recover normal or near-normal cardiac function.[17]

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Towards fewer handicapped children

Ensuring that the whole population of Britain can enjoy the benefits of present medical knowledge is a political, administrative, and educational problem, and as such was really beyond the remit of the conference "Medical research into the prevention of crippling in children" sponsored by Action Research and Birthright and held last week at the Royal College of Obstetricians and Gynaecologists. Nevertheless, many of the speakers expressed their frustration that the results of their research are being made available so slowly throughout the country. Several thought that the incidence of severe handicap could be halved by applying what is already known. The conference was convinced that at very least facilities for resuscitation should be available in every hospital where babies are born.

The conference, which concerned itself mostly with severe mental retardation, cerebral palsy, and congenital anomalies, was attended by geneticists, obstetricians, paediatricians, epidemiologists, and basic research workers, including molecular biologists, and many speakers thought that this interdisciplinary discussion in itself was an important advance. One of the first problems identified was that no reliable figures are available of the incidence of severe handicap in Britain. The conference suggested that a register of birth defects should be started as soon as possible in all regions—computers could make this easy. The Scottish Home and Health Department plans to start such a register next year, but the DHSS is lagging behind. Birth defect registers already exist in Sweden and New South Wales and allow evaluation of new treatments as well as giving an idea of service needs. Registers would encourage the careful epidemiological research needed to identify teratogens and investigate inter-regional variations.

Geneticists are working hard to develop non-invasive techniques of diagnosis—particularly for Down's syndrome. They also want to be able to diagnose earlier—at present when the diagnosis of Down's syndrome is made half the mothers are more than 20 weeks' pregnant. Non-invasive techniques and earlier diagnosis should make possible the prenatal diagnosis of a higher proportion of cases. The technique of recombinant DNA mapping may allow prenatal diagnosis of many more conditions.

The obstetricians reported on the possibilities of prevention based on ultrasonography and fetoscopy. Many more conditions, including some forms of congenital heart disease, can now be diagnosed prenatally, when the right kind of resuscitation measures can be provided for delivery. Even so, the necessary skills are available in only a few centres. Fetoscopy, which allows fetal blood and skin to be sampled, opens up many possibilities, but the 7% fetal loss and the 8% incidence of preterm labour reported with present techniques have discouraged their use except for the diagnosis of the most serious disorders.

Epidemiologists suggest that paediatricians have a lesser part to play in reducing severe handicap, which mostly results from prenatal problems, but a considerable role in reducing milder handicap. Most of the principles of modern intensive neonatal care have been developed from work in animals and more of this is needed—obstetricians and geneticists were agreed on that. Particular problems are those of managing recurrent apnoea in the severely preterm infant; and finding reliable ways of measuring blood pressure, intracerebral blood flow, and intracranial pressure in preterm infants. These technical problems are being solved, but the conference heard less about the relation between severe handicap and social class. Severe handicap, particularly spastic diplegia, is known to be more common in lower socioeconomic groups, but we need more detailed information. Speakers believed that in the absence of large social change researchers might best employ themselves identifying which particular components of socioeconomic deprivation result in an increased incidence of handicap. A current Office of Health Economics briefing[1] shows that the British perinatal mortality is higher than the Swedish rate partly because twice as many babies (7%) born in Britain are below 2500 g, and this is mostly a function of class. A low-birth-weight baby has just as good a chance of survival in Britain as in Sweden. This perhaps confirms that the main ways forward in reducing the incidence of handicap are preventive, epidemiological, and prenatal measures.

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Solanine poisoning

Potatoes are such a common feature of the Western diet that most people are surprised to learn that they are the produce of a poisonous plant. In fact potato stems and leaves contain a series of alkaloidal glycosides, termed solanines, which are highly toxic. The normal tuber contains only small amounts of solanines in the peel and none in the flesh. Poisoning due to feeding the leaves and stems to domestic animals is well recognised, and one instance of poisoning in man was traced to the use of leaves and young shoots as a boiled green vegetable.[1] The main hazard, however, comes from eating "greened" potatoes.

Greening and sprouting occur when potato tubers are exposed to light or are stored in adverse conditions, and these processes are associated with the production of the alkaloids. Initially this occurs at the sites of increased metabolic activity, such as the "eyes"; but eventually solanines can be detected in the flesh of the tuber, and the normal, high concentration-gradient between the peel and the flesh is lost. Fortunately, few people cook greened or sprouted potatoes because of their appearance and their bitter, unpleasant taste;