other controls (the unrelated comparison group) in most tests, though they fell somewhat short of them in performance tests of I.Q. But the previously malnourished children did substantially worse than either comparison group on all counts.

No previous studies have used more appropriate children for comparison, though Hertzig and colleagues are careful to point out most of the deficiencies of their own investigation as well as those of others in this regard. Perhaps they underemphasize the fact that of their three groups only their malnourished group had been treated in hospital. However, it would be virtually impossible to control for this important factor, and it seems unlikely that the stay in hospital would account for the substantial deficits found in the later performance of the previously malnourished children.

An important question, less clearly discussed in this paper, is whether the developing human brain has a period of vulnerability comparable, for example, with that which has been shown in experimental animals to be related to the brain's "growth spurt." In this transient period of rapid brain growth even moderate growth retardation can probably permanently disturb the physical development of the brain. To interpret this finding for man is impossible without at least knowing accurately the timing and duration of the growth spurt of the human brain. An erroneous impression is widespread that this period comes to an end in the fifth month of life or slightly later, in the sixth to ninth months, as assumed by Hertzig and colleagues. The Jamaican children were admitted to hospital at different times throughout the whole of their first two postnatal years. Thus, if the supposed early end to the brain's growth spurt were true it should have been possible to demonstrate a distinction between those malnourished before about 9 months old and those aged between 9 months and 2 years of age on admission.

J. Dobbing, however, has recently shown in a quantitative survey of a large number of developing human brains that the growth spurt in not over by 9 months of age. In fact, it extends, in terms of cell-division alone, from the second trimester of gestation until at least 18 months postnatally. And the easily measured later components of the growth spurt, including accumulation of myelin lipid, continue well into the third and fourth year. Synaptic connexions almost certainly develop throughout the same period of life. A fair comparison between the findings in experimental animals and those in the Jamaican children would therefore lead to the prediction of very little difference in relation to the timing of the malnutrition within the first two years of life (always provided the physical development of the brain has anything to do with the mental performance measured by Hertzig and colleagues?), and this is what was actually found.

Clearly it would be a poor reward for all the expensive research that has been carried out if the only conclusion is that we should feed our children. However, if it can be shown that there are specific ages when the brain has a once-for-all opportunity to grow properly—and this does seem likely at least for its physical growth—then the social conclusions could be much more important. The search for periods of enhanced vulnerability is therefore important. So is the investigation of whether lack of any particular nutrient is particularly hazardous to brain growth. A further question is whether mere retardation of somatic growth while the brain is specially vulnerable can cause permanent disturbances in its structure and function. This is probably one research area in which the factors limiting progress are money and men rather than a shortage of ideas.

Subtypes of Australia Antigen

In 1969 C. Levene and B. S. Blumberg reported that different samples of Australia (or hepatitis-associated) antigen were not serologically identical. This has since been confirmed, and a picture is now emerging of the antigenic properties of Australia antigen and of the distribution of the subtypes in the community.

G. L. Le Bouvier has shown that all samples of Australia antigen share a common antigen a but differ in the possession of a second antigen, which may be either d or y, the two second antigens never being found together. Most samples can be classified as ad or ay, but a small proportion react only as a. Le Bouvier has detected an additional antigen x in most samples, and other antigens have also been described.

In spite of the evident complexities of its antigenic structure, most preparations of Australia antigen are either ad or ay, and some interesting differences in the distribution of these subtypes are already apparent. Subtype ay, for example, is common in drug addicts, whereas ad tends to be the predominant subtype among asymptomatic carriers. Most, but not all, cases of acute hepatitis are due to ay, which is the subtype of the Willowbrook MS-2 serum pool with which many of the classic studies on serum hepatitis have been made. The ay subtype was responsible for the exceptionally serious outbreak of hepatitis in the Edinburgh renal unit and has been the predominant subtype in other renal units also. However, in some renal units ad has been more common than ay. Ad is the predominant subtype among blood donors. As expected, the subtypes breed true in the sense that all cases in a single outbreak have been due to the same subtype. Serial samples from patients have also shown that subtypes do not change and that the same one is carried by patients over long periods of time.

Recently S. Iwarson, L. Magnus, and A. Lindholm reported on the results of serotyping samples of Australia antigen found among the donors and recipients of blood transfusions at the Sahlgren Hospital in Göteborg, Sweden. They found that 56% of the donors were ad whereas 75% of patients with hepatitis following transfusion were ay. Like other workers they noted a strong association between subtype ay and drug addiction, and in fact nearly half of the ay-positive donors were addicts. There was a high incidence of liver disease among 13 donors with ay in their blood, of whom six had acute hepatitis, three chronic persistent hepatitis, and one non-specific reactive hepatitis. In contrast, 16 of 18 ad-positive donors had normal liver function tests. There was a surprisingly low incidence of clinical

In July 1972 as an impasse seemed to have been reached the Hospital Junior Staffs Group Council asked the profession's representatives to approach the Department of Employment to arrange for arbitration under the Industrial Courts Act 1919. By Christmas despite prodding from the profession the Department of Health had not replied to this request. Thus because the consent of both parties to a dispute is required before arbitration can start, nothing has happened so far, though Sir Keith Joseph has twice discussed the problem with the B.M.A. and promised a reply in February.

Dr. C. E. Astley, Chairman of the Central Committee for Hospital Medical Services, has now drawn attention to the dispute in the lay press, mentioning particularly the risks facing doctors taking part in flying squad duties or working in dialysis units. Doctors will be the first to acknowledge that other workers in the health services such as nurses, ambulance men, and laboratory technicians face similar hazards, and the N.H.S. superannuation scheme—at present under review—makes some provisions for staff accidents in the course of duty. Accidents to N.H.S. staff on flying squad duty are not frequent and the screening of patients and staff has reduced the risk of working in dialysis units. Nevertheless, the death or serious injury of the breadwinner is a shattering financial as well as emotional blow to a family. Furthermore, the junior doctors see themselves in a unique position because the long-term financial loss to their families in the event of a tragedy is potentially greater than that for other N.H.S. staff. They therefore argue that existing N.H.S. provisions are inadequate, with the Secretary of State's discretionary powers playing a key part in any decision on benefit. According to a recent letter in the B.M.J. this lack of adequate "insurance cover" is already affecting the planning of "exercises for major disasters," and the situation has come to a head in two hospital regions where staff representations have prompted the boards to consider starting their own schemes for compensation. If regional boards acknowledge that there is a case to answer then the Department of Health should at least accept the call for arbitration.

The juniors' representatives are doubly irritated by the protracted talks about special risks, because apart from their conviction of the justice of the case they view the delay as symptomatic of the Government's seemingly lackadaisical approach to all their problems. This attitude was apparent well before the present general incomes freeze seized up any negotiations with financial implications, for apart from the discussions on special risks negotiations to revise the extra-duty payments scheme—started at the end of 1971—have made little progress. It was cumulative frustration over their relations with the Government that finally prompted the juniors' representatives to withdraw in the middle of the last negotiating session with the Department of Health. Such a decision cannot have been lightly taken and the H. J. S. Group Council is to review the whole position when it meets next week.

Though the senior negotiators did not withdraw, discontent is not confined to the juniors. Mr. A. H. Grabham, Chairman of the C.C.H.M.S. Negotiating Committee, has condemned the Government's attitude, and comments at the January meeting of the main committee demonstrate the unease that exists among hospital doctors. The Government should respond to these symptoms of discontent and show its willingness to talk seriously. Otherwise the unease may grow into something worse. A year in which the N.H.S. is being