Acute Hemiplegia in Childhood

SIR,—In the past the aetiology of cerebral catastrophes in childhood has remained unsolved from the developmental point of view. This is epitomized in the facts that (1) neonates dying of convulsions have never demonstrated a lesion of the forebrain, such as cysts, though cysts are frequently found in infants dying of status epilepticus after six months; (2) acute hemiplegia occurs in a child apparently neurologically normal after birth; (3) cerebral palsy does not reveal itself till approximately the sixth post-natal month.

This time factor is the main stumbling-block to the acceptance of developmental theories in relation to epilepsy, acute hemiplegia (*sic*) in childhood, and cerebral palsy. It has led to the conclusion that cysts of the brain and other abnormalities cannot be developmental in origin but are due to degenerative processes precipitated by environmental factors operating after birth. Experimental evidence seems to support such a hypothesis, and all efforts are directed to the investigation of environmental causes; however, not all cases can be explained in this way.

One of the basic conclusions reached by me in 1961 was that the human brain develops functionally by definite stages and that the forebrain is inactive during the first six months of post-natal life. Under such circumstances maldevelopment of the forebrain would be masked until about the sixth month of post-natal life. Hence convulsions occurring before the sixth month would be related not to the forebrain but directly to the midbrain—that is, all fits or convulsions leading to unconsciousness (though often precipitated by conditions in the forebrain after the sixth month) are midbrain finally. True

unconsciousness would only occur with spread to the midbrain. This helps in understanding the different forms of epilepsy and allows a clear-cut delineation between epilepsy and hysteria.

If we apply the same basic reasoning to the aetiology of acute hemiplegia, the likely causative lesion is a developmental abnormality of the cerebral vessels which does not reveal itself until approximately the sixth month, when stress is put on the vascular supply. The lesion of the forebrain might be an established one such as cysts or atrophy, but the initial crisis might well be delayed until stress is put on the vascular supply. The condition would affect areas of the brain rather than specific centres and mental subnormality would therefore be a common finding.

Gross abnormality of the vascular supply would lead to intrauterine death or obvious abnormality at birth. Angiography would be unlikely to reveal minor aberrations, and in the presence of the established syndrome the calibre of the vessel would not necessarily reflect the true position. Late atrophy of the hemisphere indicates increasing vascular inefficiency, and mental subnormality would be inevitable.

There are, of course, other causes, but in these the aetiology would be obvious. Nevertheless, continued study and the accumulation of evidence over the years indicates that my explanation is undoubtedly correct. Much work still requires to be done, and we should be doing it.—I am, etc.,

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Diabetes Mellitus and Insipidus

SIR,—I was very interested to read the article by Dr. S. Raiti and his colleagues on the independent hereditary patterns of diabetes mellitus and insipidus (28 December, p. 1625). Though Dr. G. R. Frazer (15 February, p. 433) produced evidence against Raiti and his colleagues' conclusions the following case history seems to support the original thesis.

Case History.—The family tree of the two families (L and F) is illustrated in the Figure, and it will be seen that one of the more remarkable things is the longevity of several of the

members of family L, in spite of their disability. Seven members of this family underwent tests of posterior pituitary function (water-load test, with and without injections of nicotine and pitressin), and in all of them the results were consistent with diabetes insipidus. Another feature is that as these patients have grown older their daily intake of fluid has tended to get less—for instance, a decline from 12–15 litres to 3–4 litres of water daily. Further, during a period of stress, such as fever or fatigue, the urinary output is also diminished; we have also seen this in another patient with proved posterior pituitary diabetes insipidus,¹ who excreted less urine during the acute and convalescent stages of

typhoid fever, and similarly at the onset of the menopause. A possible explanation of this feature is a transient decrease in adrenocortical function,² while in the case of the menopause the increased gonadotrophins produced may compete with corticotrophin.

The mode of inheritance in diabetes insipidus has recently been dealt with by Jaross and Henssge,³ who analysed about 50 family trees from the cases so far reported. Their conclusion does not support Forsmann's⁴ contention that the trait is a sex-linked recessive character. In our family L the heredity seems to have a non-sex-linked dominant pattern with rather weak penetrance, as is seen from the fact that only one of the non-identical twins in the fifth generation was affected.

The inheritance of diabetes mellitus in family F follows the former pattern and is not influenced by the presence of the diabetes insipidus trait, thus confirming the work of Raiti and his colleagues.—I am, etc.,

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Subcutaneous Phycomycosis

SIR,—Drs. D. P. Burkitt, A. M. M. Wilson, and D. B. Jelliffe reported 31 cases of subcutaneous phycomycosis seen in Uganda (27 June, p. 1669).

In India myself and colleagues¹ have reported a case of subcutaneous phycomycosis affecting a boy aged about 5 years.

This boy at first had a boil on the lower part of his left arm. This healed after two to three hot applications of a paste of wheat flour, but the healed site used to itch occasionally. A swelling started growing from the site; it gradually spread up and down the left limb, till in six months it occupied an area extending from the middle of the arm to the upper one-third of the forearm. On examination of the swelling about six or seven scattered nodules varying in size from 2 to 3 cm. were felt beneath the skin. The nodules were free from the skin except near the site of the boil. Here the skin was found to be thickened and excoriated, and a few pin-point red spots were seen near by.

Biopsy of the nodules revealed a few spherules in the subcutaneous fatty tissue. The spherules contained material that was periodic-acid-Schiff-positive, surrounded by eosinophilic material. Culture of the biopsy tissue yielded growth of *Basidiobolus ranarum* with its diagnostic zygospores. In studies of the culture we² have reported that the fungus secreted a periodic-acid-Schiff-positive material which on a moist medium diffused out like a membrane, while on ageing medium it became adherent to the cell-walls of the zygospores and chlamydospores. The latter structures on intraperitoneal injections in mice³ produced a few fatty nodules on the surface of the peritoneum. In one mouse one nodule was seen on the surface of the liver. Histology of the liver nodule showed that the material which was present around the chlamydospores or zygospores had crumbled down to a granular mass. This mass seemed to have produced tissue necrosis and neutrophil-cell response. When the same fungus was kept growing at room temperature it lost its periodic-acid-Schiff-positive coat in one year. Intraperitoneal injections at this time failed to produce any peritoneal nodules in the mice.