CHILDHOOD AUTISM: AN INVESTIGATION OF AETIOLOGICAL FACTORS IN TWENTY-FIVE CASES

DEAR SIR,

The paper by Lobascher *et al.*, in the November, 1970, issue (pp. 525–9) makes two suggestions about the causes of childhood autism which seem at first sight to be incompatible, namely that there is a strong (40 per cent) inherited element, more particularly from the father's side in this illness, and secondly that it is associated with overdue birth dates and prolonged parturition. These two statements could, however, be reconciled if the foetus triggered its own birth.

It is known (Comline, Silver and Silver, 1970) that in animals pregnancy can be indefinitely prolonged if either the pituitary or the adrenals of the foetus are removed, whence it is clear that it is the foetal adrenals which trigger the birth. That this is correct has been shown by injecting ACTH or cortisol into the foetal (as distinct from the maternal) circulation of a hypophysectomized foetus. This immediately precipitates labour. Is it not possible that the fault in autistic children resides in the adrenals (or alternatively in the pituitary)?

Incidentally it would not follow from this that the prolonged pre-natal period, with its risk of anoxia etc., was the *cause* of autism. The delayed birth might simply be one of the symptoms of a disorder of the adrenals which makes itself felt in other ways at a later date. The fact that autism tends not to develop till the age of two or three does suggest that it is not due to birth injury but rather to the fact that some system which ought to come into operation at this time fails to do so, perhaps because of the same deficiency which led to the prolonged pre-natal life.

It is claimed (Turnbull and Anderson, 1970) that the date of birth in man can be predicted from the levels of oestriol in the maternal urine at 34 weeks and that the higher the levels the earlier the birth. Also oestriol synthesis in pregancy is known to be due to the foetal-placental unit (Driscoll, 1969). Since the foetus can inherit autism from its father the fault cannot be in the placenta which performs the final stage of synthesis, the conversion of dehydroepiandrosterone sulphate to oestriol. The error must, therefore, lie in the route by which the foetal adrenals convert pregnenolone via 17OH pregnenolone and dehydroepiandrosterone to dehydroepiandrosterone sulphate. (It cannot be due to a fault in pregnenolone metabolism because this is the substrate for synthesis of all the adrenal cortical hormones and a failure here would lead to widespread and easily recognizable disorders).

If the condition persisted after birth one would expect oestriol levels to be abnormally low but not those of the other adrenal cortical hormones which are synthesized by a different route. Hence autism could be due to low levels of oestriol and of one, or more, of its precursors. It could not be due to abnormally high levels of one of the latter substances due to blockade at some point in the route because persons with adrenal carcinoma excrete prodigious quantities of these intermediates (Wilson, 1960) but they have no autistic symptoms.

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A LOCAL MENTAL HEALTH LIAISON COMMITTEE

DEAR SIR,

The Memorandum on 'Future Patterns of Care for the Mentally Subnormal' (*British Journal of Psychiatry*, November, 1970), emphasizes the need for co-ordination of the various services for the mentally subnormal. The Circular H.M. (65) 104 on 'Improving the Effectiveness of the Hospital Service for the Mentally Subnormal' mentions in paragraph 9 the need to strengthen links between hospitals for the mentally handicapped and the community, and suggests that one way of bringing this about is the local mental health liaison committee.

Such a local liaison committee has been established in an area of the West Riding of Yorkshire and is meeting at two- to three-monthly intervals at a hospital for the mentally handicapped. The members of the committee are drawn from the local health authority and the hospital service. They include the Principal County Medical Officer for Mental