phylactic penicillin and those not so treated. Since other workers have demonstrated the harmful effects of antibiotic prophylaxis, we consider that the use of penicillin prophylactically should be abandoned in the " clean " unconscious poisoned patient.

SUMMARY

In order to determine the incidence of respiratory infection in unconscious poisoned patients, and the value of prophylactic penicillin in the prevention of these infections, 177 consecutive cases were considered.

144 patients were admitted to the trial. Of 71 not given penicillin, 5 became infected; and, of 73 receiving prophylactic penicillin, 3 became infected. In most instances the infection was not severe.

33 patients were excluded from the trial. 18 developed infection, which was often serious.

Unconscious poisoned patients, on admission, can readily be designated " clean " or " unclean "—i.e., already infected or, because of aspiration, likely to be so. Prophylactic penicillin should not be given to the former, but most " unclean " patients develop serious respiratory infections which require energetic treatment.

Preliminary Communications

FOLIC ACID METABOLISM AND HUMAN EMBRYOPATHY

THE effect of folate deficiency on the development of rat embryos has been shown by Nelson.[1] Those embryos which were not resorbed were almost all malformed. Fœtal malformations have also been reported after giving a folic acid antagonist, aminopterin, to pregnant women.[2 3] The malformations were varied, but were all severe.

A study in Liverpool suggested a possible relationship between fœtal malformation and defective folate metabolism in the mother,[4] and we have investigated this possibility in greater detail.

MATERIAL AND METHODS

In 1964, 98 women (in the five main maternity units in Liverpool) gave birth to infants with severe malformations, principally of the central nervous system.

A formiminoglutamic acid (FIGLU) excretion test was done as soon as the malformation was diagnosed prenatally, or within 2 to 3 days of delivery for all 98 mothers. The assay technique, using high-voltage electrophoresis, was that described by Hibbard.[5]

We tried to match each mother with one delivered of a normal infant in the same hospital. These controls were selected with respect to the following criteria: age (±2 years), parity (±one pregnancy, except primiparæ who were matched with primiparæ), and the time of conception and gestation (±2 weeks).

Of 98 mothers of malformed infants, 54 were matched satisfactorily with controls.

Assays of FIGLU excretion were completed prior to collation with clinical data.

RESULTS

The results of FIGLU excretion tests in these patients are shown in the accompanying table.

RESULTS OF FIGLU EXCRETION TESTS IN 98 MOTHERS OF MALFORMED INFANTS

Mothers	FIGLU excretion test		Total	% FIGLU-positive
	Positive	Negative		
Of all malformed infants ..	61	37	98	62
Of all infants with C.N.S. malformations	48	25	73	66
Matched pairs:				
Mothers of malformed infants	35	19	54	65
Mothers of normal infants	8	46	54	17
Mothers of infants with C.N.S. malformation	24	11	35	69
Mothers of normal infants	6	29	35	17

DISCUSSION

There seems to be a significant relationship between malformation of the fœtus and defective folate metabolism in the mother. 11·4% of 167 women attending one of the hospitals in this study had positive FIGLU tests.[4] The women delivered of malformed infants show an incidence which is approximately 5 times this normal.

The results are based on the investigation of a metabolic defect present in late pregnancy or immediately after delivery and do not necessarily indicate that such a defect was present at the time of embryogenesis. Furthermore, a positive FIGLU test may indicate defective absorption or metabolism rather than deficient intake of folate. The familial occurrence of serious nervous system malformations might be mediated, in some instances, through a genetically determined defect of folate metabolism.

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KETOSIS-RESISTANT YOUNG DIABETICS

DIABETICS below the age of 40 (" growth-onset" diabetics), who do not develop ketoacidosis even in the absence of insulin therapy, have recently attracted much attention. In tropical countries some characterisation of such cases is being attempted as regards body-build, insulin-dosage, or response to oral sulphonylureas, although no distinct biochemical or metabolic variation has yet been ascribed to this group.

Stephen and Kinsell [6] have suggested that there is a significant islet-cell reserve in cases which respond satisfactorily to a very low carbohydrate diet and treatment with sulphonylureas.

In this Institute 27% of cases with " growth-onset " diabetes did not develop ketoacidosis when no insulin was given to them for periods of up to 30 days. [7] Plasma-insulin-like activity and response to oral sulphonylureas have been studied in 15 cases of ketoacidosis-resistant " growth-onset " diabetes. This group comprised 8 males and 7 females, with ages ranging from 12 to 32 years (mean 23 years). The duration of the disease in these cases was 2 weeks to 5 years (mean 13·55 months); in 8 cases the duration of the disease was less than 1 year.

The plasma-insulin-activity values, done by the rat-

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