Sweat testing for cystic fibrosis

Sir,

Bray et al. (Archives, 1978, 53, 483) conclude that the mass screening of newborn babies with chloride ion-selective electrodes is a feasible method for the diagnosis of cystic fibrosis; they also suggest that this procedure might be used in conjunction with meconium analysis to reduce the incidence of false positive results.

In 1972 we reported the details of a child with proved cystic fibrosis in whom raised sweat sodium but normal sweat chloride levels were obtained during treatment with cloxacillin (Griffiths and Bull, 1972). Attention was drawn to the implication of the finding with regard to screening procedures, and the suggestion was made that, in at least some children with cystic fibrosis, the cloxacillin radical might replace the chloride ion in the sweat.

Infants might also be exposed to drugs administered to their mothers (either by transplacental passage or excretion in the breast milk). Although Beechams Ltd have no direct information on cloxacillin, after an oral dose of 250 mg of fluocoxacillin, amniotic fluid concentrations of less than 2.5 μg/ml and breast milk concentrations of less than 0.1 μg/ml were obtained (personal communication).

When mass screening for cystic fibrosis is to be undertaken using methods based on the estimation of sweat chloride levels, it is therefore prudent to inquire whether the infant is receiving medication from any source and, if so, to interpret the results with caution.

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Dr Bray and co-workers comment:

We welcome the opportunity of commenting on the letter from Drs Griffiths and Bull. The instance mentioned of a child with proved cystic fibrosis in whom normal sweat chloride was obtained together with raised sweat sodium (Griffiths and Bull, 1972) is known to us (Bray et al., 1975). One of us (P.T.B.) came across a similar case about 2 years ago, giving credence to the view that ionised penicillamine displaces the chloride in excreted sweat.

These are instances of false-negative sweat chloride readings, but there is no evidence yet from our 1205 tests of any false-negative cystic fibrosis sweat chloride (Bray et al., 1978); the statistical chance of this is in any case small. Nevertheless, the two cases cited above do give the cause for concern that must arise for false negative results of any screening programme. The aim must be to recognise their cause and eliminate them.

The above two cases may be related to the observation (di Sant’Agnese, 1975) that IM injection of aldosterone lowers sweat electrolyte levels in patients with cystic fibrosis to the normal range. These several cases suggest a need for a systematic investigation of the effect of medication on sweat ion levels in general as well as in cystic fibrosis cases.

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References


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