integrating them, and beginning to answer the very questions that are raised in this article—that is, what is the need for protection, what is the need for work, who should carry this out, and how should it be supervised?

If we follow the DHSS guidelines I will gladly be handing over the chairing of such conferences to my social work colleagues. I hope I will continue to be present and that myself and my colleagues will still continue to do assessment work with families that I hope will assist conferences to make the sort of decisions that sadly do not seem to have occurred in Drs Chapman and Woodmansey’s experiences.

References

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Sir,
The Society of Clinical Psychiatrists state that ‘case conferences are inherently ineffectual’. I disagree. Child abuse case conferences are often ineffectual, but not inherently so.

The case conference is like a ship, and its aim should be to reach a stated destination. It is 14 years since the Tunbridge Wells study group made the child abuse case conference its cardinal recommendation. Is it any wonder that in that time the ship has acquired some barnacles? In addition, too many passengers have come on board.

A case conference can exchange information, decide on registration and the nomination of a primary worker, and offer to the primary worker various forms of support.

Bearing in mind this aim, I believe:
(i) Too many people attend, including those who have neither information nor an involvement with later management.
(ii) Information is poorly given. It should be the aim to circulate written information before the day of the case conference.
(iii) Far too much discussion takes place on the details of management. This can only be a matter for the primary worker and even if a valid decision is made at a case conference it is, as stated in the clinical psychiatrists’ document, only valid for that moment. ‘The decision cannot be made once and for all’.1
(iv) Most minutes are ineptly written, chairmen not being skilled as committee clerks. The anxieties of professional workers can be discussed without being minut ed. Minutes should record the location and constitution of the meeting with the decisions taken.

The case conference is a working method. Without it professionals would again begin to act in isolation. Without it the choice of primary worker would be seen as arbitrary and as setting one group of professionals above the others.

So I agree with much of the thinking of the Society of Clinical Psychiatrists but not with their conclusion. Without the case conference cooperation in child abuse would cease and the anxiety of professional workers would not be allayed.

References

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The sweat test

Sir,
Dr Littlewood’s annotation on the sweat test is to be welcomed as misdiagnosis of cystic fibrosis is a continuing and serious problem. We agree that most misdiagnoses can be avoided if experienced laboratory staff perform the sweat test with meticulous attention to detail and the results are interpreted in the light of the clinical findings.

Two points arise from the annotation, however, that we feel need further comment. Contrary to Dr Littlewood’s opening paragraph, the sweat test in most hospitals does not usually imply measurement of both sodium and chloride. In our experience many centres measure only one ion, and there is a need to reinforce to both biochemists and paediatricians the importance of measuring sodium and chloride. Secondly, we do not support Dr Littlewood’s conclusion about the use of pancreatic function tests. Diagnostic difficulty is more likely to occur in the 10% of patients with cystic fibrosis who have adequate exocrine pancreatic function, and it is in these patients that further investigation of pancreatic function may not be helpful.

In our own prospective study of 344 patients over two years 441 sweat tests were performed at our hospital, and only nine fell into an equivocal group. One of these nine was subsequently confirmed to have cystic fibrosis. The other eight patients all had sodium values considerably higher than the chloride values; they all had normal pancreatic function as judged by normal results of faecal chymotrypsin and para-aminobenzoic acid tests and, in two patients, normal secretin-pancreozymin stimulation tests. It was the interpretation of the results of their sweat test together with their natural history that excluded the diagnosis of cystic fibrosis rather than reliance on normal pancreatic function tests. We think that it is misleading to suggest that demonstration of abnormal pancreatic function is the gold standard for the confirmation of the diagnosis of cystic fibrosis in equivocal cases. Until deoxyribonucleic acid technology can identify the gene the
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diagnosis of cystic fibrosis will depend on the correct interpretation of both sweat sodium and chloride in the light of the clinical findings, with support from investigation of pancreatic function.

Reference

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Dr Littlewood comments:
Dr Green and colleagues observe that many laboratories do not measure both sweat sodium and chloride; thus, in marginal cases, it is impossible to determine whether the chloride is higher than the sodium. While I would support the value of their observation that the chloride is commonly higher than the sodium in patients with cystic fibrosis who have marginal values and agree with their advice to measure both electrolytes, the higher chloride value in patients with cystic fibrosis can only be regarded as useful supportive evidence for the diagnosis.1

I would not accept that ‘it was the interpretation of the results of the sweat test together with the natural history that excluded the diagnosis’. Unfortunately, Dr Green and colleagues provide little evidence that their small series of children with marginal results of the sweat test did not have cystic fibrosis. Certainly, normal results of a para-amino-tenzoic acid test and faecal chymotrypsin test cannot be taken to exclude a minor degree of pancreatic involvement.

All evidence to the present suggests that demonstration of normal pancreatic function by pancreozymin secretin stimulation, particularly a normal bicarbonate secretion, remains the most reliable supportive evidence for the exclusion of cystic fibrosis.2 3 4

Our experience at the Leeds regional cystic fibrosis unit, derived from comprehensive assessment of over 200 patients with cystic fibrosis, in addition to many referred where the diagnosis was in doubt, suggests that individuals with cystic fibrosis who have marginal sweat electrolyte values usually have additional supportive evidence—for example, pseudomonas lung disease, obvious malabsorption, and pancreatic abnormality. Furthermore, there seems to be no direct correlation between the degree of sweat electrolyte abnormality and either the severity of the pancreatic lesion or the clinical score.

Thus I would reiterate the suggested diagnostic criteria as previously stated.4 While the relation of sodium to chloride is a valuable observation when a reliable sweat test gives a marginal result, confirmation or exclusion of the diagnosis must thereafter depend on findings other than the sweat test.

It should be re-emphasised that the common diagnostic difficulties and not uncommon actual mistakes usually result from an incorrect determination of the sweat electrolytes by the biochemist and an uncritical acceptance of the result by the clinician who also fails to show any gastrointestinal lesion or request a confirmatory sweat test at a later date.

References

Small for dates babies: are they really a problem?

Sir.

Doctors Jones and Robertson conclude, in their recent good paper, that small for dates (SFD) babies of 37 weeks’ gestation or more pose few neonatal problems.1 That this is so in their neonatal unit is because of the unit’s admirable and justified reputation and of its surrounding population, in general, with a high standard of antenatal and other health care.

The only reason I write is lest the title of their paper masks an important background that has two basic features. Firstly, of infants born with low birth weight in the developing world, it is estimated the component of SFD infants over preterm infants is nearly three times greater than that in the developed world (80% as opposed to 30%). This suggests that a reversible condition exists: prevention of intrauterine growth failure. Under conditions in the developing world many of these SFD infants contribute to very high perinatal and infant mortality and morbidity.

Secondly, the important question of outcome. Experienced neonatologists, like these authors, know that clinically there are broadly two kinds of SFD infants: those that ‘do well’ and those who do not. Recent work has carried this further.2 Ultrasonography and anthropometry suggest that proportional fetal smallness, with small head size, in fetuses destined to be SFD infants leads to those who exhibit poor postnatal catch up growth, including that of head (and brain?) size. By contrast, those fetuses who are disproportionately small (with ‘normal’ size fetal heads) seem to exhibit good postnatal catch up. Perhaps, then, there is a distribution curve for outcome—the non—‘catch uppers’ at one end, with the vigorous ‘catch uppers’ at the other.

I suspect, because of the reasons I have suggested, that if Doctors Jones and Robertson followed their sample of SFD infants they would find them to be at the latter end.

May I repeat that my comments are the opposite of criticism of their good paper; only that their title might lead us to complacency about all SFD infants.