Annotations

Alcohol and the fetus

'Do you think that the time has come for an annotation on the foetal (sic) alcohol syndrome and whether or not it exists?' (Editor, Archives)

The continuing debate about the existence of the fetal alcohol syndrome only continues because the Editor's seemingly simple question is in fact two questions which may have different answers:

Question 1: Can alcohol damage the fetus? The answer is unequivocally, Yes.

Question 2: Are the fetal consequences sufficiently clearly defined as to justify the use of the word 'syndrome'? The answer is unequivocally, Maybe.

When we attribute clinical states to particular agencies the specificity of both is variable. Chicken pox is a highly specific rash attributable to a highly specific virus. Down's syndrome is a fairly specific clinical entity attributable to a very specific genetic fault. Congenital rubella syndrome is a largely non-specific spectrum of defects attributable to a highly specific virus. Congenital microcephaly is a specific clinical entity attributable to all manner of things.

We need, therefore, to examine the specificity of the effects and of the agency; that is to say, the fetal consequences of alcohol (as exemplified by alcohol abuse) and the confounding variables that may be associated with alcohol.

Fetal alcohol effects

'The clinical features of FAS (fetal alcohol syndrome) are as follows: (1) prenatal and postnatal growth deficiency, (2) CNS dysfunction, (3) a particular pattern of facial characteristics, and (4) major organ system malformations.'

'In any specific patient each individual anomaly can vary in severity and any subcombination of anomalies can occur. When a person's cluster of anomalies is inadequate for confident syndrome identification, we suggest the term "possible fetal alcohol effects" be used.'¹

(1) Babies damaged by alcohol in utero are often light for dates and may be preterm as well. Head circumference is usually reduced but is in proportion to length. There are two unusual features of the growth retardation associated with prenatal expo-

sure to alcohol. Firstly, postnatal weight and height may *diverge* from the lower centiles. Secondly, bone age is *not* usually delayed.

(2) The neurodevelopmental disorders associated with alcohol exposure are very variable. Irritability in the neonatal period and moderate mental retardation are perhaps the most characteristic. 'In some patients, behaviour and intelligence can be altered without associated diagnostic changes in growth, head circumference, or facial morphogenesis'.¹ The limited information available on neuropathology indicates that abnormalities of neuronal migration are common.

(3) The facial characteristics have proved to be the most controversial aspect of the fetal alcohol syndrome debate. Descriptions of facies are notoriously unhelpful and some of the published photographs seem to bear little resemblance to one another. Nevertheless, those with wide experience score about 80% when 'face watching' on the newborns' nursery. (Having spotted pyloric stenosis from the facies yesterday, who are we to carp?).

(4) Structural malformations, so important in most syndromes, only affect a minority of alcohol damaged babies. Cardiac, neurological, and skeletal defects predominate but *any* organ may be affected.

Does this description constitute a syndrome? Or even a definition? More to the point, does it really matter? Possibly the use of the term fetal alcohol syndrome has diverted energies from preventive strategies (which are difficult) to semantic arguments (which are easy). 'Syndromes' have been described in relation to a number of drugs—phenytoin (hydantoin), warfarin—but the important thing is for people to be aware of the risks. 'Fetal alcohol effects' may be a better term.

Definitions are necessary for determining prevalences, for comparative studies, and for other purposes. Where no confirmatory laboratory tests are available (as is the case for fetal alcohol syndrome) definitions are necessarily arbitrary and may not be the same for all people (cf febrile seizures). It is sufficient that in communications where the definition is important, it is stated.

Alcohol and confounding variables

Alcohol *abuse* tends to be associated with poor nutrition, ill health, smoking, and use or abuse of other drugs. Some of these factors are relatively easy to ascertain (for example smoking) and data can be appropriately adjusted.² For others, routine histories are notoriously fallacious, and detailed individual case studies may contribute more to a broad understanding.³

There are known (and doubtless unknown) interactions between alcohol and other drugs. The effects of alcohol on the metabolism of folic acid and other essential nutrients may aggravate dietary deficiencies. These are among the factors that make it very difficult to assess the possible risk of moderate and light drinking in pregnancy. There are also bound to be individual variations in response to a given alcohol intake. Binge drinking (a large quantity in a short time) may pose quite different dangers from regular consumption of smaller quantities. It is scarcely surprising that attempts to establish a dose-response relation are only successful if very crude scales are used.

There is indirect evidence of the specificity of alcohol. Firstly, the constellation of defects that constitute the full fetal alcohol syndrome have not been described in the offspring of the malnourished, heavy smokers or drug addicts, except when they are *also* abusing alcohol. The other factors may modify the outcome but are not sufficient to cause the same constellation of problems. Secondly, using animal models (and recognising their limitations), alcohol seems to be very specific in its embryopathic activities.

The zero option

Another area of lively debate relating to fetal alcohol syndrome is the 'safe dose'. Alcohol intakes above 80 g daily pose a major threat. There are sufficient grounds to accept that daily intakes of 20 to 80 g may be harmful. What of smaller intakes corresponding to one drink a day? There is little positive evidence that in the absence of other adverse factors this carries a significant risk to the fetus. Absolute harmlessness, however, can no more be proved for alcohol than for any other drug, and by the same token it makes sense to avoid alcohol as one would avoid unnecessary drugs, especially in early pregnancy. The total abstinence advocated by the United States Surgeon General and by the Royal College of Psychiatrists (at the second time of asking) is valid, *not* because we know

that small doses are dangerous but because we cannot know that they are safe.

If it exists, why don't I see it?

Perhaps you are privileged to work in an area where the citizens live pure lives, or at least separate their impurities from their pregnancies. Davis *et al*⁴ found that 49% of 973 antenatal women were abstaining totally, and only one admitted to consuming more than 70 ml alcohol per day.

Perhaps you are not looking hard enough. Beattie *et al*⁵ looked in the west of Scotland and found 40 cases, of which 22 had been born in 1980–81. At least 25% of the 40 had congenital heart lesions. Sixty per cent were in social class V (cf 7.7% for Scotland). Postnatal growth was poor, and catch up growth was not seen in any child. Every one of the 40 had a neurodevelopmental problem, although this was mild in 19.

The human factor

Would the last bastions of disbelief have crumbled had we been discussing hashish rather than alcohol? Sins are other people's bad habits, and alcohol abuse is drinking more than we drink. Would the fight against smoking have been even harder if lung cancer had been called 'cigarette syndrome'? There is enough evidence to support the belief that alcohol damages far more unborn babies than does rubella. For rubella we have a National Rubella Council with royal patronage and a national programme for eradication.

It is time to stop bickering about the meaning of words and to tackle the real problem.

References

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- 5 Beattie JO, Day RE, Cockburn F, Garg RA. Alcohol and the fetus in the west of Scotland. *Br Med J* 1983;**287**:17–20.

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