We do think that it is important for every child to have experience of warm physical contact, and we hope that young children of both sexes will continue to be cuddled by parents of both sexes. This contact is the ingredient for healthy development in any child. It has, however, nothing to do with 'sexualisation' of relationships and we entirely agree with Clarke *et al* on the abusive nature of sexualist parent-child interaction.

Simple concepts of power do not quite fit when dealing with clinical issues of sexual abuse of children in the family. We have found it much more helpful to talk about the notion of parental responsibility and structural dependence of the child. Whatever happens between parents and children, parents are responsible for protecting their children and are in any circumstance held responsible for their actions towards the child. This also clarifies the issue that however seductive the child may in fact be, she or he can never in any way be responsible or to blame for the abuse.

Child sexual abuse within the family has been brought up mainly by professionals of both sexes and is only now being taken up again by feminists, usually being put on very much the same level as rape. In child sexual abuse there are certainly some cases similar to rape. The presentation and dynamic, however, of long term child sexual abuse within the family has otherwise very different characteristics from those of rape in terms of structural dependence, intergeneration of boundaries, parental care, and issues of trust, as well as violence. The context of dependency on a parent accounts for the fact that actual physical violence is comparatively rare in long term child sexual abuse within the family. This does not, however, mean that we do not find powerful threats of violence and that the abusive interaction may not be as damaging, or even more damaging, than rape is. It makes, however, for a very different form of abusive dynamic and interaction that require different forms of intervention.

Finally, we certainly do not have a 'cosy view' of sexual abuse and we regard this statement, among others in the last paragraph, as emotive and unhelpful. Nor do we in any way deny the facts of violence or the threat of violence. On the contrary, these issues were very much behind our taking the suffering of sexually abused children seriously, and starting the present programme. Feedback from the girls and boys involved, and from the parents, has satisfied us that our approach is to some degree helpful. We agree, however, that this approach may have to be modified as our own experience increases and that of others is added. In addition, as we indicated in our paper, careful evaluation will have to be undertaken in future.

Pulmonary interstitial emphysema

Sir,

The paper by Greenough *et al*[1] is very useful in stating in a trial form what has been suspected anecdotally. We have, however, just completed our own much smaller series of preterm infants with pulmonary interstitial emphysema and far from being able to point a finger at high ventilatory pressures, we were rather concerned to note that in several cases the peak inspiratory pressure was low, that is less than 22 cm of water, and the length of ventilation was often short—sometimes less than three days. In no case did we have a major problem with malposition of the endotracheal tube. Fortunately we did not have the problem of a large percentage of these children developing pneumothoraces when pulmonary interstitial emphysema had developed and we continued on a low rate ventilatory regimen.

There are two points of concern. Is it really justifiable to take all babies from the age of 24 to 35 weeks of gestation and claim that they are all suffering from the same problem and therefore the aetiology of pulmonary emphysema in all of them is the same. Secondly, the conclusion that because they found a positive correlation between pulmonary interstitial emphysema and high peak pressure ventilation, a controlled study using fast rate ventilation from birth is indicated, is not justifiable. They looked at only four aetiological associations, only one of which was significant. Surely they are falling into the trap of assuming that the aetiological factor with positive correlation is the most important, whereas they may not actually have looked for the relevant aetiological factors.

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Dr Greenough and co-workers comment:

We thank Dr Rose for his interest in our paper,[1] it is unfortunate that we can make no comment on his results which are not stated. We would, however, point out that it is particularly dangerous to draw negative conclusions, for example concerning the possible aetiological associations of pulmonary interstitial emphysema, using the small numbers he states were investigated in Inverness.

In our own study of the possible aetiological factors associated with pulmonary interstitial emphysema—gestational age, birthweight, type of resuscitation, timing of ventilation, endotracheal tube position, and use of high peak pressures—only the latter two were found to be significantly associated with the development of the disease. Dr Rose is mistaken in stating that we implied that the aetiology was the same in all infants.

Unlike the experience of Dr Rose, there are now several reports suggesting, as we had done, that fast rate, low pressure ventilation was beneficial in pulmonary interstitial emphysema.[2–4] Recently Field *et al* [5] showed that infants ventilated at fast rates (greater than 100/min) tend to be apnoeic and as a consequence are easier to ventilate. Certainly the infant's spontaneous respiration during ventilation can be disadvantageous[6] and its suppression may be another reason why fast rate, low pressure ventilation in so many cases seems to be beneficial in pulmonary interstitial emphysema.[1–4] In view of those reports and the high mortality and morbidity of pulmonary interstitial emphysema, surely it is justifiable to suggest that a different form of ventilation should be considered in an attempt to reduce the incidence.

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Reflex anoxic seizures

Sir,

I am grateful to Dr Bower[1] for drawing attention to reflex anoxic seizures but I take strong issue with his preference for 'pallid syncope'. Of course there are many children who get pale and floppy and unresponsive, who may have vasovagal syncope, and for whom the term pallid syncope is perfectly acceptable. But what I mean by a reflex anoxic seizure is 'a particular type of fit which is neither epileptic nor due to breath-holding, but rather results from brief stopping of the heart through excess activity in the vagus nerve'. A typical example in the form of venepuncture fits has recently been published,[2] but it is important to recognise that the patients do not always go pale, nor do observers always notice the pallor. I believe I have seen more anoxic seizures after ocular compression than anyone else (over 300) and can assert that pallor is not constant, particularly is it not observed in children who are not reported as pale in the natural attacks.

An anoxic seizure is very much a seizure, as Gastaut has well described. It may be violent and dramatic.[3] It is not sufficient to call such an event convulsive syncope for there is yet another type of seizure which must be distinguished, that is the anoxic-epileptic seizure[4] in which the syncope is concluded by a train of spike and wave with clonic component. If anoxic seizures are not specifically recognised, they will be called epileptic, even, I suspect, in 1985.

Although I think precision in terminology is helpful in communication between doctors, different words may be more helpful with patients and parents. When I talk about reflex anoxic seizures I call them 'fainting fits and not epileptic fits'. I explain how the vagus makes the heart stop, but that it can only keep on telling the heart to slow down while it is getting enough blood so as soon as it does not the heart will speed up again and all will be well. I nowadays use ocular compression mainly as an aid to reassurance, and agree that in most cases a meticulous history will give the diagnosis. I do not think, however, that paediatricians need be worried about unsubstantiated dangers of the technique when it has to be used for reassurance, investigation, or research.

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Sir,

I enjoyed reading Dr Bower's annotation.[1] He makes the point that most paediatricians would be reluctant to use the oculo-cardiac reflex to show increased vagal reactivity in children with the pallid form of breath holding. May I suggest a technique that I have found very useful when given a suggestive story of this disorder by the parents. Explaining what the disorder is about to the parents, one asks that the child be laid down on the examination couch without preamble and that the upper garments be removed. The doctor stands ready with stethoscope and, at the first sign of a yell which is certain to occur in response to this sudden 'rough' handling, one is easily able to detect the dramatic slowing of the heart which will accompany the cry. The doctor should prepare himself to be moderately frightened by how dramatic the pallid breath holding attack can be.

I presented a paper on this topic at the Second Rhodesian Medical Congress in 1972 which I entitled 'Breath-holding—an Adams-Stokes attack'.

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Neck radiographs in croup syndrome

Sir,

In recent correspondence, Dr Porter[1] implied that in Aberdeen the initial routine investigation for possible epiglottitis was lateral neck radiographs rather than laryngoscopy. We feel it would be unfortunate if that comment and its potential influence on the practice of others went without reply.

Since the mid 1970s The Royal Aberdeen Children's Hospital has practised a policy of protective intubation in cases of severe croup, an experience we are currently reviewing. Our routine in children with severe stridor is not to attempt clinical or radiological diagnosis with their potentially dangerous delays, but to proceed to laryngoscopy by experienced staff (ear nose and throat and anaesthetic). There have been no problems to date with