midwife, thought that the shape of the head was due to modelling and that it would be fine. We were later informed, however, that the child was microcephalic and that his head would grow at a slower rate than his body, leaving him mentally retarded. We were given no hope for the future.

After one week we took the child home and cared for him. Our emotional condition at this stage is very difficult to describe. The midwife who visited us suggested that the child would be better off in care, at least temporarily. After a lot of tears and heart searching we decided that at least until we could cope the baby should be fostered. He was in care for two days only before being taken to hospital with feeding difficulties.

We later went for genetic advice, and the doctor, after hearing our story, immediately thought that the problem was due to the fact that my wife had a bicornuate uterus and that the baby had suffered from cranial constraint. This was the first time that this theory had been mentioned to us.

On hearing that our son was in hospital my wife visited the child often. I was more reluctant and frankly used to get very upset. The staff there said they were not treating the child as microcephalic but simply as a baby with an unusually shaped head. At no time did they put any pressure on us to take the child home. This at the time was a great comfort.

After a further two weeks, I came home from work and told my wife of my decision to bring the child home and bring him up whatever his condition. She was delighted. We then visited the hospital, and the sister told us the good news of the results of the baby's tests, which proved that he was perfectly normal. You can imagine our delight. I feel it is important to emphasise that we had decided to take the child before we knew the results of the tests.

On reflection, I do not believe that anyone could criticise my wife and me for the way we acted. We were told so many different stories and so many different theories by so many different doctors that the whole episode from our point of view was a catastrophe. I am now happily able to tell you that our son is perfect although a little boisterous.

RALPH and JUILLET DOYLE

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Unexplained carotinaemia and baby food

SIR,—We have discussed the matter raised by Dr George Brown (p 1596) with our consultant paediatrician. He says that it is well recognised that a large amount of carotene in the diet may cause a person to look yellow, and the way of correcting this is to stop eating carrots. It does not seem to cause any problem apart from the yellow colour of the skin. It does not mean that there is anything wrong with the baby products such as Gallia which include carrot in the formulation, as it can occur in any baby who is given carrots in any form. He says that it does not cause anaemia.

Although carotinaemia is well recognised, it does not seem to be very common. It may be that some people have a particular tendency to get carotinaemia when they take carrots. In any event a large quantity of carrots in relation to the baby's size would have to be consumed.

We introduced Gallia in the United Kingdom in the autumn of 1981 and have sold several million jars. Apart from the letter from Dr Brown we have had only two other cases reported to us, one from Barnes (south west London) and the other from Cornwall.

M T MCHATTON
Managing director

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Thrombosis in systemic lupus erythematosus

SIR,—Arterial and venous thrombosis is obviously a serious complication of systemic lupus erythematosus, but I would like to point out that Dr M L Boey and others (8 October, p 1021) have not included in their data the number of patients with nephrotic syndrome in the group with and the group without lupus anticoagulant. This is an important omission because nephrotic syndrome per se is associated with an increased incidence of thromboembolic complication, including peripheral venous thrombosis, pulmonary thromboembolism, arterial occlusions (coronary and cerebral), and renal vein thrombosis.[1] Patients with nephrotic syndrome have a modest rise of factor V, factor IX, combined factor VII, factor X, and fibrinogen and accelerated thromboplastin generation.[2,3] Antithrombin III concentration may also be depressed.[4] Recently, platelet activation and hyperaggregability as a consequence of the nephrotic syndrome were also reported.[5,6]

As renal disease is common in systemic lupus erythematosus, and 40% of patients with lupus nephritis presented with nephrotic syndrome,[7] it is mandatory to include this information before drawing any conclusion about the association between thrombosis in systemic lupus erythematosus and other variables like the presence of a circulating anticoagulant.

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**We sent a copy of this letter to Dr Boey, who replies below.—ED, *BMJ*.

SIR,—Of the 60 patients tested for the presence of circulating lupus anticoagulant, five had nephrotic syndrome—three (10%) of the 31 patients with lupus anticoagulant activity and two (7%) of the 29 without.

We did not find a significant correlation between the presence of the circulating lupus anticoagulant and nephritis or nephrotic syndrome in our patients. Of the 18 patients with thrombotic episodes, only two had nephrotic syndrome.

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A letter from Margaret Mead's daughter

SIR,—In the context of a discussion of the deliberate falsification of results, Dr Terry Hamblin (30 July, p 355) refers to the researches of my mother, Margaret Mead, in Samoa, citing Derek Freeman's recent attack on her work as his source.[1] It seems to me probable, however, that his actual source is one of the popular magazines that did their best to amplify the debate. Dr Hamblin's text strongly implies that Freeman challenged the "reliability" of the Samoan work as fraudulent rather than erroneous. In fact, although Freeman's work is rich with innuendo, he nowhere makes an accusation of deliberate fraud.

This is not the place to discuss the ways in which I believe my mother's interpretation of her youthful fieldwork requires correction or the further distortions introduced by Freeman, which have been fully discussed in many scholarly reviews. Because I believe, however, that debate and correction are central to scientific progress, I think Dr Hamblin does us all a disservice by sloppily equating even a vitriolic attack, conducted in a highly inappropriate glare of publicity, with an accusation of fraud. The only such accusation that has been before the reading public is the one implied in his letter.

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1 Freeman D. *Margaret Mead and Samoa: the making and unmaking of an anthropological myth*. Cambridge (Mass) and London: Harvard University Press, 1983.

Association between use of cotton tipped swabs and cerumen plugs

SIR,—I thank Dr Peter Baxter (29 October, p 1260) for drawing attention to one of the most common and important problems encountered in child health clinics. The observation that the use of cotton tipped swabs by most mothers is an important cause of injury to the external canal and the formation of cerumen plugs requires further evaluation.

Dr Baxter reports 90% of the children in his study with cerumen plugs had their ears cleaned regularly with the cotton tipped swabs; this does not appear to be different in any way from the day to day observation in child health clinics all over the country. The use of cotton tipped swabs by most mothers is a routine washing ritual that they find very convenient and that is almost always recommended by the family doctors and clinic doctors. If the formation of cerumen plugs is associated with the use of cotton swabs, possibly because of some unknown mechanism, then perhaps a change in the chemical nature of the commercially available swabs might be necessary. Furthermore, undoubtedly, probing of the external auditory canal with cotton tipped swabs is associated with deafness, injury, and otitis externa. I agree with Dr