information be bounced around the media before critical analysis and evaluation by the medical or scientific communities.

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1 Marsh P. Porton International, a herpes vaccine, and the CAMR. *Br Med J* 1990;**300**:1291. (19 May.)
2 Skinner GRB. Nice work for everybody except the patient. *Vaccine* 1989;**7**:382-4.

SIR,—The news article on Porton International[1] was misleading and contained some important inaccuracies.

Mr Peter Marsh says that the herpes vaccine developed by Dr Skinner of Birmingham University took much longer than expected to progress through clinical trials in the United States and that the trials have now been abandoned. Both statements are inaccurate and misleading. As readers will be aware genital herpes is a complex condition and there are several aspects of the disease that can be addressed by the product developed by Dr Skinner. Initially Porton intended to concentrate on the use of the product as a vaccine to prevent or mitigate initial infection. But after an analysis of the scientific and commercial potential of the product and discussion with all the parties concerned in the development programme it was decided that it should be evaluated as a treatment to alleviate the condition in patients with herpes. Having taken this decision well structured double blind clinical trials for this indication were set up in the United States at three centres, and these have been carried out to the highest standards. While it can be argued that had we proceeded with trials for prevention of the infection they would have been completed earlier these trials were conducted in the fastest time possible consistent with trial design, patient recruitment, and the requirements of the Food and Drug Administration.

Mr Marsh says that Porton has taken a decision to abandon the trials. This is untrue. The trials have been completed on time, fully analysed, and a preliminary indication of the results made public in a letter to shareholders on 19 April.

The aim of clinical trials is to conduct objective experiments to establish whether a particular treatment is beneficial or not. Inevitably not every trial will produce a positive result in terms of showing clinical benefits. But well conducted trials can have immense value scientifically, and we believe that this is the case with our herpes trials. Though we are disappointed by the lack of clinical efficacy, we are proud to have carried out this work and supported an idea derived in a British university. Far from having abandoned this project we are still considering ways in which it can be brought to the market for the benefit of patients and their consorts.

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1 Marsh P. Porton International, a herpes vaccine, and the CAMR. *Br Med J* 1990;**300**:1291. (19 May.)

Meningococcal meningitis

SIR,—I was pleased to see that the editorial by Drs Philip D Welsby and Clayton L Golledge emphasised the variability of the rash in meningococcal meningitis because most short paediatric textbooks for junior hospital staff or general practitioners refer only to petechial or purpuric rashes.[1]

I recently reviewed the charts of 91 cases of acute meningococcal infection in children in the north east of Scotland. In all five cases of septicaemia without meningitis a purpuric rash developed eventually, but in four this was initially macular and evolved over one to eight hours. Of the 20 cases of meningitis with positive blood culture, six never developed a rash, six had macular rashes (one becoming purpuric), and in only eight was the rash purpuric from the onset.

Among the remaining 66 cases of meningitis blood cultures were negative (56 cases) or not taken (10 cases) and 26 never developed any rash, although 26 did have a petechial or purpuric rash at some stage in the illness. In 20 children the rash was initially recorded as macular or morbilliform and progressed to haemorrhagic skin lesions in only five.

This is important because a non-specific rash in a febrile child is often taken to indicate a viral illness, engendering a false sense of security. Two of the above cases were initially diagnosed as measles, one as chickenpox, and one as rubella—fortunately without ill effect. In one other case the rash was recorded as "macular—not a meningococcal rash" —and antibiotics were withheld until the child was found moribund the next morning.

In an ill child, particularly during an epidemic, fever and any rash may mean meningococcal infection and penicillin can be life saving.

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1 Welsby PD, Golledge CL. Meningococcal meningitis. *Br Med J* 1990;**300**:1150-1. (5 May.)

Illness preceding sudden infant death

SIR,—Dr Ruth E Gilbert and colleagues found that minor signs of illness, including snuffles and cough, preceded sudden infant death.[1]

We present data on the syndrome in the southern hemisphere. Because of the marked increase in the number of sudden infant deaths in winter a comparative study of the syndrome was conducted in Melbourne (latitude 38°S) and in Brisbane (latitude 28°S) where the winter is much less severe.[2] The study yielded an incidence of the sudden infant death syndrome of 1·6/1000 live births in Brisbane compared with an incidence of 2·2 in Melbourne and showed that infant deaths under 3 months showed no seasonal variation. One third of infants were aged above 3 months in Brisbane and half in Melbourne.

In Tasmania, Australia's most southerly state (latitude 41-43°S), the syndrome affects 4·4/1000 live births with nearly two thirds of affected infants aged more than 3 months.[3] Thus, in Australia as one travels southwards along the eastern coast, the incidence of the sudden infant death syndrome and the proportion of affected infants aged over 3 months both increase.

A recent study of the sudden infant death syndrome in New Zealand, which ranges from latitude 35°S to 47°S, used five broad geographical areas of increasing latitude.[4] The results show that there is a north-south gradient for the incidence of the syndrome—from 3·28/1000 live births in the north of the country to 6·46 in the most southern region. With the exception of the north of the South Island there is an increasing percentage of deaths over the age of 3 months with increasing latitude.

With the exception of the North Island of New Zealand, the populations of the areas mentioned above are of European origin, mainly Anglo-Saxon. In the North Island of New Zealand there is a large Maori population which has a high incidence of the sudden infant death syndrome (6·5/1000 live births) compared with that in the non-Maori population (3·8). Maternal and infant health care are good in Australia and New Zealand and of comparable standard. We also believe that in the studies reported above there has been consistency in the diagnosis of the sudden infant death syndrome.

The increased incidence of the syndrome with increased latitude in these countries suggests that one or more environmental factors are responsible for some infant deaths. The effect of this factor (or factors) appears to be most obvious in infants over 3 months. We believe that certain respiratory virus infections must be considered as the responsible factor. The seasonal and annual incidences of the sudden infant death syndrome correlate with the incidence of bronchiolitis in a community,[5] and tracheitis is commonly found in infants who die suddenly when aged over 3 months but not in younger affected infants or controls.[6] The incidence of the syndrome also correlates with isolations of respiratory syncytial virus in a population and, to a lesser extent, of influenza and para-influenza viruses.[7] These viruses can be isolated from the respiratory tracts of a significant number of infants aged over 3 months with sudden infant death syndrome, but not from infants under this age or from appropriate controls,[8] and they are known to produce apnoeic attacks in young infants.[9]

We agree that there are two types of infant deaths[10]—those occurring in infants under 12 weeks and those occurring in those over this age. We believe that most deaths in the older group are precipitated by certain respiratory viruses.

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Gall bladder function after endoscopic sphincterotomy

SIR,—The report by Mr L A Desa and colleagues of gall bladder dysfunction after endoscopic sphincterotomy is methodologically flawed and thus its conclusions are meaningless.[1]

Firstly, technetium-99m HIDA scanning is a technique designed to investigate patency, not function, of the biliary tree.[2] The true functional state of the gall bladder can be shown only by confirming its contractile response to cholecystokinin. Secondly, normal gall bladders may take up to four hours to accumulate isotope,[2] yet delayed images were taken at only two hours after injection. Thirdly, no conclusions can be drawn about individual patients because there are no control data. Each patient should have acted as his own control by having an isotope scan before sphincterotomy—without these data the report of any effect of sphincterotomy on the results of scintigraphic scanning is totally invalid. Finally, extrapolations