certain points made in the correspondence arising from the paper by Dr Peter J Fleming and colleagues.[1,2]

Three of the letters criticised the questioning of bereaved families within three to four hours of the infant's death, when the parents are distressed and apparently may be subject to "confusion and selective recall." After our son died time stood still, each minute was like an hour, and the accuracy with which my wife and I recall events is quite phenomenal. I believe that in the three to four hours before the parents were interviewed there was not time for any bias to occur. On the contrary, after such a tragedy parents want to read as much as possible about what has happened and only when information has been gleaned from books or other bereaved relatives could opinions be subject to recall bias.

The second point is that of the distress caused by media reporting. Anything published in any of the medical journals on this subject seems excessively liable to media hype. The fact that this article came up with some conclusions on both position and wrapping of infants has reduced the media distortion that usually occurs. Most bereaved parents, although they may be upset by media reporting, are grateful for any information important in the cause of sudden infant death.

I do not think that terms of position need be defined. The important point is whether the child was prone or not and a "trained individual" is not necessary to determine this, as Drs M P Wailoo and S A Petersen suggest. The quote from *The Complete Book of Babycare from Conception to Three Years* suggesting that babies may choke when placed supine is based on an unproved premise.

As bereaved parents we obtained a great deal of reassurance from the paper by Dr Fleming and colleagues. The agony of sudden infant death is the parents' feeling of inadequacy and helplessness. To give parents something to act on themselves, such as not placing the baby prone and reducing the amount of covers, is very helpful.

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**This correspondence is now closed.—ED, *BMJ*.

Decreased salivary epidermal growth factor in rheumatoid disease

SIR,—We have a case that may support the conclusions of Dr S M Kelly and colleagues.[1] A 58 year old woman with Sjögren's syndrome presented with symptoms of heartburn and retrosternal pain. Endoscopy of the upper gastrointestinal tract showed an aggressive form of erosive oesophagitis. Oesophageal motility studies showed the pressure of the lower oesophageal sphincter to be normal at 18 cm of water; oesophageal peristaltic activity was within the normal range.

She produced 0·7 ml of saliva in 20 minutes. After stimulation with malic acid the volume increased to 0·9 ml in 20 minutes. Epidermal growth factor concentrations were low (0·16 and 0·14 ng/ml in resting and stimulated saliva respectively). The volume of specimens was insufficient to permit measurement of bicarbonate concentration.

As the motility of the oesophagus was normal we concluded that the reduced output of saliva was important in causing erosive oesophagitis. The diminished volume of saliva may have deprived her of two important salivary components: bicarbonate, which would have neutralised any inappropriately refluxed gastric acid, and epidermal growth factor, which may have compromised mucosal regeneration.

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1 Kelly SM, Crampton J, Hunter JO. Decreased salivary epidermal growth factor in rheumatoid disease: a possible mechanism for increased susceptibility to gastric ulceration. *BMJ* 1990;**301**: 422-3. (1 September.)

SIR,—We were surprised that Dr S M Kelly and colleagues[1] did not mention our reports of a deficiency of salivary epidermal growth factor in rheumatoid arthritis.[2,3]

Like them, we showed reduced salivary volume in patients with rheumatoid disease and the sicca syndrome, but we found that the concentration of epidermal growth factor was higher in these patients so that the overall output was the same as in patients with rheumatoid disease who did not have the sicca syndrome. Thus, although we originally hypothesised that autoimmune disease of the salivary glands manifest by the sicca syndrome might predispose to a deficiency of salivary epidermal growth factor, our data suggest that reduced production in rheumatoid arthritis may occur by an independent mechanism. This could be drug related as indomethacin has been claimed to inhibit the production of epidermal growth factor.[4] We found no effect of aspirin in normal volunteers, but the possibility that reduced epidermal growth factor is another manifestation of the toxicity of non-steroidal anti-inflammatory drugs requires further investigation.

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Three types of erythromelalgia

SIR,—Drs J P H Drenth and J J Michiels classified erythromelalgia into three syndromes.[1] We believe that this may be an oversimplification.

We are caring for two patients with myeloproliferative disease who presented to our department with warm, erythematous painful feet and ankles. Symptoms were exacerbated by exercise and heat and temporarily relieved by cold, rest, and leg elevation. Both patients, therefore, have erythromelalgia associated with thrombocythaemia, but neither has responded to normalisation of the platelet count or to a trial of aspirin, although one patient recently responded to methysergide. Biopsy of affected skin showed no abnormality. We find it difficult to classify our patients using the criteria of Drs Drenth and Michiels. Equally, patients who fit their description of primary erythromelalgia but respond to treatment defy classification.[2-5]

The basis for the third variant of erythromelalgia (somewhat confusingly termed secondary erythermalgia) warrants analysis. As warm skin is an integral part of the erythromelalgia triad we find it surprising that thromboarteritis obliterans, arteriosclerosis, and vascular disease have been included.[6] Furthermore, the uncommon association with gout, connective tissue disorders, and neurological conditions makes us wary of using the term secondary erythermalgia in such cases. It has been suggested that such associations are coincidental.[7]

All is not red or white in the classification of erythromelalgia. We urge caution before accepting the authors' classification, which may encourage therapeutic nihilism.

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Training for care assistants

SIR,—We fully support Professor Elaine Murphy's comments on the need to improve the training, status, and effectiveness of care assistants.[1]

We have been running regular training courses for carers on the mental and physical wellbeing of elderly people for the past two and a half years. Anyone who works with or looks after an elderly person, including relatives, can attend. The project is supported by a charity. Despite having worked with the elderly for a long time most carers have had no formal training. Teaching people who work in different settings together gives them the opportunity to learn from each other and widen their views. We emphasise the value of the carers themselves, encouraging them to talk, nuturing their ideas, and attempting to improve their poor self image.

The problem is how to reach those who would like some training. Advertisements in journals are read by only a small proportion, and information is often not passed on by more senior staff.

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1 Murphy E. Training for care assistants. *BMJ* 1990;**301**:506. (15 September.)

Output of medical research from India

SIR,—Few of the problems mentioned by Dr Sunil K Pandya are unique to India.[1] He has not directly addressed what may be the single most important reason for the deficit of Indian medical research.

Important breakthroughs in medical research in the West have surely been a direct result of an appreciable investment of educational time in the basic science disciplines in medical schools. Most academics teaching in medical schools in the United States and Europe actively participate in biomedical research. Indeed, unlike with their counterparts in India, their very survival is based on output of quality research—the "publish or