pines, which raise seizure thresholds and reduce seizure durations; the use of minimal currents with unknown consequences to seizure pattern and duration; and the failure to monitor seizures, both to describe the electrographic durations and patterns and to assure us that an effective fit did, indeed, occur. The last factor is particularly important. In their survey of British practice, Pippard and Ellam[15] found that even sophisticated observers could misjudge seizure duration and decide that a short fit was "effective," when it was not.

The controversy about seizures and currents will continue until seizure durations and seizure patterns are monitored objectively and researchers determine the parameters of the fit that have the highest correlation with antidepressant efficacy. A change of focus is needed—from the electric current (and, in the United States, the electrode location) to the biochemical, neurohumoral, and electrographic factors in the fit which accompany and which facilitate clinical change.

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Clinical range of neonatal rotavirus gastroenteritis

SIR,—I read with interest the paper by Dr J Dearlove and others (7 May, p 1473) concerning rotavirus gastroenteritis. In the past four months rotavirus has been identified by enzyme immunoassay in nine out of the 10 babies who died from sudden infant death syndrome whom I examined and in neither of the two infants who died from other causes in the same period. In no case was there morphological evidence of definite enteritis but in four of the cases of sudden infant death syndrome, including the rotavirus negative case, there was evidence of an upper respiratory infection, with *Haemophilus influenzae* being cultured in three. This is similar to the findings of Yolken and Murphy in Baltimore.[1] No other viruses were isolated.

The significance, if any, of rotavirus in sudden infant death syndrome is not apparent, but I would be interested to know if other colleagues have had any similar experiences.

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[1] Yolken R, Murphy M. Sudden infant death syndrome associated with rotavirus infection. *J Med Virol* 1982;**10**:291-6.

SIR,—Dr J Dearlove and others (7 May, p 1473) suggest that "neonatal rotavirus infection may occasionally cause severe gastrointestinal problems." We would like to add our own similar experience of rotavirus infection in a neonatal intensive care unit, which was gained while undertaking a prospective study of non-bacterial infection in preterm babies between November 1981 and September 1982.

Rotavirus was detected by immunoassay (Rotazyme, Abbott Laboratories) and confirmed by electron microscopy in the stools of 5/170 babies. In two cases rotavirus infection was subclinical, a feature which has been reported previously,[1] but among the other cases gastrointestinal infection was associated with necrotising enterocolitis in two babies and bloody diarrhoea in the remaining case.

Although these numbers are small, we believe that they may be important in providing corroborative support to the evidence provided by Dr Dearlove and others, in that they come from a unit where rotavirus infection is not endemic, thereby preventing the suspicion that infection was coincidental.

Rotavirus infection was identified, however, in only two of the eight cases of necrotising enterocolitis studied during this period, and it is clear that a multicentre study is needed to collect sufficient cases of necrotising enterocolitis and bloody diarrhoea in newborns to evaluate properly this association.

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Scoliosis in the community

SIR,—I was concerned to read the letter by Dr Andrew G King (30 April, p 1442) about my article on scoliosis in the community (19 February, p 615), as it was precisely this confusional state about terminology, the difference between screening and early detection, and the fundamental principles of epidemiology that my article was intended to clarify.

The wise and experienced men on the terminology committee appreciated full well that in defining idiopathic scoliosis as "a structural spinal curvature for which no cause is established"[1] reconsideration would be indicated as and when epidemiological research produced more information. Accordingly, when 40% of scolioses detected in the community are attributable to an inequality in leg length it would be incorrect by any terminological standard to regard all scolioses in the community as idiopathic. The term "schooliosis," which Dr King regards as flippant, is therefore a splendid word and was indeed coined by a distinguished past president of the Scoliosis Research Society, although unfortunately he was never a member of the terminology committee.

There is regrettably little evidence concerning the validity of screening,[2] and there are no data about sensitivity and specificity as published programmes do not include information referable to false negatives. Important prerequisites for a screening programme are that it should make better use of finances than available alternatives and that there should be a satisfactory form of treatment.[3] The former is certainly not the case, and the latter is questionable.

With curve magnitude at presentation so high, nobody would dispute the need for early detection. What is being disputed is whether screening is the way to go about it or whether these funds should not be channelled in other directions. A recent review by an academic epidemiological team came to conclusions similar to my own.[4] They stated that there is an urgent need to coordinate and increase research designed to determine the aetiology, incidence, prevalence, and course of idiopathic scoliosis and that this is where resources should be directed.

Dr King therefore has a lot of homework to do, and I would suggest careful perusals of *Epidemiology for the Uninitiated*[5] and the review by Professor Warren and his colleagues, both compulsory reading for all those interested in the epidemiology of scoliosis and screening fanatics in particular. An admirable starting point, however, would be my recent article in the *BMJ* (19 February, p 615) entitled "Scoliosis in the Community."

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Underdiagnosis and undertreatment of asthma in childhood

SIR,—The recent articles by Dr A N Speight and others (16 April, p 1253) and Dr D A Lee and others (16 April, p 1256) clearly show the underdiagnosis of asthma in children; but this is a problem that applies equally to adults. In children the definition of asthma in the purely functional terms of the Ciba Foundation[1] has led to the recognition that recurrent "bronchitis" and asthma in childhood are virtually always the same thing. Paradoxically too literal interpretation of the Ciba definitions in adults has resulted in an artificial separation between asthma on the one hand and chronic airway obstruction on the other, leading to the misconceptions that obstruction in asthma is always potentially completely reversible and that there is no variability in the chronic obstruction associated with chronic bronchitis.

Indeed, it has become increasingly apparent that in some patients with asthma the obstruction may be partly reversible, particularly in the elderly. This can be seen both after an