therefore any such effect should, for the most part, have been minimised since in this situation compliance is low and the time constant of the respiratory system short.

(3) We have carried out a systematic assessment of the effects of both respiratory rate and inspiratory to expiratory ratio on the pattern of interaction between spontaneous and mechanical ventilation. Both of these parameters can be used to manipulate the infant's pattern of interaction in the vast majority of cases. We did not find the pattern of active expiration against the ventilator inevitably produced a pneumothorax.

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Neonatal auditory brainstem response cannot reliably diagnose brainstem death

Sir,

Dear and Godfrey¹ are quite correct 'to sound a note of caution regarding the interpretation of the auditory brainstem response' (or brainstem auditory evoked potential) in relation to diagnosis of brainstem death. Neurophysiological investigations are a measure of function of the nervous system and their dynamic aspects must be appreciated. Loss of later brainstem auditory evoked potential components after a cerebral insult need not necessarily imply structural damage to the whole brainstem and may occasionally be reversible.^{2,3} Similarly an isoelectric electroencephalogram soon after a period of, for example, cerebral ischaemia, may only imply temporary 'paralysis' and complete recovery of cortical function can occur. The brainstem auditory evoked potential, as with other neurophysiological tests, is a useful clinical sign especially in comatose patients. As with any one clinical sign, it should be considered in the context of the history and other clinical signs.

Serial electroencephalograms after a cerebral insult have proved invaluable in assessing the degree and reversibility of damage to the cerebral cortex. In these circumstances the brainstem auditory evoked potential findings combined with the electroencephalogram provide information about cortical and brainstem function unobtainable by clinical tests alone. It would be a pity if a valuable clinical sign such as the auditory evoked potential were considered in

isolation and if the mistake of expecting the loss of these components alone to diagnose brainstem death³ were continued. In our experience serial brainstem auditory evoked potentials together with other neurophysiological investigations do offer reliable prognostic information even in the neonatal period.

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Continuous measurement of subarachnoid pressure in the severely asphyxiated newborn

Sirs,

The study by Levene and Evans¹ contains some interesting observations. The underlying supposition is that there is benefit in reducing intracranial pressure (in a way that improves cerebral perfusion pressure) after birth asphyxia. Although this is a plausible notion, I know of no human experimental evidence to support it adequately. I acknowledge that there would be considerable difficulty in obtaining such evidence, which would require a study incorporating more badly asphyxiated babies than most of us will see in a lifetime, but it is surely premature to regard as unethical withholding the treatment which they 'tentatively recommend'. They might argue that mannitol should only be given if intracranial pressure monitoring is performed, and that a procedure as invasive as placing a subdural catheter could not be justified if the information provided was ignored; but in that case how do we proceed? I would be interested to know what experimental approaches to this important issue they think might reasonably be conducted.

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Drs Levene and Evans comment:

We thank Dr Dear for his interest in our paper. Our approach to the problem of postasphyxial cerebral oedema has been to ask three questions:

- (1) Does raised intracranial pressure occur after birth asphyxia?