under local was when she started laughing just at the most critical stage of the extraction!

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SIR,—I was interested to read Dr John Scott Price's thoughts on chronic depressive illness (6 May, p 1200).

I wonder if many psychiatrists understand, as he fortunately does, the considerable advances made in stereotactic procedures for the relief of many intractable chronic neurological disorders. Since 1963 we have had depth microelectrode recording and audio-monitoring for very precise target localisation so that the lesion can be small and exact. We now know where many of the important conduction pathways are and how they can be located. Intractable chronic neurological disease not responding adequately to known drug regimens can be relieved with great benefit to the patients. I would question whether a small precise cerebral lesion—which it is hoped and expected will be irreversible—is as damaging as some of the present drug regimens often given in the vain hope of improvement and with a significant morbidity in intractable neurological disorder. However, no matter how skilful the modern surgical neurologist is, I defy him to operate with any precision on the psyche. Psychosurgery is a thoroughly bad term and should be abandoned. Stereotactic surgery is a term used for the relief of disorders of cerebral function (so-called functional disorders)—of sensation (intractable pain), of movement and muscle tone (the dyskinesias, intractable epilepsy, and hypertonicity), and behavioural disorder.

Once more let us try to say what we mean and also define the efficacy of our various methods of treatment, drug regimens, and surgery. I welcome the new controlled trials. "Many informed people consider leucotomy in any form to be a barbaric procedure," writes Dr Price. "Leucotomy"—the old-fashioned radical section of frontal-lobe white matter—is never used now, and are these people informed of modern surgery? Perhaps informed people do not travel as much as I thought.

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Management of severe acute asthma

SIR,—In your excellent leading article on the management of severe acute asthma (8 April, p 873) you seem to have misunderstood one of the studies you quote.¹ Dr Williams and I found that patients with this condition did not respond to nebulised salbutamol and yet they did respond to subsequent administration of intravenous salbutamol on the day of admission. Over the next few days, as the steroid therapy took effect, inhaled salbutamol became increasingly effective and the incremental improvement after the intravenous drug less so. The study was designed to find whether our patients with acute severe asthma (carefully defined in the paper) responded to nebulised salbutamol (they didn't) and, if not, whether this was due to failure of the drug to reach its site of action or to loss of receptor sensitivity to the drug. These were practical questions on the answers to which treatment may be based—namely, to administer sal-

butamol by nebuliser but to anticipate that in the most severely ill patients it may not be effective and to be prepared to give intravenous drugs as well. The essential thing is for physicians to measure the response. We did not intend, as you appear to think, to compare the relative efficacies of the two methods of administration—which, I agree, would have required a different trial design.

I should point out that you mention another of our papers² that did not in fact deal with the subject to which you were referring but was concerned with the metabolic effects of intravenous salbutamol.

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¹ Williams, S J, and Seaton, A, *Thorax*, 1977, **32**, 555.

² Nogrady, S G, Hartley, J P R, and Seaton, A, *Thorax*, 1977, **32**, 559.

Schumann's hand injury

SIR,—I would like to comment on the article by Dr R A Henson and Professor H Urich (8 April, p 900). As a biographer of Schumann, I think it important to clarify certain issues which their paper obscures.

When Dr Henson and Professor Urich state: "There seems little doubt that the condition was neurologically determined," and "We can confidently assert that neurosyphilis did not cause Schumann's condition" it should be stressed that they are referring to Schumann's *hand* injury and not to his terminal illness. The difference is important. For more than 20 years Schumann presented a baffling array of symptoms, including giddiness, tinnitus, convulsions, hot and cold flushes, violent sweating, and, towards the end, massive memory failure and insanity. In 1855, a year before his death, his first biographer, Wasielewski, visited him in the asylum at Eendenich and watched him, unobserved, extemporising at the piano and described him unforgettably "like a machine whose springs are broken, but which still tries to work, jerking convulsively." After reviewing all the medical evidence and carefully considering all the previous diagnoses (ranging from a brain tumour to schizophrenia) Dr Eliot Slater and Dr Alfred Meyer came forward in 1959 with what was then an original conclusion.¹ The only disease to fit all the facts, they maintained, was tertiary syphilis. Their diagnosis was in no way weakened when, in 1973, Schumann's *Tagebücher* were published for the first time, in which the composer chronicled his encounters with the opposite sex during his student days at Leipzig and Heidelberg in some detail.

When I embarked on my two biographical studies of the composer (the first of which was used as a source by Dr Henson and Professor Urich) I based my account of Schumann's illness on Slater and Meyer. The responsibility for becoming the first musical biographer to state that Schumann died of tertiary syphilis was not one that I took lightly. Nothing in Dr Henson and Professor Urich's paper, however, changes that diagnosis. What their paper does is to place a question-mark over the *link* (first proposed by Dr Eric Sams in the *Musical Times* in December 1971) between the hand injury and the terminal illness. That link was mercury, according to Sams, with which Schumann was treated to cure his syphilis but which poisoned him and caused motor paralysis of certain fingers of the

right hand. Dr Henson and Professor Urich dismiss this idea, and they may be right to do so. But what do they resurrect in its place? They fall back on the astonishing notion that "piano playing was the source of Schumann's trouble." After the presentation of so much neurological detail we expected a lion and the authors present a mouse. Their conclusion will be unacceptable to most musicians. Playing the piano simply cannot result in a hand injury so chronic that it results in partial paralysis, lasts for 26 years, renders the hand-writing unintelligible, and makes it impossible to hold a simple object such as a baton without having it tied to the wrist. Show us a similar case in the whole of musical history.

It is also a source of dismay to read: "There were many finger strengtheners on the market at the time, and . . . Schumann ordered one in 1837." Did it ever arrive? There is not a shred of hard evidence to suggest that Schumann ever used this "mechanical aid," least of all that he damaged his hand doing so, although the 19th century biographers certainly fostered the myth. And even if he had Dr Henson and Professor Urich cannot reconcile this with the awkward fact that Schumann's hand injury first emerged seven years earlier.

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¹ Slater, E, and Meyer, A, *Confinia Psychiatrica*, 1959, **2**, 65.

Diazepam in pre-eclamptic toxæmia

SIR,—Mr G V P Chamberlain and others (11 March, p 626) point out that the use of diazepam for pre-eclamptic toxæmia is popular among British obstetricians but has never been subjected to controlled clinical trials. Dr R J Rowlatt (15 April, p 985) rightly reminds us of the dangers to the fetus, both from high doses during labour and from lower doses for longer periods during pregnancy.

While there is obviously a need for controlled trials for some of the treatments used in the management of pre-eclampsia, I would argue that a controlled trial concerning the use of low-dose long-term diazepam in mild pre-eclampsia cannot be justified. Firstly, enough is known of the harmful effects on the fetus for benzodiazepines to be contraindicated in pregnancy in this context. Secondly, there is no rationale for their use in the first place. If pre-eclampsia were recognised as a primarily psychosomatic disease there might be some justification for trying the effects of tranquillisers, but this is not an orthodox view at present. The main line of reasoning behind the use of diazepam in pre-eclampsia appears to be as follows: "Because diazepam is a useful drug in fulminating pre-eclampsia and eclampsia it might be useful in smaller doses in pre-eclampsia." This reasoning is, of course, irrational, as in the former instance diazepam is being used as an anticonvulsant, whereas in the latter it is simply being used as a tranquilliser. Another factor leading to the use of diazepam is that when a patient is admitted for bed rest the doctor feels he has to do *something*, irrespective of whether the mother is anxious or not. Perhaps it would be safer for all concerned if the doctor prescribed the diazepam for himself!

A similar phenomenon probably occurs when diazepam is being used more justifiably