0.15 mg twice or thrice daily for seven days; blood was collected every day before the morning dose of the drug and on the fourth and seventh days blood samples were also taken at hourly intervals for 3 h after the first daily dose of clonidine. No significant increase in serum GH was detected during the acute studies, though minor elevations occurred in three male subjects after intravenous clonidine (net increase ranging between 2.3 and 4.3 $\mu\text{g l}^{-1}$) and in two of them after oral administration of the drug (net increase 2.4-4.6 $\mu\text{g l}^{-1}$). During the seven days of treatment only one man and one woman patient exhibited isolated increases in serum GH concentration of minor magnitude (less than 5 $\mu\text{g l}^{-1}$).

Though we have no personal data on the effects of α -adrenoceptor agonists on GH secretion in normal subjects, Lal *et al.*¹ using clonidine, and Drs Lancranjan and Marbach, using BS 100-141, found that they caused an increase in serum GH levels in healthy male volunteers. Clonidine was also found to stimulate GH secretion in adult male rhesus monkeys² and rats.³ Though aging has been associated with reduced spontaneous and stimulated GH release,¹⁻³ Drs Lancranjan and Marbach found no increase in plasma GH levels in young hypertensive patients under chronic treatment with BS 100-141. Therefore the possibility cannot be excluded that the failure of α -adrenoceptor agonists to stimulate GH secretion in hypertensive subjects might be due to some alteration in α -adrenoceptors associated with the hypertensive state.

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¹ Lal, S., *et al.*, *Journal of Clinical Endocrinology and Metabolism*, 1975, **41**, 827.

² Chambers, J. W., and Brown, G. M., *Endocrinology*, 1976, **98**, 420.

³ Ruch, W., *et al.*, *Experientia*, 1976, **32**, 529.

⁴ Weitzman, M. D., *Annual Review of Medicine*, 1976, **27**, 225.

⁵ Bazzarre, T. L., *et al.*, in *Proceedings of the IIIrd International Symposium on Growth Hormone and Related Peptides*, Milan, 1975, ed A. Peelle and E. E. Müller, Amsterdam, Excerpta Medica, 1976.

Hospital inpatient provision for patients with dementia

SIR,—Dr David Jolley (21 May, p 1335) doubts the DHSS plan of 2.5-3.0 beds for elderly demented for every 1000 people over 65 because this estimate is based only on the number of demented patients in mental hospitals in 1971. He also wants to include those in geriatric beds and Part III accommodation, making 5 beds per 1000. But has Dr Jolley included elderly demented patients in the wards of general hospitals? There are usually two or three in my wards waiting almost indefinitely for transfer.

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Wound sepsis: effect of incidental appendicectomy

SIR,—In a series of 224 patients we found¹ that removal of a normal appendix together with the gall bladder caused a higher rate of wound sepsis than cholecystectomy alone in patients not protected by cephaloridine. We

have analysed a consecutive series of 131 patients undergoing elective truncal vagotomy and pyloroplasty for peptic ulcer. All other gastric operations were excluded. Sixty-one patients had vagotomy and pyloroplasty and 70 incidental appendicectomy as well. The primary wound sepsis rates were 3.34 (8.8%) and 2.30 (6.7%) respectively in patients protected by cephaloridine and 6.27 (22.2%) and 10.40 (25.0%) in the others. There are no significant differences between the rates with and without appendicectomy.

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¹ Pollock, A. V., and Evans, M., *British Medical Journal*, 1977, **1**, 20.

Use of ritodrine in pregnant diabetics

SIR,—Following several reports of the successful use of ritodrine in pregnant women the data sheet for ritodrine has been altered as Dr C. J. Chandler suggests (30 April, p 1159). It will now read as follows:

Precautions

(Paragraph 2) Although glucose and insulin profiles in normal pregnant women have been shown in controlled clinical trials not to alter under Yutopar therapy, it is recommended that in the diabetic patient monitoring of glucose levels be undertaken.

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Lithium carbonate in schizoaffective states

SIR,—I was interested to read about Dr W. W. Sargent's use of lithium carbonate in schizoaffective states (4 June, p 1470). At this unit there are two outpatients currently attending who suffer from recurring bouts of schizoaffective illness and for whom the use of lithium carbonate has been found to be of great help.

Case 1—An 18-year-old male patient who had his first attack at the age of 13. The clinical picture then was of very florid and bizarre delusions, with auditory hallucinations, thought disorder, and accompanied by great psychomotor activity. A diagnosis of schizophrenia was made and phenothiazine medication instituted. A further similar florid psychotic breakdown occurred two years later. Last year while attending this unit and while still on a high level of long-acting phenothiazine medication he developed a third attack. It was very interesting to observe how the attack developed. He became more active, more restless, showed pressure of speech, and for the next 14 days he showed gross insomnia. All of these features were quite resistant to even high doses of phenothiazines. At the end of this period he showed a typical acute schizophrenic picture with gross thought disorder, delusions, and hallucinations. However, when put on lithium carbonate he rapidly stabilised, and over the past eight months continuation of lithium carbonate along with a small dose of phenothiazines has stabilised him so well that he is now in open employment for the first time in his life.

Case 2—A 28-year-old female patient who had five recurring schizoaffective attacks for 11 years resulting in 18 months of hospitalisation over this period of time. She too had been on a high level of long-acting phenothiazine medication, which on

its own had failed utterly to prevent relapses, which, when they occurred, always presented clinically with excitement, ideas of reference, thought disorder, delusions, and hallucinations. Her last attack was ushered in by an acceleration of psychomotor activity accompanied by a severe insomnia state. After about 10 days she then began to express delusions of a religious and paranoid nature, showed ideas of reference, and had auditory hallucinations. In this state she too was quite unresponsive to phenothiazines. However, response to lithium carbonate was both rapid and sustained, and her progress in the nine months since then has been surprisingly good. She is rapidly discarding her "chronic patient" image and, as her parents report, "is returning to her old self."

The above cases are interesting for two reasons. One is the diagnostic one: schizoaffective illness is a definite clinical entity containing as it does features common to both schizophrenic and affective states. However, it is important to make the diagnosis accurately as otherwise the patient will usually be classified as schizophrenic and will therefore receive inadequate medication. In this regard there is a lot to be said for Ollerenshaw's¹ recommendation that acute schizophrenia should be regarded as simply a variant of the manic phase of manic-depressive illness. Secondly, and more intriguingly, what exactly is the role of sleep deprivation in these states? Could, for instance, severe sleep loss, as in the above two cases, be the missing ingredient which converts a manic state into an acute schizophrenic one? The role of sleep deprivation in producing psychiatric symptoms is not fully clear,² but the kind of sleep loss in a manic state must surely be very considerable and perhaps nearer to being a total sleep loss state than that produced experimentally.

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¹ Ollerenshaw, D. P., *British Journal of Psychiatry*, 1973, **122**, 517.

² Oswald, I., *Sleep*, 3rd edn. London, Pelican, 1974.

Microsomal enzymes in malnutrition

SIR,—We apologise for commenting so late on the interesting article by Dr K. Krishnaswamy and Dr A. N. Naidu entitled "Microsomal enzymes in malnutrition as determined by plasma half life of antipyrine," (26 February, p 538), but our personal half life for absorption of the *BMJ* is currently prolonged. We believe that this article, quoted in your recent leading article (21 May, p 1304), is somewhat misleading.

In the summary we read that "the half life of antipyrine was modified in undernourished subjects and those with nutritional oedema." In the article, however, it is clearly shown that there is no difference in antipyrine half life between the group of undernourished subjects who were smokers (and exposed to pesticides) and the appropriate control group of smokers. It is not possible to judge whether the half life of antipyrine was modified in those with nutritional oedema as we appear not to be told if this group (group 4) were smokers; the mean half life for group 4 was apparently longer, although no P value was given, than that for the smoking controls (group 2) but no different from that for the non-smoking controls (group 1).

Even if the half life of antipyrine were prolonged in patients with nutritional oedema when compared with the appropriate control subjects, this could at least partly result from a