swelling or expressed a preference for this posture. I do, however, maintain the lower limbs in the horizontal position.

A patient can just as readily faint in the horizontal as in the sitting position. Some time ago I had a young, healthy female patient who, while in the supine position, fainted following the insertion of the intravenous needle before any methohexitone had been injected. She became unconscious, with marked pallor, a moist skin, and bradycardia. She did not regain consciousness until she was tilted into the Trendelenburg position and pure oxygen was administered. She was subsequently anaesthetized uneventfully. The important thing is to recognize a faint immediately, whatever position the patient is in, and to treat it expeditiously.—I am, etc.,

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1 S.A.A.D. Digest, 1974, 2, 103.

Mental Changes in Parkinsonism

SIR,—Your leading article (6 April, p. 1) provides an excellent summary of the frequent occurrence of dementia in Parkinsonism. It is also rightly sceptical of attributing this to cerebrovascular disease.

In describing both the common reversible and the rare permanent mental sequelae of treatment with levodopa you cite the papers of Andén *et al.*¹ and the recent report of Wolf and Davis.² It is of great interest that both papers describe cerebral atrophy demonstrated by air encephalography and confirmed at necropsy.² Evidence of diffuse cerebral atrophy in Parkinsonism has previously been adduced by Svennilson *et al.*,³ and was a not infrequent finding at air ventriculography performed prior to stereotactic surgery in the 1960s. Organic mental changes were more frequent in such patients, and evidence of primitive reflexes, especially the glabella tap,^{4,5} were common. We find therefore, a tetrad consisting of: (1) Parkinsonism, (2) dementia of varying degree, (3) primitive reflexes, and (4) cerebral atrophy. This forms a distinctive clinical syndrome, but one which is seen in the advancing or late cases of Parkinson's disease. It is not exclusive to this disease and can be found in the different clinical context of Alzheimer's presenile dementia.⁶

In a clinical study of 65 patients with dementia (mean age 61 yr; 42 male, 23 female; symptom duration 0.2 yr in 80% of patients), we found "Parkinsonian signs" in no less than 40 cases (61%) (table I). One

TABLE I—Parkinsonian Signs Present in 40 Patients Presenting with Presenile Dementia

Sign	No. of Patients
Facial Masking	30
Akinesia	18
Rigidity	22
Tremor	19
Glabella tap reflex	32

or more primitive reflexes were elicited in 32 (80%) of them (table II). Cortical atrophy or ventricular dilatation was seen at air encephalography in 92% patients, as would be expected in Alzheimer's disease.

I would emphasize that the clinical picture in those presenting with presenile dementia

TABLE II—Primitive Reflexes Present in 40 Patients with Presenile Dementia and Parkinsonian Signs

Reflex	No. of Patients
Glabella tap reflex	32
Snout/sucking reflex	26
Grasp reflex	16
Palmo-mental reflex	21

is quite distinct from Parkinson's disease in the early stages. As the two disorders progress, however, the overlap of their clinical features can on occasion be striking, though the severe tremor and flexion dystonia of axial muscles seen in Parkinsonism is not seen in those presenting with dementia. A further feature we have noticed is the singular failure of levodopa to influence the extrapyramidal disorder in either clinical context, and indeed profound depression, worsening of the dementia, and hallucinosis are liable to occur at low dose levels (0.5 to 1.5 g/24 hr).

These findings suggest the possibility that the cerebral atrophy may be a part of the primary disorder in both Alzheimer's and Parkinson's diseases; and that in the late stages there may therefore exist a *supranuclear* disorder of basal ganglia function.⁷ This may be important in modulating neurotransmission in both dopaminergic and neuromelanin granules at a lower level, as a result of the neuronal depletion and atrophy of the cortex. The lack of therapeutic response to levodopa and the "hypersensitivity" to this drug in those patients with the tetrad of features described are explicable on this basis.—I am, etc.,

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- 1 Andén, N. -E., *et al.*, *Acta Medica Scandinavica*, 1970, 187, 247.
- 2 Wolf, S. M., and Davis, R. L., *Archives of Neurology*, 1973, 29, 276.
- 3 Svennilson, E., *et al.*, *Acta Psychiatrica Scandinavica*, 1960, 35, 358.
- 4 Pearce, J., Aziz, H., and Gallagher, J. C., *Journal of Neurology, Neurosurgery and Psychiatry*, 1968, 31, 501.
- 5 Pearce, J., and Pearce, I., *Postgraduate Medical Journal*, 1971, 47, 794.
- 6 Pearce, J., and Miller, E., *Clinical Aspects of Dementia*, London, Baillière Tindall, 1973.
- 7 Pearce, J., *European Neurology*. In press.

Hangover and Testosterone

SIR,—Chronic alcohol intake is known to alter the secondary sex characteristics in men. These changes have been attributed to altered steroid metabolism in a cirrhotic liver. However, ethanol itself, without any liver damage, has been found to affect the metabolism of steroid sulphates.¹ As an extension of our earlier hangover study² we studied the effects and after-effects of ethanol on the plasma concentration of free testosterone, which, in contrast to the steroid sulphates, is a biologically active hormone.

Mean (± S.E.M.) Plasma Testosterone and LH Concentrations during Alcohol Intoxication and Hangover in 10 Subjects

Time (hr)	Plasma Testosterone (nmol/l.)		Plasma LH (IU/l.)	
	Without Ethanol	With Ethanol	Without Ethanol	With Ethanol
0	20.1 ± 2.4	18.2 ± 1.9	8.2 ± 1.6	12.1 ± 2.5
4	15.2 ± 2.1	15.9 ± 1.4	8.8 ± 2.2	9.4 ± 1.7
8	17.7 ± 2.1	17.2 ± 2.4	6.4 ± 1.3	6.8 ± 1.8
12	23.0 ± 2.0	16.3 ± 2.8	6.3 ± 1.8	7.8 ± 2.3
15	20.2 ± 1.8	10.5 ± 2.5	6.6 ± 1.6	10.2 ± 2.3
20	19.3 ± 1.6	8.3 ± 2.1	6.0 ± 1.2	12.2 ± 2.2

Ten healthy male students aged 19 to 25 volunteered for the experiment. Each received ethanol 1.5 g/kg as a 20% aqueous solution which he drank at a constant rate during a period of three hours. The experiments were started at 6 p.m. By the next morning all the ethanol had been eliminated and most of the subjects had a more or less severe hangover. Each also participated in another experimental session during which he drank water only. Thus each subject served as his own control. Blood samples for determining levels of luteinizing hormone (LH) and testosterone were taken at the start of drinking and at 4, 8, 12, 15, and 20 hours afterwards. Concentrations were estimated by radioimmunoassay.

During the period of acute intoxication there were no changes in plasma testosterone concentration, but during the hangover (12-20 hours after the start of drinking) the testosterone level was only about half what it was during the control period (table). In addition, the decrease seemed to correlate with the intensity of the hangover. The decrease was not due to inhibited LH secretion because plasma LH concentration was not decreased by ethanol. On the contrary, there was a slight increase in LH level during the hangover.

Though the biochemical mechanism and physiological significance of the decrease in plasma testosterone concentration during the post-alcoholic phase are not yet known we want to report this finding, because it indicates that ethanol can alter the metabolism of biologically active sex hormones without causing any liver damage. If the testosterone level is constantly low during chronic alcohol intake this may explain some of the endocrine abnormalities found in chronic alcoholics.—We are, etc.,

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- 1 Cronholm, T., and Sjövall, J., *European Journal of Biochemistry*, 1970, 13, 124.
- 2 Ylikahri, R. H., *et al.*, *European Journal of Clinical Investigation*. In press.

Pseudotumours of the Orbit

SIR,—I was interested to read the leading article (6 April, p. 5) on pseudotumours of the orbit. One cause of pseudotumour in the elderly, temporal or giant-cell arteritis, was unmentioned. I reviewed 43 cases of temporal arteritis and found three that had presented with proptosis and chemosis (in one case bilateral) sufficient to warrant the term pseudotumour of the orbit. Diplopia occurred in two of the cases and one of these developed unilateral blindness. In all three cases there was marked disablement and severe orbital pain.

If pseudotumour of the orbit presents in a patient over the age of 60-65 years temporal arteritis should be considered and other manifestations, especially girdle myalgia or arthralgia, sought. A raised E.S.R. may be

found in many cases of pseudotumour whatever the cause and consequently is of little help,^{1,2} but adequate temporal artery biopsy may help in diagnosis and should also be performed. Though the condition may remit spontaneously, in temporal arteritis delay in diagnosis may allow further irreversible arteritic complications to occur. Corticosteroids in adequate dosage produce dramatic symptomatic benefit and objective improvement follows within a week, so that it may be clinically prudent to treat elderly patients with a trial of corticosteroids rather than resort to prolonged investigation or transfer to a specialized centre for further procedures.

I tentatively suggest that many of the idiopathic granulomatous pseudotumours of the orbit in elderly patients responding to corticosteroids are in reality caused either by temporal arteritis or Wegener's granulomatosis, possibly in the "limited" form described by Carrington and Liebow.³ After a successful trial of corticosteroids the patient should receive prolonged follow-up to prevent recurrence of the parent disease when corticosteroids are reduced or discontinued.—I am, etc.,

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- 1 Jackson, H., *British Journal of Ophthalmology*, 1958, 42, 212.
- 2 Coop, M. E., *British Journal of Ophthalmology*, 1961, 45, 513.
- 3 Carrington, C. B., and Liebow, A. A., *American Journal of Medicine*, 1966, 41, 497.

Hypothermia, Thrombosis, and Acute Pancreatitis

SIR,—The report by Drs. E. P. Savides and B. I. Hoffbrand (30 March, p. 614) of a fatal case of hypothermia, in which acute pancreatitis was found in association with thrombosis in the upper superior pancreaticoduodenal vein and its intrapancreatic branches, merits comment.

First, though visceral thrombosis and infarction seem to be complications of hypothermia rather than of pancreatitis it is unlikely that the acute pancreatitis so commonly found in patients with accidental hypothermia can be explained on this basis. Pancreatic venous thrombosis in association with acute pancreatitis has not been mentioned in any of the major series of cases of accidental hypothermia so far published.^{1,2} It remains conceivable, however, that even in the absence of such thrombosis ischaemia may still contribute to the pancreatitis.³

Second, the presenting clinical features of generalized and rebound tenderness of the abdomen are not characteristic of the acute pancreatitis in those with accidental hypothermia.^{1,2} In the last few years I have extensively studied more than 100 patients with accidental hypothermia and I have found the signs of an acute abdomen only in those patients in whom one of two associated conditions was present: (1) perforation of an acute gastric or duodenal ulcer with secondary peritonitis; (2) mesenteric vascular occlusion with gangrene of part of the small bowel.

None of my patients in whom pancreatitis was confirmed at necropsy had shown signs of an acute abdomen. It follows therefore that in the case described by Drs. Savides and Hoffbrand an alternative explanation for

their findings is that the hypothermia may simply have been an epiphenomenon in a patient originally suffering from acute pancreatitis, with the venous thrombosis being a later complication of the hypothermia. Like others,⁴ I have found that in the elderly patient with accidental hypothermia the hypothermia is usually a secondary phenomenon in someone suffering from a primary serious underlying illness, the latter often dominating the clinical picture and determining the main need for any therapy. None the less, now that Drs. Savides and Hoffbrand have drawn attention to their finding it will be interesting to see how often it is found by others.—I am, etc.

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- 1 Duguid, H., Simpson, R. G., and Stowers, J. M., *Lancet*, 1961, 2, 1213.
- 2 Mant, A. K., *British Journal of Hospital Medicine*, 1969, 2, 1095.
- 3 Maclean, D., Murison, J., and Griffiths, P. D., *British Medical Journal*, 1973, 4, 757.
- 4 Weyman, A. E., Greenbaum, D. M., and Grace, W. J., *American Journal of Medicine*, 1974, 56, 13.

SIR,—We were interested to read the account by Drs. E. P. Savides and B. I. Hoffbrand of a case of acute pancreatitis associated with hypothermia (30 March, p. 614). It appeared from their review of the literature that acute pancreatitis, as well as other common complications of hypothermia, may have been precipitated by circulatory stasis. The incidence of thrombosis in various vessels has been found to be very high at necropsies following hypothermic deaths.

A possible explanation of this finding may be the increase in blood viscosity with a fall in temperature. The accompanying figure shows the change in viscosity of the

same sample of whole blood at different temperatures ranging from 25°C to 40°C. The viscosities were determined at a number of shear rates, from 2.62 sec⁻¹ to 230 sec⁻¹. The result illustrates the significant effect of temperature on viscosity.

A patient with a rectal temperature of 30.5°C such as the one described by Drs. Savides and Hoffbrand would have had a blood viscosity at most shear rates 25–40% higher than that at normal body tempera-

ture. This would have resulted in a decrease in blood flow sufficient to increase significantly the chances of thrombosis.—We are, etc.,

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Adverse Reactions to Intra-amniotic Urea and Prostaglandin

SIR,—The adverse reaction at the time of an intra-amniotic injection of urea and prostaglandins reported by Mr. A. H. Ross and Mr. W. L. Whitehouse (30 March, p. 642) is not a feature that we have noticed in 200 patients treated by this method, using a fixed dose of 80 g of urea and variable doses of prostaglandins E₂ and F_{2α}.

The symptoms described strongly suggest that a relatively large dose of prostaglandin had rapidly reached the systemic circulation. Such an occurrence is possible if the needle or cannula used becomes misplaced outside the gestation sac, and because of this we advise that a cannula with side drainage holes be used and that care be taken to ensure that there is free flow-back of liquor into the syringe throughout the injection procedure. If not, we refrain from injecting the agents. We also prefer to wait until after 16 weeks because the method is technically easier, there being less likelihood of the amniotic sac being drained following aspiration of the advised 100 ml of liquor and of resultant trauma to the placenta, even with the routine use of ultrasound localization. We feel that the aspiration of some liquor is required since the urea solution administered (80 g of urea added to 80 ml of Hartmann's solution makes approximately 140 ml) may rarely lead to over-distension of the sac with loss of the abortifacient agents and subsequent uterine activity. Our main concern about possible complications occurring with this technique is that of excessive uterine activity resulting in cervical injuries as recently described (4 May, p. 271), and has led to the evaluation of using low doses of prostaglandin—for example, 2.5 mg of prostaglandin E₂.—We are, etc.,

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Ideal Consultation

SIR,—In the course of his generous review of *Six Minutes for the Patient* (6 April, p. 65), Dr. Andrew Smith makes a statement which, unqualified, might appear to perpetuate the mind-body dichotomy, which I am sure was not his intention. He writes, "the patient with tonsillitis needs penicillin, not insight." Without wanting to split semantic hairs might I suggest that it is the tonsillitis which needs penicillin, and that the patient-as-a-person may stand in need of something more? The trouble is that we simply do not know how to assess this need accurately, our present methods being relatively primitive. As Dr. Smith says, the