While a survey of reports on this topic showed no mention of peripheral nerve paresis during TPN parenteral nutrition, I found two cases of Wernicke encephalopathy after prolonged intravenous therapy that were probably due to thiamine deficiency.² Since the patients mentioned by Dr Ladefoged and Dr Jarum received only 9.7 mg thiamine once or twice a week, I think that a thiamine deficiency could probably explain the paresis of the peroneal nerves and perhaps of the radial nerves too.

These facts should alert the clinician to suspect a "parenteral-nutrition-related beri beri" if polyneuropathy without any evident cause appears in patients fed intravenously (the determination of transketolase levels might prove a reliable laboratory test in this case). They suggest that about 0.3 mg thiamine/kg/day should be given to patients receiving parenteral nutrition as has been recently advocated.³

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¹ Ziporin, Z. Z., *et al*, *Journal of Nutrition*, 1965, **85**, 297.
² Nadel, A. M., and Burger, P. C., *Journal of the American Medical Association*, 1976, **235**, 2403.

³ Wretling, A., *Surgical Clinics of North America*, 1978, **58**, 1055.

How to take a clinical examination

SIR,—I started to read the article by Dr J F Stokes (13 January, p 98) with interest because he has been concerned with the clinical examination of the MRCP for a number of years, but as I read my disappointment and later annoyance grew. Regarding the Adie pupil, he suggests that the examiner will have arranged for the pupil to be dilated if the daylight in the ward is bright and suggests that the candidate should ask the patient if he has had drops in his eye, which would presumably have been homatropine. He does not seem to appreciate that the drops should be 2.5% mecholyl, which constricts the Adie pupil but not the normal pupil.¹ If homatropine had been put into the eye to dilate the pupil, it is doubtful if it would be possible to recognise the Adie pupil anyhow. Nowhere in the article does he suggest that the urine should be tested or a sample asked for or that the blood pressure should be measured.

Towards the end of the article, Dr Stokes remarks that it is better not to appear obviously pregnant, which must be offensive to female candidates. Many of the most distinguished doctors in Britain today are women and it is irrelevant if they took the MRCP when pregnant.

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¹ Beeson, P. B., and McDermott, W., *Textbook of Medicine*, p 328. Philadelphia, Saunders, 1971.

* * * We sent a copy of this letter to Dr Stokes, whose reply is printed below.—Ed, *BMJ*.

SIR,—The lines are crossed. I had tried to put myself in the position of a candidate who found a patient with unequal pupils and was uncertain whether one had been dilated to make it easier for him to see the fundus or whether he was faced with a case of Adie's syndrome. The question of whether I am able or unable to distinguish between the uses of homatropine and mecholyl does not arise.

I do not undervalue the importance of the blood pressure or of urine testing. In an article of limited length it seemed best to concentrate on points which might be less generally recognised.

If susceptibilities have been offended by a reference, in what I had hoped might be helpful terms, to the variable reactions I have observed in examiners to a physiological state (which may be met at graduating as well as postgraduate examinations), it is a matter for regret—though I do find it difficult to see how it can be interpreted as being denigratory to our admirable company of medical women.

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Early growth retardation in diabetic pregnancy

SIR,—The observation of Drs Jan Fog Pedersen and Lars Mølsted-Pedersen (6 January, p 18) that in a group of insulin-dependent diabetics 10 of 35 patients whose fetuses were "small for dates," by ultrasound measurement of the crown-rump length between 7 and 14 weeks postmenstrual age, subsequently delivered smaller babies than patients in whom the fetal crown-rump length measurements were appropriate for dates is interesting. However, fuller clinical detail is required before their hypothesis that there is a different pattern of growth retardation in diabetic pregnancies can be accepted.

In considering such a small number of cases the mean birth weight can readily be biased by an uneven distribution of the fetal sex, birth rank, maternal size, cigarette use, and the presence of complications such as pre-eclampsia. For example, the average effect of the first three factors at term would be as follows: male *v* female, +150 g; first pregnancy *v* subsequent pregnancies, -150 g; 175 cm/70 kg mothers *v* 150 cm/45 kg mothers, +600 g.¹ Also babies with congenital abnormalities should be excluded in view of their common association with intrauterine growth retardation. If after these factors are allowed for a significant difference in the mean birth weights remains, the hypothesis that the fetal age is less than the postmenstrual age in the "small" group cannot simply be dismissed as unlikely because the menstrual history was certain. There is considerable evidence to suggest that ovulation is not nearly as constant as implied in Naegle's rule. Boyce *et al*² studied 317 pregnancies with reliable basal body temperature charts and found that in 30% conception occurred more than two weeks beyond the usual day of ovulation calculated by Naegle's rule. The gestational age and birth-weight limits used in compiling the control series would be expected to exclude such cases and could account for the different distributions of crown-rump length measurements in the control and diabetic series.

Ultrasonic fetal measurement in early pregnancy has repeatedly been shown to predict the onset of spontaneous labour resulting in the delivery of a mature fetus more precisely than an impeccable menstrual history.³ Experience shows that when a fetal measurement falls below the normal range for the gestation age because of growth retardation the growth rate usually remains below normal. But a small measurement associated with a normal growth rate (as in Pederson and Mølsted-Pederson's cases)

indicates incorrect assignment of the gestational age.³

If our hypothesis is correct that the fetal age of the "small" group was on average 12 days less than that of the "normal-size" group, then a simple calculation from normal birth-weight data¹ suggests that this could account for a 300-500 g weight difference.

We are particularly concerned that obstetricians may be persuaded to disregard the possibility of an error in the expected date of delivery predicted by the menstrual history and deliver such fetuses earlier than would be their normal practice in diabetic pregnancy.

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¹ Thomson, A. M., *et al*, *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1968, **75**, 903.

² Boyce, A., *et al*, *American Journal of Obstetrics and Gynecology*, 1976, **125**, 911.

³ Campbell, S., in *Fetal Physiology and Medicine*, ed R W Beard and P Nathanielsz. London, W B Saunders Co. Ltd, 1976.

Ergotamine tartrate overdose

SIR,—It is interesting to note that the difficulties of giving an effective treatment with ergotamine tartrate in migraine are given publicity. I refer to a letter from some well-known physicians with an interest in migraine whose signatures are headed by Dr J N Blau (27 January, p 265).

Most surprisingly, hypnosis and autohypnosis are seldom mentioned in connection with this distressing complaint—it is without doubt the treatment of choice when simple analgesics fail. Much of the poor reputation of hypnosis of past years has been due to poor patient selection, setting of unrealistic goals, and uncertainty of application; but none of these apply where the treatment of migraine is concerned. Impressive results have been reported by Harding,¹ Cedercrutz,² and Perkins.³

I was involved in a multicentre trial to compare hypnosis and autohypnosis with prophylactic prochlorperazine mesylate (Stemetil) and therapeutic ergotamine tartrate.⁴ Independent assessment was applied by a "blind" observer so far as possible after a 12-month trial period. It was found that those having hypnosis or autohypnosis had significantly fewer attacks in the first six months of the trial than the six months preceding the trial, and in the last six months than the first six months. The chemotherapy group showed no significant change for either the period prior to the trial or the two six-month trial periods.

There were 13 patients having grade IV attacks prior to the trial in the hypnosis group. This was reduced to five in the last three months of the trial. Ten patients were having grade IV attacks in the chemotherapy group, which went up to 14 after chemotherapy. Finally, out of 23 patients having hypnotherapy, 10 patients had complete remission with no attacks in the last three months, while only three of the 24 chemotherapy patients showed similar remission.

While it is perfectly true that there is a tremendous variability in hypnotic susceptibility, the majority of patients can be

hypnotised and trained in autohypnosis to avert an attack. This can be perfected so that it is effective in a fraction of the time for chemotherapy, without any side effects, without any need for "sleeping away" the illness, and without the incapacity.

Hypnosis is more time consuming than writing a prescription, but far less so than most physicians realise; and it has the added merit of permanent remission in appreciable numbers of sufferers.

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¹ Harding, C H, in *Proceedings of the International Congress of Hypnosis and Psychosomatic Medicine, Paris 1956*, ed J Lassner. Berlin, Springer-Verlag, 1967.

² Cedercreutz, C, in *Hypnosis at its Bicentenary. Papers from the Seventh International Congress of Hypnosis and Psychosomatic Medicine*, ed F Frankel and H Zamansky. New York, Plenum, 1978.

³ Perkins, J, *British Journal of Clinical Hypnosis*, 1975, 5, 2.

⁴ Anderson, J A, Basker, M S, and Dalton, R, *International Journal of Clinical and Experimental Hypnosis*, 1975, 5, 2.

Intermittent ophthalmoplegia

SIR,—We note the constructive comments of Dr J F Falconer Smith (13 January, p 124) and we welcome the chance to provide further details of our patient (2 December, p 1534).

In a short report we were indeed unable to give all the data supporting the statement that our patient was clinically euthyroid; before treatment she was examined many times by us, and once by the members of the section of endocrinology of the Royal Society of Medicine.

We had already considered using L-thyroxine and in June 1978 treatment was commenced with 0.2 mg/day; we have observations for six months on this dose. Serum thyroid-stimulating hormone (TSH) became 1.3 mU/l (normal <1.4 mU/l) and serum thyroxine and T₃ rose slightly, towards the upper limit of our normal range. The patient remained clinically euthyroid, but there was weight loss of 4 kg to 67 kg. While she was taking thyroxine, there were two mild attacks of ophthalmoplegia, in August and October 1978 (when she was on prednisone 10 mg/day), and a severe attack in December 1978 (when she was on prednisone 7.5 mg/day).

Thus, despite treatment with thyroxine, intermittent attacks of ophthalmoplegia continue to occur, and appear to be more severe when the dose of prednisone is reduced. The frequency of attacks appears to have diminished. However, a further period of observation is necessary to be sure of this, and to find the optimum dosage of medicines.

After we wrote the original report, the serum level of TSH antibody was kindly measured by Professor R Hall and Dr B Smith, and the result was normal. Other assays were kindly done by Dr K Mashiter.

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ABC of Ophthalmology

SIR,—Mr P A Gardiner in "ABC of Ophthalmology" is to be congratulated on the very helpful current articles on eye conditions which interest general practitioners. His work is just

what many GPs want, and deserves much praise.

I would like to point out, however, that in his reference to the physiology of accommodation (2 December, p 1555) he has been misleading in saying that "the lens can be squeezed by its controlling muscle. . . . This is known as accommodation." This is the opposite of what happens, as the lens becomes more spherical when the suspensory ligament is relaxed by contraction of the ciliary muscle: no squeezing at all is involved. Mr Gardiner also makes a reference to a connection between refractive errors and colour vision, which is largely misleading.

Your editorial staff is not, in my opinion, to be congratulated on its publishing a contentious letter on myopia (20 January, p 198). Its most unsuitable title is "Myopia or juvenile expansile glaucoma" and it does little to help readers of the *BMJ*. Its author, Mr T S-B Kelly is an experienced consultant in ophthalmology, but his views are expressed as if they are facts, rather than theories, and are supposed to be substantiated by his sweeping statements and curious explanations, which appear to me to be largely fallacious and would be rejected by the great majority of ophthalmologists everywhere. It is strange to suggest that too much accommodation is the cause of myopia, a condition in which there is reduced accommodation; in hypermetropia there is increased accommodation but it seldom progresses into myopia. "Anterior chamber anomalies and other causes not yet found" in relation to nearly 10% of myopes is a reference, clearly, to figments of Mr Kelly's imagination. It is probable that bifocal spectacles (with or without special contact lenses) are as valuable in preventing pathological myopia as bee stings are in arresting so-called retinitis pigmentosa, in spite of Mr Kelly's overbearing enthusiasm for his recommended regimen. Is it not rather pathetic to hear that Mr Kelly admires the implantation of electrodes or transducers into the vitreous cavity to seek confirmation of his theory? (Would he submit his own eyes to such procedures?)

I and my colleagues will continue to serve our myopic patients with orthodox spectacles as in the past without feeling guilty of neglecting their best interests.

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Bleeding massive gastric ulcer on diflunisal (Dolobid)

SIR,—Dolobid is hailed as a major advance in analgesic therapy having, unlike aspirin, no significant effect on gastrointestinal blood loss. A perforated duodenal ulcer was recently reported by Drs R W Talbot and Helen Rees (28 October, p 1229) in a patient on diflunisal (Dolobid) and they ask, "Will it continue to be as trouble-free as predicted?" The following is a case report of a patient with major bleeding from a gastric ulcer while taking diflunisal for only 10 days.

This very active 78-year-old woman had no previous history suggestive of peptic ulceration. She regularly took slow oxprenolol 160 mg and digoxin 0.0625 mg daily and she took sorbitrate occasionally. She was afflicted by herpes zoster in the distribution of left D9. For the severe pain she took diflunisal. After 10 days' treatment with this drug she lost her appetite and vomited. The next day she had melaena. Three days later (during which time she continued to take diflunisal)

she vomited a small amount of blood. She was admitted to hospital, where the haemoglobin was found to be 6.6 g/dl. At gastroscopy the next day a very extensive superficial ulcer was seen extending from the cardia to the angulus on the lesser curve. There were patches of clot in the ulcer indicating recent bleeding. There was a small ulcer 1 cm away. The duodenum was normal. Biopsies of the ulcer margin showed no evidence of malignancy. She was transfused with 4 units of blood and given cimetidine 200 mg three times a day and 400 mg at night. The diflunisal was stopped, and the other drugs were continued. Her gastrointestinal symptoms disappeared and at gastroscopy six weeks later there was only a linear scar along the lesser curve where the ulcer had been. There was no ulceration.

The temporal relationship between administration of diflunisal and the bleeding, the absence of prior gastrointestinal symptoms, and the healing of the ulcer when diflunisal was stopped (albeit she was also given cimetidine) suggests that the diflunisal caused the gastric ulcer and the bleeding.

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Miniature peak-flow meters

SIR,—I was most interested to see Dr B J Freedman's letter (27 January, p 268) about the Pneumometer Minimus. Owing to editorial condensation it was not made quite clear in my paper (9 December, p 1627) that this instrument was a precursor of the Peak-Flow Meter. It uses a fixed orifice and a pressure gauge as originally described by Hadorn.¹ For various reasons, fully discussed by McKerrow and myself,² we did not regard this as a satisfactory method.

Although the Pneumometer is much the same size as the mini-meter it is nearly four times as heavy and its price is similar to that of the standard model. In addition, the standard meter's correlation coefficient of 0.902, compared with 0.990 for the mini-meter, means that it is only about one-third as precise. The instrument is still available and is, I believe, quite widely used on the Continent but has little sale in this country.

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¹ Hadorn, W, *Schweizer medizinische Wochenschrift*, 1942, 23, 946.

² Wright, B M, and McKerrow, C B, *British Medical Journal*, 1959, 2, 1041.

Seat-belt legislation

SIR,—In the Parliament section of News and Notes (27 January, p 276) it is noted that the chairman of the BMA Council has written to all members of Parliament telling them of the BMA's policy in regard to seat belts. The 1976 Private Member's Road Traffic (Seat Belts) Bill had the support of the Government and after successful passage through the Commons was defeated in the House of Lords on 26 April 1977 by 55 votes to 53—a majority of 2.

Careful analysis of the Lords' debate, as fully reported in *Hansard*, showed that most of the opponents were prepared to concede that compulsion would save lives and reduce serious injuries; but they argued that (a) some people would be injured as a result of wearing seat belts who would have escaped if they had not been wearing them; (b) it would impose a