SIR,—Your leading article on this topic (20 December, p. 704) was very interesting, but I feel that your figure of 24 cases of such reactions represents only a small proportion of the whole.

I have seen two patients during the past year, both middle-aged females with urinary-tract infections, who presented with acute dyspnoea and pyrexia shortly after starting a course of nitrofurantoin. The first had successfully completed a course of 100 mg. four times daily for seven days nine months previously, but to the second the drug was entirely new.

Both cases were reported to the Committee on Safety of Drugs, which source I was surprised to find omitted from your references.—I am, etc.,

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Septic Shock

SIR,—Your leading article on septic shock (3 January, p. 3) suggests that an antibiotic active against most Gram-negative bacilli should be used to initiate treatment before the causative organism is known. We disagree with this, and with your choice of kanamycin. The organisms isolated from patients with septic shock are usually, but not always, Gram-negative. We have seen streptococci and clostridia, both of which are resistant to kanamycin, as well as staphylococci, which are usually sensitive. Furthermore, *Pseudomonas aeruginosa* and *Bacteroides* are always resistant to kanamycin and some strains of *Klebsiella* may be.

We have used a mixture of gentamycin and ampicillin or cephaloridine with considerable success in treating these patients. This combination is effective against most kanamycin resistant bacteria, of which possibly the most important is *Ps. aeruginosa*. In the past two months we have seen two patients with pseudomonas septicaemia and shock.

The total dose of gentamycin given is usually small because, as with kanamycin, the antibiotics can be changed to give more specific treatment when the infecting organism is recovered. We recommend an initial dose of 80-120 mg. of gentamycin and regulate subsequent dosage according to the renal function. If this is impaired estimation of the serum gentamycin level will be necessary if treatment with this antibiotic is to be continued. Gentamycin can be given either by intramuscular injection or intravenous infusion over a period of 5-10 minutes. Ampicillin is given either by continuous or by intermittent intravenous infusion to a total of 8-12 g. per day. If the patient is known to be hypersensitive to penicillin, cephaloridine is substituted in a total dose of 2-4 g. per day, either by intramuscular injection or by intermittent intravenous infusion. Apart from an occasional hypersensitivity reaction, presumably to ampicillin, no toxic effects have occurred with these antibiotics. In particular we have seen no ototoxicity.

We feel that the antibiotic selected for the

immediate treatment of patients with septic shock should be bactericidal and provide the broadest possible cover. We therefore recommend gentamycin in combination with ampicillin or cephaloridine.—We are, etc.,

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SIR,—I feel that your leading article on septic shock (3 January, p. 3) places too little emphasis on the part of disseminated intravascular coagulation, to which you pay only perfunctory reference. Corrigan and others¹ in a careful study found thrombocytopenia in 60% of 36 patients with septicaemia and associated multiple coagulation defects, particularly in those with hypotension.

Though it is true that Gram-negative septicaemia in man may not present the full histological features of the Schwartzman reaction as seen in the rabbit,² yet functional evidence of defibrination is ever increasing³ and there are many isolated reports of fibrin thrombi at necropsy. Sandritter found hyaline thrombi in 70% cases septic shock and 19% cases haemorrhagic shock,⁴ and although it may not be clear at present whether disseminated intravascular coagulation is an integral part of all types of shock, as Hardaway postulates,⁵ yet of all the types of shock septic shock is the safest and surest in which to employ heparin therapy.

Furthermore, the work of Whittaker *et al.*⁶ suggests that associated catecholamine release in septic shock is another factor causing disseminated intravascular coagulation and that this is preventable by α -adrenergic blockade. With such a high mortality why indeed are you so cool about this additional aspect of rational therapy, yet extolling the virtue of isoprenaline, when in Gram-negative shock with vasoconstriction "the periphery holds the blood fast . . . instead of leading it into the veins and the anaemic heart works fruitlessly"?—I am, etc.,

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- 4 Sandritter, W., *Deutsches Medizinisches Wochenschrift*, 1967, 13, 408.
- 5 Hardaway, R. M., *Syndromes of Disseminated Intravascular Coagulation*, 1966, Thomas, Springfield, Illinois.
- 6 Whittaker, A. N., McKay, D. G., and Csavossy, I., *American Journal of Pathology*, 1969, 56, 153.
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SIR,—Your leading article (3 January, p. 3) mentions that in the treatment of septic shock correction may be required for acidosis. May I stress, from personal experi-

ence of the resuscitation of a deeply unconscious child with septic shock, that estimation of base deficit and its correction by the infusion of sodium bicarbonate in suitable amounts may be life saving.—I am, etc.,

W. ELLIS.

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REFERENCE

Ellis, W., and Dobson, M., *Journal of Bone and Joint Surgery*, 1964, 46B, 747.

Spinal Board for Road Casualties

SIR,—Since the publication of the series "Road Accidents and the Family Doctor," and the subsequent correspondence related to spinal injuries in particular, there have been many inquiries concerning the use of the short spinal board during extrication of the injured from wreckage and the journey to hospital.

This spinal board was originally described by Dr. J. D. Farrington¹ and ours is an adaptation of it (Fig. 1) drawn to scale. The

board is made from good quality $\frac{1}{2}$ -in. (1.25 cm.) plywood, with all the edges rounded and the board well waxed to facilitate sliding into position. The head-piece has "saw edges" to prevent head bandages slipping. Two 8-foot Britax safety straps and buckles are used to position the patient on the board. Overall width of the board is 35.6 cm.

Before the board is put into use, the patient must first be fitted with a cervical

collar. The board is then slid gently behind the patient, making sure that the lower end is well below the level of the sacrum. The two Britax straps are passed through the upper handholds, behind the board, out from the lower handholds, and around the patient's thigh from the outside to the inside, and then under and over the thigh to the chest buckle (Fig. 2). The straps must

be applied as high as possible in the groin. The forehead can be held in position by triangular bandages or a Velcro type band. The arms are normally outside the straps, and not as shown.

The injured and the board now being one unit, any movements for extricating the patient will not worsen a spinal injury. It sometimes helps to tie the ankles together and to have them supported by a rescuer if fractures have occurred below the knee. The handholds in the board are useful for lifting purposes but within the Road Accident After Care Scheme the "blanket lift" is used with the short spinal board.² Once the casualty is lying on the ambulance stretcher the buckles are loosened to allow the legs to extend. The board provides a firm base for any type of stretcher.—I am, etc.,

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- 1 Farrington, J. D., *Bulletin of the American College of Surgeons*, 1967, 52, 121.
- 2 Easton, K. C., *British Medical Journal*, 1968, 3, 123.

Facial Pain

SIR,—We should like to comment on Dr. J. B. Foster's article on facial pain (13 December, p. 667). While welcoming the mention of temporomandibular neuralgia as a cause of facial pain, we would point out that, although the majority of patients are women (the ratio of male to female patients being 1:3), they come in the age group 15-40 years.

A study of the patients attending the temporomandibular joint clinic at this hospital indicates that the percentage of patients (male and female) under 40 years of age, is as high

as 64%, a figure similar to that of other reports,^{1,2} and which can scarcely be considered to include middle age. The number of completely edentulous patients suffering from temporomandibular joint dysfunction is small—13%—and that reported by Franks³ even lower at 7%. The fault with the dentures is either an increased vertical dimension or an incorrect occlusion causing a displacement on closure, and similar findings were reported by Thomson¹ and Franks³.

Overclosure as a cause of temporomandibular joint pain in the edentulous patient is unusual, although it sometimes occurs in the partly edentulous patient when the posterior natural teeth are lost and not replaced by dentures.—We are, etc.,

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REFERENCES

- 1 Thomson, H., *British Dental Journal*, 1960, 107, 243.
- 2 Franks, A. S., *Dental Practitioner and Dental Record*, 1964, 15, 94.
- 3 Franks, A. S., *British Journal of Oral Surgery*, 1967, 5, 157.

SIR,—In his article (13 December, p. 667) Dr. John B. Foster does not mention a new operation I heard of in the U.S.A. for trigeminal neuralgia. I know of a patient there who was completely cured of this condition by this operation after all other treatments failed and she was near to suicide. The performance of the operation does not interfere with the Gasserian ganglion or the trigeminal nerve, but the osseous foramina through which the affected nerve branch passes are surgically widened. Apparently a *tic douloureux* is caused by the acute swelling of a trigeminal nerve branch, which then gets pinched in the bony foramen too narrow for it during an attack.—I am, etc.,

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Ovulation after the Pill

SIR,—In the perennial discussions on the ill effects of oral contraceptives one aspect which I have not yet seen recorded is their contribution to neonatal deaths. Trying to establish a reliable estimated date of delivery is a daily problem for the practising obstetrician, and for three classes of patients, who make up about 75% of our clinic clientele, it is virtually impossible. These are: Polynesians, unmarried girls, and pill users.

In the latter group it has until recently been assumed that the last menstrual period has been a satisfactory basis from which to calculate maturity, but it is now known that ovulation does not invariably recur 17 days after cessation of the hormones. The patient may have amenorrhoea for some weeks or months, and then conceive at the first, but delayed, ovulation.

If accurate maturity is essential to the obstetric management, as in cases of toxæmia, diabetes, or Rh immunization, labour may be induced at, say, 38 weeks and a baby of 34 weeks may be inadvertently de-

livered to face the otherwise avoidable hazards of prematurity. Radiological and laboratory assessments of maturity are not complete safeguards for the fetus. The difficulty would be partially resolved if patients were to have at least two normal periods after discontinuing the pills.—I am, etc.,

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Oral Contraceptives and Depression

SIR,—Your leading article on oral contraception and depression (15 November, p. 380), reviews the clinical evidence for the emergence of this emotional reaction, which appears to be related to the progestogen content of the pills used. The possible biochemical mechanism discussed seemed to involve a disturbance of tryptophan metabolism, with the net effect of a functional deficiency of pyridoxine (a coenzyme in the decarboxylation to 5-hydroxytryptamine).

In the same issue, an additional clinical syndrome is reported in patients taking oral contraceptives—namely, involuntary movements, resembling chorea (p. 404). Of especial interest is the fact that in four of the five cases described the patients had suffered from rheumatic chorea some years previously. The authors discuss the possibility that a vascular mechanism, activated by the contraceptive agents, underlay the relapses of chorea, in view of various reports of changes in blood clotting properties induced by these drugs.

However, an alternative explanation which could perhaps link these two clinical manifestations, depression and involuntary movements, follows from observations by Dr. A. Barbeau on a family suffering from Huntington's chorea.¹ In younger members of this family the initial symptoms were rigidity and akinesia. Treatment with L-dopa in doses of only 2 g. daily abolished these features, but led to the emergence of classical choreic symptoms previously absent. In older members of the family, who had formerly shown only rigidity but now presented with some rigidity together with chorea, the rigidity was abolished and the chorea aggravated on 1 g. daily of L-dopa. Pertinent to the present communication is the observation that in this patient and in others, a similar aggravation of the chorea could be provoked by the administration of a daily dose of 1 g. L-tryptophan.

It seems reasonable therefore to postulate that a disturbance of tryptophan metabolism might be a common factor leading to both depressive and choreiform syndromes in patients taking oral contraceptives. An investigation of tryptophan metabolism in the chorea group, and perhaps the response of these women to pyridoxine therapy would seem to be justified. Indeed a similar line of inquiry in Sydenham's chorea may well be worth while; the basic mechanisms of this bizarre disorder are not understood and could possibly be biochemically mediated.—I am, etc.,

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REFERENCE

- 1 Barbeau, A., *Lancet*, 1969, 2, 1066.