Supplementation of an anaesthetic usually infers the addition of further volatile or intravenous agents. As hyperventilation will have a negligible effect on nitrous oxide uptake, Dr. Leaming is merely potentiating or enhancing nitrous oxide anaesthesia by simultaneously reducing cerebral flow.—We are, etc.,

J. M. LEIGH.
C. M. CONWAY.

Department of Anaesthetics,
Westminster Hospital,
London S.W.1.

REFERENCES

1 Bergman, N. A., *Anesthesiology*, 1967, **28**, 517.
2 Nunn, J. F., Bergman, N. A., and Coleman, A. J., *British Journal of Anaesthesia*, 1965, **37**, 898.
3 Sykes, M. K., Young, W. E., and Robinson, B. E., *British Journal of Anaesthesia*, 1965, **37**, 314.
4 Prys-Roberts, C., Kelman, G. R., and Greenbaum, R., *Anaesthesia*, 1967, **22**, 257.
5 Moya, F., Morissima, H. O., Shnider, S. M., and James, L. S., *American Journal of Obstetrics and Gynaecology*, 1965, **91**, 76.
6 Barcroft, J., *Researches on Pre-natal Life*, Springfield, Illinois, Thomas, 1947.
7 James, L. S., Weisbrot, I. M., Prince, C. E., Holaday, D. A., and Apgar, V., *Journal of Pediatrics*, 1958, **52**, 379.

SIR,—I have read Dr. H. L. Leaming's letter (4 October, p. 51) on awareness during anaesthesia, and would be the first to congratulate him on a very fine clinical record.

However, his remark that "there is no justification or rational reason for administering a greater concentration of oxygen in the respired gases than that existing in the atmosphere" must surely be in doubt. Recent studies have demonstrated the existence of an intra-pulmonary arteriovenous shunt of some 10% during the state of general anaesthesia, and the effect of this added to the effect of ventilation/perfusion inequalities is to lower the arterial oxygen tension—a potentially dangerous situation. It is to overcome this fall in oxygen tension that has prompted anaesthetists to increase the oxygen concentration in the inspired gas mixture, and it is surely the difficulty in combining this with the problems of maintaining unconsciousness in the patient that has stimulated the recent letters on the subject in your columns.—I am, etc.,

G. W. MORRISS.

Military Hospital,
Colchester, Essex.

Carbenicillin Resistance in Pseudomonas aeruginosa

SIR,—We were interested to observe the findings of Dr. J. H. Darrell and colleagues (19 July, p. 141), as we have been investigating recently the emergence of strains of *Ps. aeruginosa* which show degrees of resistance to carbenicillin higher than those described by them and in previous reports.[1] The detailed findings are presented in the Table, from which it can be seen that six of the ten strains isolated showed, when tested against carbenicillin, a minimum inhibitory concentration (M.I.C.) of 5,000 μg./ml. or more. Initially the M.I.C.s were measured by tube-dilution and replica plate methods, but the results here presented were obtained after retesting using the method of Dr. Darrell and colleagues.

All patients, with the exception of Case 8, who had received a 10-day course of carbenicillin, had received ampicillin on admission and prior to the isolation of the carbenicillin-resistant strains of *Ps. aeruginosa*. It is felt that the presence of these resistant strains bore no relationship to previous exposure to antibiotics, in particular ampicillin, as it can be seen from the Table that there was considerable variation in the interval between institution of the antibiotic therapy and the isolation of *Ps. aeruginosa*. In two instances—namely, Cases 2 and 6—highly resistant strains were isolated after only four days of hospitalization and ampicillin therapy, whereas with Case 1 the resistant pseudomonas was first detected 30 days after antibiotic therapy had been terminated.

In no instance was the *Ps. aeruginosa* considered to be significant on clinical grounds, which would substantiate the opinion expressed recently that the mere presence of this organism is no absolute indication for specific antibiotic therapy.[2] This clinical impression was reinforced by failure in most instances to reisolate the same organism from repeat specimens. The presence of several pyocine types and the absence of a definite epidemiological pattern would suggest that cross-infection played no significant part in the spread of these organisms.

Since the completion of the series we are now isolating carbenicillin-resistant strains of *Ps. aeruginosa* with increasing frequency in this laboratory.

We wish to express our thanks to Dr. R. R. Gillies, of the bacteriology department, University of Edinburgh, for carrying out the pyocine typing of the strains studied, and to Miss Dora A. McNellis for technical assistance.

—We are, etc.,

W. A. BLACK.
R. W. A. GIRDWOOD.

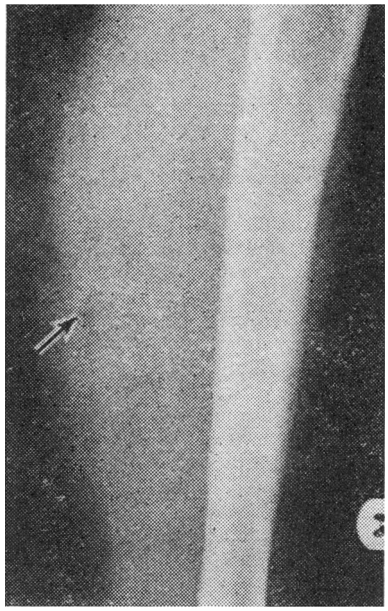
University Department of Bacteriology,
Royal Infirmary,
Glasgow C.4.

Patient	Type of Specimen	Number of Days between Admission and Isolation of Resistant *Pseudomonas aeruginosa*	M.I.C. (μg/ml.)	Pyocine Type
1	Burn swab	60	5,000	1c†
2	Burn swab Burn swab	4 12	10,000–20,000 < 35	1c 1c†
3	Burn swab	8	10,000	1b
4	Urine	30	150	1b*
5	Urine	30	300	1b*
6	Burn swab	4	10,000	1a
7	Burn swab Burn swab	4 10	< 35 10,000	1h 1a
8	Burn swab	14	10,000–20,000	1a

* These strains are absolutely identical and differ very slightly from the other 1b subtype.
† These strains are absolutely identical and differ very slightly from the other 1c subtype.

Tennis Leg

SIR,—In your leading article (6 September, p. 543) no mention is made of soft-tissue radiography as an aid to the diagnosis of tennis leg. Occasionally a gap in the gastrocnemius at its insertion into the Achilles tendon is visible in lateral views of the leg.



The Figure shows a radiograph of the right leg of a 56-year-old woman who had pain in the calf of sudden onset. Examination revealed local tenderness and reduced power in the calf, but no palpable gap, bruising, or oedema.—I am, etc.,

DOUGLAS GOLDING.

Princess Alexandra Hospital and
Harlow Group,
Herts and Essex.

Fibrin Degradation Products

SIR,—We have noted the report of Dr. J. Bonner and others (19 July, p. 137) on the levels of fibrin degradation products (F.D.P.) in the serum of healthy women during normal pregnancy, labour, and the puerperium.

We have made a similar study,[1 2] but with somewhat different findings. We do not agree that the level of F.D.P. is normal in late pregnancy, since we find levels approximately twice normal in sera of women 35 or more weeks pregnant. This confirms the observation of Woodfield *et al.*[3] However, we do agree that a further rise occurs during labour. Our observations indicate that this is maxi-