found in practice to be of equal value and also safe in the diagnosis of two small series of mute psychiatric inpatients.[2 3]

"Poor-risk" patients, especially those susceptible to respiratory or cardiovascular depression, will need extra care, but even then intravenous diazepam and lorazepam have been shown to be less hazardous than intravenous barbiturates.[4] Care is also necessary with epileptics receiving parenteral barbiturates or paraldehyde,[5] patients taking Mandrax[6] and during curarization.[7] Apnoea with diazepam alone has been reported in an 80-year-old patient.[8] Such risks must be balanced against the importance of diagnosing and treating the underlying cause of the mutism.—I am, etc.,

H. G. EGDELL

Airedale General Hospital,
Keighley, Yorks

1 Sargent, W., and Slater, E., *An Introduction to Physical Methods of Treatment in Psychiatry*, p. 145. London, Churchill Livingstone, 1972.
2 Al-Hasani, L. J., and Egdell, H. G. (to be published).
3 Gillespie, F. A., *Canadian Psychiatric Association Journal*, 1971, **16**, 445
4 Knapp, R. B., and Fierre, L., in *Excerpto Medica International Congress Series* 1972, No. 261, 89.
5 Bell, D. S., *British Medical Journal*, 1969, **1**, 159.
6 Doughty, A., *British Medical Journal*, 1970, **2**, 239.
7 Cheymol, J., Driessche, J., van den, Allain, P., and Eben-Moussi, E., *Anesthésie, Analgésie, Réanimation*, 1967, **24**, 329.
8 Buskop, J. J., Price, M., and Molnar, I., *New England Journal of Medicine*, 1967, **277**, 316.

Shake Test on Amniotic Fluid and the Respiratory Distress Syndrome

SIR,—May we comment on the findings of Dr. S. G. Bhagwanani and others (24 March, p. 697)? We can confirm that a negative shake test[1] on amniotic fluid is of poor predictive value for respiratory distress syndrome (R.D.S.) whereas a positive zone result reflects the absence of risk of R.D.S.

From our study of 86 shake tests performed on amniotic fluid obtained by amniocentesis or hindwater amniotomy within 36 hours of delivery at 34 to 42 weeks' gestation 58 gave positive zone results, 17 gave intermediate zone results, and 11 gave negative zone results. Of the 86 only one neonate (34 weeks' gestation) with a negative shake test and lecithin concentration of 3·88 mg/100 ml showed any evidence of R.D.S., but this was attributed to maternal chlormethiazole therapy for severe pre-eclampsia during labour. On 54 specimens obtained from 50 of the 86 cases lecithin concentrations were estimated by the method of Bhagwanani *et al.*[2] The means and ranges for each shake test (see table) are comparable with her data.

Means and Ranges of Amniotic Fluid Lecithin Concentration Related to Shake Test Zone

Shake Test Zone	No. of Cases	Mean Lecithin Concentration (mg/100 ml)	Range of Lecithin Concentration (mg/100 ml)
Negative	12	4·17	3·18-7·50
Intermediate	13	8·40	4·50-17·0
Positive	29	19·04	10·65-28·35

It is interesting to speculate on the significance of our false negative results. Of the 11 cases with negative zone results (34 to 42 weeks' gestation) there was a measured excess of amniotic fluid at hindwater amniotomy in four cases (2,300 ml—3,900 ml) and an unmeasured great excess in another. These excessive volumes of amniotic fluid may have influenced the interpretation of these shake test results in the prediction of R.D.S. We attribute this to a dilution effect and suggest that this may be one explanation for false negative tests which are dependent on amniotic fluid phospholipid concentrations, because hydramnios is not always apparent clinically. Dr. Bhagwanani reports two cases of pulmonary hypoplasia with low results. We have also encountered this abnormality associated with a negative shake test in a fresh stillbirth with multiple congenital abnormalities, but there was also a gross excess of amniotic fluid (4,000 ml).

Finally, we would like to comment on the critical lecithin concentration of 3·5 mg/100 ml recommended by Dr. Bhagwanani[3] in relation to the shake test criteria in the prognosis of R.D.S. Definitive pulmonary function tests[4] were performed on four of the infants with negative zone tests. All four gave normal values for minute volume, lung compliance and lung volume, but two who had had lecithin concentrations of 5·70 mg/100 ml and 7·35 mg/100 ml respectively showed evidence of pulmonary hypoperfusion. The effective pulmonary blood flow values for these infants were 109 ml/kg/min and 111 ml/kg/min (normal, 164 ± 37 ml/kg/min). Pulmonary hypofusion has been shown by Chu *et al.*,[5] to be an important factor in the development of R.D.S. In neither of these two cases described was there an excessive volume of amniotic fluid. We suspect that the critical lecithin level may be too low at 3·5 mg/100 ml if this degree of functional immaturity is to be avoided, and it is at least possible that the apparent margin of safety provided by the shake test is necessary, but we suspect that the critical level lies between those two standards.—We are, etc.,

P. M. FISHER
H. W. SUTHERLAND

Department of Obstetrics and Gynaecology,
University of Aberdeen

R. DINWIDDIE
G. RUSSELL

Department of Child Health
University of Aberdeen

1 Clements, J. A., *et al.*, *New England Journal of Medicine*, 1972, **286**, 1077.
2 Bhagwanani, S. G., Fahmy, D. and Turnbull, A. C., *Lancet*, 1972, **2**, 66.
3 Bhagwanani, S. G., Fahmy, D. and Turnbull, A. C. *Lancet*, 1972, **1**, 159.
4 Dinwiddie, R., and Russell, G., *Biology of the Neonate*, 1972, **21**, 83.
5 Chu *et al.*, *Pediatrics*, 1967, **40**, 709.

Fungal Infections of the Spine

SIR,—The unnamed pathologists who made the diagnosis of cryptococcosis in the case of spinal disease described by Mr. P. Balasubramanian and Professor J. F. Silva (7 April, p. 27) have the thanks of those who are disturbed by the proportion of cases of fungal infection of the spine that goes unrecognized and therefore inappropriately treated. In 20 years I have seen 24 cases of mycosis with predominantly spinal lesions. Only six were diagnosed early enough for successful treatment (three cases of *Blastomyces dermatitidis* infection, two of cryptococcosis, and one of coccidioidomycosis). In 18 cases the disease was misdiagnosed until too late to prevent paraplegia, fatal secondary sepsis, or extension of the fungal infection (three cases each of *B. dermatitidis* infection, *Histoplasma duboisii* infection, and cryptococcosis, two each of candidosis and *Nocardia* brasiliensis infection, and single cases of sporotrichosis and of infection by *Histoplasma capsulatum, Phialophora gougerotii, Torulopsis glabrata,* and an unidentified myceliate fungus).

Mr. Balasubramaniam and Professor Silva refer to the differential diagnosis of cryptococcosis, tuberculosis, and sarcoidosis. Clinically, meningitis, Addison's disease, and osteitis due to tuberculosis might be confused with the corresponding manifestations of cryptococcosis. Rarely, tuberculosis and cryptococcosis coexist. Histologically, cryptococcosis should not be mistaken for tuberculosis; while every variation of the tissue reaction of tuberculosis is duplicated in cryptococcosis, the fungi are there to be seen, usually without difficulty.

The distinction between cryptococcosis and sarcoidosis should not be a problem. The cryptococcus is exceptionally rarely the cause of a sarcoid reaction. If sarcoid lesions in a proved case of cryptococcosis cannot be shown to contain cryptococci, the explanation is usually that they are in fact lesions of sarcoidosis and not a manifestation of the infection; sarcoidosis is second only to classic Hodgkin's disease in predisposing to cryptococcosis.[1] Of 188 cases of cryptococcosis that I have seen, 43 (23%) were associated with Hodgkin's disease and 22 (12%) with sarcoidosis. To argue that in these cases there is in reality no sarcoidosis but a peculiar reaction to the fungus that exactly simulates sarcoidosis is merely to parallel the discredited view that because cryptococcosis may be associated with Hodgkin's disease the latter is in those cases not Hodgkin's disease but a peculiar reaction to the fungus that exactly simulates Hodgkin's disease. The important fact is that certain chronic diseases of the lymphoreticular system specifically lower resistance to cryptococcosis (as they do to dormant histoplasmosis, coccidioidomycosis, blastomycoses and—let us not forget it—tuberculosis).

One diagnostic point noted by Mr. Balasubramaniam and Professor Silva needs amplification. As with comparable tuberculous lesions, open operation on fungal lesions of bone carries a substantial risk of secondary bacterial infection, even today. Accurate diagnosis being essential, diagnostic procedures that introduce a hazard must be undertaken with deliberation.—I am, etc.,

W. ST. C. SYMMERS

Northwood, Middlesex

1 Symmers, W. St. C., *Lancet*, 1967, **1**, 159.

Bacterial Resistance in the Community

SIR,—We wish to comment on Dr. J. E. M. Whitehead's article (28 April, p. 224) which raises the question of bacterial resistance to commonly used antibiotics as seen in general practice.

Dr. Whitehead implies that penicillin-sensitive strains of *Staphylococcus aureus* are predominant outside hospital. In a recent 15-month study of pathogens causing skin and soft-tissue infections in a general prac-

tice of 5,400 patients 66 strains of *Staph. aureus* were isolated. Of these, 36% were sensitive to penicillin, 71% sensitive to tetracycline, and 94% sensitive to erythromycin. Gram-negative pathogens were rarely isolated from such lesions. A study of strains of *Escherichia coli* causing urinary infection[1] over a five-year period (total number of isolates 225) showed that in 1968, 99% were sensitive to ampicillin and 90% to sulphonamide, whereas in 1972, 66% were sensitive to ampicillin and 44% to sulphonamide.

It is likely that the emergence of these resistant organisms is associated with a standard antibiotic prescribing policy in the practice, since 1967, of penicillin for skin and upper respiratory tract infections and of sulphonamide or ampicillin for urinary infections. The acquisition of resistance by common bacterial pathogens to frequently used antibiotics seems to be almost as great a problem in our general practice as in Dr. Whitehead's hospital population.—We are, etc.,

B. T. B. MANNERS
P. R. GROB
G. P. J. BEYNON
F. J. GIBBS

Addlestone, Surrey

1 Manners, B. T. B., Grob, P. R., Dulake, C., and Grieve, N. W. T., *Proceedings of Second National Symposium on Urinary Infection (1972)*. In press.

SIR,—It is commonly believed that staphylococci encountered in the community are predominantly sensitive to penicillin while those in hospital are largely insensitive, a view perhaps encouraged by Dr. J. E. M. Whitehead (28 April, p. 224).

I have recently taken nose and throat swabs from all boys entering this detention centre over a six-month period, who came from a large area of the north of England. Staphylococci were isolated from 132 out of 315 boys (42%). These strains were insensitive as follows: penicillin 78%; streptomycin 2%; cloxacillin 1%; cephaloridine 1%; erythromycin 2%; tetracycline 2%; co-trimoxazole 3%.

It would seem that crystalline penicillin can no longer be considered the drug of first choice in the treatment of staphylococcal infections, though this assumes that the strains isolated from carriers are those which could cause sepsis.—I am, etc.,

AUBREY COLLING

H.M. Detention Centre,
Kirklevington Grange,
near Yarm, Yorks

Pulmonary Embolism from a Femoral Bypass

SIR,—The hazards of a Thomas femoral bypass must be kept in mind when choosing this access to the vessels for chronic haemodialysis. A 24-year-old woman was referred to us in November 1972 with terminal renal failure. After seven unsuccessful attempts to create an arteriovenous fistula, to graft a saphenous loop, and to insert Scribner shunts in different sites a Thomas femoral bypass was eventually performed on the left femoral vessels. In February 1973 she received a cadaver homograft which functioned immediately. Soon after the transplantation clotting of the shunt was noted. Anticoagulant therapy was started to preserve the patency of the bypass in case of rejection but we had to stop anticoagulants after three weeks because of severe operative wound bleeding. From then on the shunt seemed to be obstructed by clots without any symptoms of thrombosis in the lower limb.

The function of the homograft was good (serum creatinine 1·3 mg/100 ml) when suddenly the patient died of cardiac arrest, 39 days after the transplantation. Massive pulmonary embolism was found at necropsy. An organized clot was found in the femoral vein, beginning 15 cm beneath the venous patch of the shunt and clearly disrupted a few millimeters above it. This case prompts us to recommend particular precautions in cases of femoral shunt obstruction, and to advise early removal of the prosthesis with femoral vein ligation in case of posttransplantation thrombosis.—We are, etc.,

ALAIN MEYRIER
VICTOR SCETBON
JACQUES-DOMINIQUE FIEUX

Nephrology and Transplantation Units,
Hôpital Tenon, Paris 20e

Cubital Tunnel Syndrome

SIR,—Messrs. T. J. Wadsworth and J. R. Williams in their article (17 March, p. 662) failed to indicate that it was Drs. W. Feindel and J. Stratford who first named and defined the cubital tunnel.[1 2] These authors drew an analogy between it and the carpal tunnel, described the mechanisms of compression in and clinical features of the cubital tunnel syndrome, and indicated that in some cases simple surgical decompression obviated the need for the more extensive and, in my opinion, frequently ineffective surgical procedures mentioned by Messrs. Wadsworth and Williams.—I am, etc.,

C. F. BOLTON

Victoria Hospital,
London, Ontario

1 Feindel, W., and Stratford, J., *Canadian Medical Association Journal*, 1958, **78**, 351.
2 Feindel, W., and Stratford, J., *Canadian Journal of Surgery*, 1958, **1**, 287.

Cerebrospinal Fluid in Virus Meningitis

SIR,—Contrary to what Dr. E. H. Brown states in his article on enterovirus infections (21 April, p. 169), cerebrospinal fluid (C.S.F.) protein values over 100 mg/100 ml are encountered not infrequently in virus meningitis. Analysis of 113 cases of virus meningitis seen in this department during 1971 shows that in 9% the C.S.F. protein value was above this figure (see table). The analysis also confirms the findings of Dr. J. Stevenson and myself (unpublished observations, 1969) and of Wilfert[1] that C.S.F. sugar values *can* be depressed in virus meningitis. The blood sugar levels were checked in many of these cases but found to be normal.—I am, etc.,

C.S.F. Protein (mg/100 ml)	No. of Patients	C.S.F. Sugar (mg/100 ml)	No. of Patients
Below 15	6	10–19	None
15–45	39	20–29	None
46–70	35	30–39	6
71–99	14	40–49	20
100–199	10	59–59	27
Over 200	None	Over 60	54
Not examined	9	Not examined	6
Total	113	Total	113

B. K. MANDAL

Department of Infectious Diseases,
Monsall Hospital,
Manchester

1 Wilfert, C. M., *New England Journal of Medicine*, 1969, **280**, 855.

General Practitioners' Superannuation

SIR,—I refer to the reply of the Secretary of State to the joint letter of the Chairman of the Compensation and Superannuation and General Medical Services Committees on the position of practitioners who retired before 25 March 1972 (*Supplement*, 21 April, p. 15) and offer the following observations.

Several things have been ignored, such as the case for a special increase for those caught retiring from January 1969 onwards, when the cruel inflationary spiral began to accelerate and the offer of a meeting was ignored. Curiously, the Minister breaks down his own case when he gives as his reason for back-dating the new formula a pious concern lest he cause any suffering to those G.P.s who retired in the period between 25 March 1972 and the date of termination of the negotiations.

An immediate increase of 50% for all of us who retired before 25 March 1972 would do much to remedy a difficult situation for all concerned.—I am, etc.,

N. F. FIELD

Brampton, Cumberland

Representation of Hospital Doctors

SIR,—I sense the depth of Dr. D. G. Ferriman's feelings and understand why he wrote as he did (5 May, p. 309). It is the paradox of politics that those who represent the rest do the best they can, yet the others, who might do the job better, don't come forward. Every year every Hospital Staff Committee has the opportunity to change its representatives on the Regional Committee for Hospital Medical Services. Every year each regional committee has the chance to change its representatives on the central committee. If changes are not more frequent—if the advantages of a democratic process are not seized and used—some blame must lie upon the apathy of the electorate.

Take a close look at what Dr. Ferriman calls the "Central/Regional structure" and consider the provisions made within the B.M.A. organization for the representation of hospital doctors. If you are a hospital doctor and have representatives upon your regional committee whom you don't like or don't trust you can change them by this time next year. Correspondingly, if you are a member of an R.C.H.M.S. you can, before the year is out, choose two different representatives to go to London (at the expense of the Hospital Medical Staff Defence Trust) to act for you and the good of the service.

For a while it was my pride and my privilege to serve upon the Central Consultants' and Specialists' Committee (as it then was) and later upon the C.C.H.M.S. Thoughout that time we tried by bulletins,