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.,

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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.,

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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.,

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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-