there was a deficiency of dopamine in the corpus striatum there would be a release of acetylcholine, which would produce symptoms of its own when it could not fulfil its usual function of releasing dopamine. This would account for the bradykinesia, increasing tremor, etc., which are controlled by giving benzotropine.—I am, etc.,

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Registrar Cover for Obstetric Unit

SIR,—As an elected member of the various committees representing the consultants in this country I can confirm that the enforced registrar reduction implemented by the Department has never been agreed to by any of the committees (Mr. J. H. Hughes, 12 June, p. 656.)

This high-handed manner of changing hospital staff is an affront to the consultants in Britain, which would not be of moment were it not seriously to affect the care of the patients and the responsibility of the consultant to them.

The Department has been implementing these changes surreptitiously and it is only recently that its intention has been disclosed. Meanwhile, the standard of patient care has declined in these areas. It should be noted that this occurrence was used as a "lever" in the recent debates to put through national figures for staff ratios and to set up yet another controlling committee (Central Manpower Committee).

The profession is aware of the acute danger to the patients as a result of these irresponsible bureaucratically - motivated changes in staff. Major disasters may frequently occur if experienced doctors participating in the hour-to-hour care of the gravely ill patients are removed simply to keep the "books straight."—I am, etc.,

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False Economy

SIR,—Far from sharing the pious horror expressed by Dr. A. K. Clarke (29 May, p. 530) I applaud the Department of Health's action in circularizing the profession about the comparative costs of hypnotics.

Many insomniac patients are well suited by butobarbitone or amylobarbitone, and I object to my money being used to provide them with a drug costing seven times as much, except in cases of obvious irresponsibility.—I am, etc.,

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Treatment of Syringomyelia

SIR,—Your leading article on the treatment of syringomyelia (15 May, p. 355) draws attention to the good results obtainable in the communicating form of this condition. There is, however, an unthinking repetition of Gardner's attempts to explain the association with abnormalities at the foramen magnum. There is no evidence that "stenosis of the foramen of Magendie" can ever be "of such a degree as to allow the flow of ventricular fluid at the rate it is being formed yet preclude sudden displacements of ventricular fluid during the cardiac systole." If there is impairment of the passage of fluid out of the fourth ventricle the result is hydrocephalus, not syringomyelia. His ideas that a pulse wave might be "funnelled" into the upper end of the central canal are without clinical, experimental, or theoretical basis. If a pulse wave engendered by arterial systole inside the ventricles were to destroy tissue it would produce damage within the head, not many inches away down the spinal cord.

A more rational explanation of the phenomenon is that it is secondary to fluid movements at the foramen magnum during straining. In the erect position an increase in abdominothoracic pressure may be accompanied by a movement of up to 8 ml of fluid through the foramen magnum. The cause of the upward movement is distension of the epidural veins.[1 2] Under certain conditions cerebellar ectopia or arachnoiditis may cause fluid to move downwards into the communication. The sites of initial destruction of the spinal cord are determined by the relation of grey matter to white matter.[3]

Gardner's explanation is unable to account for syringobulbia. On this account the venous mechanism appears to be obviously the more correct one, but there is additional evidence. The forces and volumes involved in coughing are over 10 times greater than those produced by the "arterial" ventricular pulse wave. Syringomyelia may occur in cases where the aqueduct is blocked. The foramen of Magendie is not blocked in all cases. The venous mechanism can be demonstrated on a fluid analogue model. The "arterial" mechanism has not been successfully demonstrated. The pulsations of arterial origin within the head are highest in children. The pulsations of venous origin are higher in adults. Symptomatic syringomyelia most commonly has its origins in early adult life.

Cases are on record in which a sudden onset of syringomyelic symptoms has occurred in response to a strain likely to give rise to venous distension.[4] The cord cavity is usually flaccid. A flaccid cord can be subjected to stresses by movement of fluid within its cavities whereas the arterial hypothesis demands that the cord be tightly inflated.

Arachnoiditis is *not* a common finding in cases of late cervical syringomyelia following injuries to the dorsal and lumbar spinal cord. In these cases the explanation is similar to the mechanism which produces syringobulbia, that the inside of the cord contains fluid and the upward driving force imparted to the cerebrospinal fluid by the epidural veins during straining drives fluid upwards within the syrinx and thus cause further cavitation.[5] The objective for operation is to make an artificial cisterna magna with free access both upwards and downwards.[6]

Lastly, your leading article does not make a clear distinction between the common, communicating form and cases in which there is an intraspinal tumour causing the syrinx. With tumours the syrinx is not in communication with the cerebrospinal fluid pathways. The investigation, treatment, and prognosis are entirely different.[6] Such cases should be clearly distinguished from cases of "communicating syringomyelia."—I am, etc.,

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Calcium Carbonate, Hypercalcaemia, and Peptic Ulcer

SIR,—I have only recently become aware of the case report "Acute Hypercalcaemia and Renal Failure after Antacid Therapy," by Dr. D. N. S. Malone and Dr. D. B. Horn (27 March, p. 709). While agreeing with their emphasis that ingestion of varying proprietary antacid preparations containing calcium may lead to renal failure and confusion with hyperparathyroidism in peptic ulcer patients, it is felt other points deserve emphasis. The pharmacologically effective dose of calcium carbonate is 4-5 g each hour if neutralization of gastric juice is to be maintained in male duodenal ulcer patients.[1 2] The administration of this enormous dose of calcium carbonate fails to accelerate the healing of gastric ulcers,[3] and the hypercalcaemia rate is approximately 30%.[4]

Consequently, it is strongly felt that intensive antacid therapy has no place in the treatment of peptic ulcer and calcium and sodium containing preparations should never be used. Calcium containing preparations carry the risk of hypercalcaemia, renal stones, and renal failure, as demonstrated by Drs. Malone and Horn, and sodium preparations the risk of heart failure in those whose cardiac reserves are limited. Any of the many sodium and calcium free antacid tablet preparations available appear to be adequate for pain relief.—I am, etc.,

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Care of Chronic Psychotics

SIR,—In your leading article (15 May, p. 351) you quote me as having shown that 30% of admissions to a particular mental hospital were of "no fixed abode". In my original paper which was read at a seminar at the Royal Medico-psychological Association meeting on 10 February, and later quoted in the *Lancet*[1] I made it clear that I was referring to male patients only. Destitution of this kind is of course far less common among women, so that the overall proportion of "no fixed abode" admissions is considerably less than 30%. Although I believe that this problem is a serious one I would not like to spoil my case by exaggerating it.