May I close by assuring students that allegations of negligence in respect of their acts or omissions are very rare. They can, moreover, rely heavily on the abundant goodwill towards them that exists in the profession.—I am, etc.,

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**The question of the legal liability of medical students working in casualty departments has also been discussed recently by our Legal Correspondent (30 October 1971, p. 311).—ED., *B.M.J.*

Examination of the Unconscious Patient

SIR,—Your leading article (6 November, p. 313) stressing the importance of carefully examining the unconscious patient provides a valuable service in emphasizing that bedside methods often provide the key to diagnosis in such circumstances. Since reversible metabolic processes or treatable supratentorial or subtentorial mass lesions can often be the cause of stupor and coma, the prompt and accurate interpretation of clinical signs can be life-saving.[1] However, our own experience about the significance of certain findings in such patients differs from that described in your leader and it may be helpful to mention these few points.

The major immediate clinical issues in the diagnosis of stupor and coma are usually to differentiate between structural and metabolic causes. With the structural lesions, one must distinguish between primary supratentorial and subtentorial abnormalities, since treatment has little to offer primary destructive lesions of the brain stem, but a great deal to offer in the presence of supratentorial masses, where effective action often prevents transtentorial herniation and subsequent irreversible brain-stem injury. These considerations make it of major importance to know the pathological anatomy and physiology that underlies certain signs and symptoms and how these signs combine and evolve in each major condition. Otherwise, one may erroneously conclude—for example, that the brain stem is threatened when it is not, or that a structural lesion has hopelessly damaged the brain when, in fact, reversible metabolic depression is the culprit.

To turn to specific points, our studies indicate that decerebrate posturing often accompanies extensive destructive or metabolic lesions confined to the hemispheres and the diencephalon and need not imply brain-stem dysfunction of mid brain, pons, or medulla. Bilateral pupillary dilatation on neck flexion is *not* a sign of impending tentorial herniation; in fact, although it sometimes accompanies brain disease, it can be a normal phenomenon easily elicited in drowsy young persons and probably represents no more than heightened ciliospinal reflexes. In contrast to your own experience, we find abnormalities in the pattern of breathing to be of considerable localizing value,[2] and at least as useful in appraising the locus and course of severe neurological illness as changes in the motor reflexes or behavioural responses to noxious stimulation. Finally, though fever admittedly has little specific neurological meaning, hypothermia deserves the physician's special attention for it almost always means metabolic depression of the brain in acutely unconscious patients.

I support heartily your suggestion that neurological units geared to treat the unconscious patient offer much that is valuable to patients, staff, and students alike. This is true whether one confines this to strokes or widens the target to develop a comprehensive acute neurological-neurosurgical unit to care for all unconscious and severely injured neurological patients as we have done at the New York Hospital.—I am, etc.,

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1 Plum, F., and Posner, J. B., *Diagnosis of Stupor and Coma*. 2nd edn., Philadelphia, F. A. Davis Co., 1972.
2 Plum, F., in Ciba Foundation Symposium. *Breathing: Hering-Breuer Centenary Symposium*. London, Churchill, 1970.

Hepatitis and Hepatoma in the Tropics

SIR,—The discovery of the association between Australia antigen and viral hepatitis has provided a specific serological marker for at least a proportion of patients with serum hepatitis and carriers of this virus. Studies on the distribution of Australia antigen in normal populations in different geographical areas of the world revealed that this antigen was very rare or absent in normal North American and European communities, but that it occurred frequently (6-25%) in the serum of apparently healthy people living in the tropics and South-east Asia.[1] It has been suggested that this high frequency of Australia antigen in the tropics is due to tattooing, scarification, ritual operations such as circumcision, and transmission by blood-sucking insects.[2]

Chronic carriage of Australia antigen is common in patients with a defective immune response—for example, lepromatous leprosy, acute and chronic lymphocytic leukaemia, and chronic renal failure. It has been shown that a background of repeated parasitic infection induces a number of immunological changes and the immunosuppressive action of malaria is well recognized. For example, McGregor and Barr[3] found a higher incidence of non-reactors to tetanus toxoid among malarious than non-malarious children in Gambia and there is considerable evidence of an early immune depression in plasmodial infection in mice.[4] The high frequency of Australia antigen in the tropics may similarly be related to this immunological phenomenon.

The recent reports of the high incidence of Australia antigen in patients with liver cancer in Senegal, Uganda, and Kenya[5] lends support to the view that such an association could well be of some aetiological significance. An altered immune reactivity in malaria may also affect the outcome of chronic infection with a virus by allowing the virus to become frankly oncogenic and or by interfering with the immune reaction to neoplastic cells. Indeed, an increased incidence of malignant lymphoma caused by murine oncogenic viruses in mice chronically infected with *Plasmodium berghei yoelli* has been demonstrated[4 6] and the hypothesis that a similar mechanism operates in chronic infection with serum hepatitis is currently under investigation.—I am, etc.,

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1 Blumberg, B. S., Friedlaender, J. S., Woodside, A., Sutnick, A. I., and London, W. T., *Proceedings of the National Academy of Sciences*, 1969, **62**, 1108.
2 Zuckerman, A. J., *Bulletin of the World Health Organization*, 1970, **42**, 957.
3 McGregor, I. A., and Barr, M., *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 1962, **36**, 364.
4 Salaman, M. H., Wedderburn, N., and Bruce-Chwatt, L. J., *Journal of General Microbiology*, 1969, **59**, 383.
5 *Nature New Biology*, 1971, **232**, 100.
6 Wedderburn, N., *Lancet*, 1970, **2**, 1114.

Transrectal Prostatic Biopsy

SIR,—Mr. W. F. Hendry and Mr. J. P. Williams quote impressive figures in their interesting article on this subject (4 December, p. 595). However, I feel that their claim for a reliable diagnosis with the Franzen needle in 82% of cases is not substantiated. Using this technique, a proportion of results can only be classified by the cytologist as "suspicious but not definitely malignant." Without other evidence of malignancy, it is debatable whether treatment would be justified. Biopsy in such cases can be repeated and the number of indeterminate results reduced.[1] If "suspicious" results are excluded, the authors have achieved a definite and correct diagnosis in 100 out of 138 cases (72%).

There is the further problem that in clinical practice, cytology results are acceptable as sufficient evidence of malignancy. The authors claim as confirmation of their diagnosis improvement in obstructive symptoms following hormone therapy—a subjective assessment. Conversely, it has been stated that false positive results should not occur,[2] and the one apparent case in this series requires explanation.

These problems associated with interpretation of the results of Franzen needle biopsy require long term follow-up studies for their elucidation, but do not detract from the value of the technique as an outpatient procedure.—I am, etc.,

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1 Sunderland, H., and Lederer, H., *British Journal of Urology*, 1971, **43**, 603.
2 Elkman, H., Hedberg, K., and Persson, P. S., *British Journal of Urology*, 1967, **39**, 544.

Value of Coronary Care Units

SIR,—The controlled trial of home care compared to hospital treatment for myocardial infarction by Dr. H. G. Mather and others (7 August, p. 334) essentially gives evidence that it is ethical to do such a study. This report has resulted in considerable comment in the public press and among medical care specialists in the United States. We therefore find the relative absence of correspondence (21 August, p. 473; 4 September, p. 581; 18 September, p. 704) on this report somewhat puzzling. The randomized study of over 1,000 coronary episodes is a unique effort in determination of the effectiveness of the coronary care unit for monitored hospital care of myocardial infarction. It raises questions concerning the value of coronary care units in community hospitals and introduces a possible hospital-induced mortality effect.

It is certainly not to deny the undoubted value of some coronary care in specialized units to raise the question whether all patients with myocardial infarction receive