May I close by assuring students that an allegation of negligence in respect of their acts on each major accident Ward. They can, moreover, rely heavily on the abundant goodwill towards them that exists in the profession.—I am, etc.,

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Deputy Secretary
Medical Protection Society Ltd.
London W.1

"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 subdural mass lesions can often be caused by subdural and coma, thorough 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 leading article 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 subdural 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 to evolve in each 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 transtentorial 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 important in appraising the likelihood of a 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 hastily 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 where the structures and techniques 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.,

FRED PLUM
New York, N.Y., U.S.A.


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 is associated with a high frequency of this virus.3 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 infec-
tion induces a number of immunological changes and the immunosuppressive action of malaria is well recognized. For example,4 McGregor and Barr5 found a higher incidence of Australia antigen positivity among the cases of malaria than in non-malarious children in Gambia and there is considerable evidence of an early immune depression in such cases.6 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 Kenya7 lends support to the view that such an association could well be of some aetiological significance. An altered immune reactivity in malaria patients may well be one of the factors leading to the outcome of chronic infection with a virus by allowing the virus to become frankly oncogenic or by interfering with the immune reaction to neoplastic cells. Indeed, an increased incidence of malignant lymphoma caused by murine oncoviruses in mice chronically infected with Plasmodium berghei yoelli has been demonstrated4 and the hypothesis that parasitic infections of this virus in chronic infection with serum hepatitis is currently under investigation.—I am etc.,

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