reviews. So to attract these we decided to rely on a mixture of straightforward patriotism and straightforward bribery (one thousand rupees). To achieve a high standard we have invited eight distinguished doctors to be active members of the editorial board, and each original article and review is assessed by two referees—one Indian and one foreign.

This is our first effort. If you like it we would be pleased to hear from you; if you do not we will be grateful if you could write and tell us what you think is wrong and how it might be corrected.

This is, after all, *The National Medical Journal of India*—a journal as much your responsibility as it is ours.

REFERENCE


Non-A, Non-B (NANB) Hepatitis

Hepatitis due to the NANB group of viruses is not as clearly identified as that due to hepatitis A virus (HAV), hepatitis B virus (HBV), and the delta agent (HDV). There is strong evidence to suggest that there are at least three different types of human NANB viruses transmitted by blood transfusion, by coagulation factor administration and by the faecal-oral route. The earliest evidence to suggest the existence of the third type of hepatitis besides infectious and serum hepatitis came through the epidemiological studies of Havens in 1956. A symposium on viral hepatitis in 1975 at Washington D.C., USA focused the attention of hepatologists on post-transfusion NANB hepatitis. Accumulated evidence three years later confirmed the occurrence of NANB hepatitis due to needle pricks. Further, by 1981 another, but less common, NANB virus was described which was transmitted through the plasma. The third type of NANB virus transmitted through contaminated water was suggested by two groups of investigators during 1980 and 1982. Of the three types, i.e., post-transfusion (PTH), post-coagulation factor administration and enteric NANB hepatitis, the first type has been reported mainly from developed countries, the third type is common in developing countries and the second type is infrequent everywhere.

About 75–95 per cent of the reported PTH from different countries is due to NANB infection. It is estimated that about ten per cent of the recipients of blood transfusion develop NANB hepatitis. This occurs more frequently with the transfusion of commercial donor blood as compared with that obtained from voluntary donors. The risk of NANB infection is high amongst females and drug addicts. About 10–25 per cent of all patients admitted to hospital with jaundice have been reported to have NANB infection even though the relationship between blood transfusion and hepatitis has been demonstrated in a very small proportion of them. PTH caused by NANB is very often anicteric or mild but it is an important cause of chronic liver diseases such as chronic hepatitis and cirrhosis. It may also be associated with liver cancer.

Coagulation factor transmitted NANB is infrequent and its incidence amongst patients other than haemophiliacs is not known. Most of the coagulation factor concentrates, anti-haemophilic factor, factor VIII, anti-haemophilic and factor IX complex have been found to transmit NANB infection.

Hepatitis due to faecal-oral NANB infection has been commonly reported in epidemic forms. Community outbreaks of this disease have been reported from India, USSR, Japan, Indonesia, Thailand, Burma, Nepal, Bangladesh, Algeria and the Ivory Coast. This problem has been extensively studied in
India. The mode of transmission of enteric NANB is similar to that of hepatitis A. Adults suffer more than children in epidemics. Pregnant women contract a severe form of the disease and have a higher mortality. There are no reported chronic sequelae. Superinfection with NANB in Hepatitis B carriers has been reported to be an important cause of fulminant and subacute hepatic failure.

The diagnosis of all the three types of NANB hepatitis is by exclusion of infection caused by HBV, HAV and HDV through appropriate serological tests. Attempts to develop a scientific serological test to confirm the diagnosis of NANB virus infection have not yet been successful. Shirachi et al. first published a report of a double immunodiffusion test for NANB hepatitis. Later counter-immunoelectrophoresis, immunofluorescence, radioimmunoassay and ELISA techniques have been used to establish sensitive and specific serological tests. It may come as a surprise to many of us that in spite of the phenomenal advances in immunodiagnostic methods for viral diseases NANB hepatitis still remains a diagnosis by exclusion.

The liver histology in NANB hepatitis is also not helpful in the aetiological diagnosis. Morphological changes are similar to those due to HAV and HBV. The following light microscopic changes, though not specific, have been described mainly in NANB hepatitis: (a) swollen, vacuolated and multilayered bile duct epithelium, (b) fatty change, (c) piecemeal necrosis, and (d) portal infiltration with polymorphonuclear cells and lymphocytes.

Three types of structures—tubular, microtubular and sponge-like inclusions—have been identified in the hepatocytes of patients with NANB hepatitis by electron microscopic studies. Shikata and his colleagues found that the monoclonal antibody 48-1 reacted with the microtubular structures. He suggested that these ultrastructural changes should be considered to be markers of NANB viral infection.

There has been very little advance on the characterization of NANB viruses. The post-blood transfusion NANB virion is reported to be a 22 to 37 nm sized particle with an outer shell and a central core. The post-coagulation factor virion is suggested to be a 22 to 27 nm spherical particle which is probably a small enveloped RNA virus. Enteric NANB is reported to be a 25 to 27 nm particle with an outer shell and an inner core. It is suggested to be a RNA virus. Despite difficulties in isolation and characterization of the NANB virus experimental models have been successfully developed. The chimpanzee and marmoset have been infected with human NANB post-transfusion and coagulation factor related viruses and preliminary studies suggest that Rhesus monkeys may be suitable models for studying enteric NANB infection.

More than two decades have passed since our attention was focused on NANB hepatitis. During this period, techniques to study the viruses have shown tremendous progress. However, the NANB viruses have eluded scientists but there is hope that soon this infection will be better understood and the means to control it elucidated in the same way as has been done for HBV and HAV infection.

REFERENCES

Intensive Respiratory Care in India

Respiratory care came of age in the West about twenty-five years ago but still remains a struggling neonate in India. The purpose of intensive respiratory care is to manage patients with acute respiratory failure, acute on chronic respiratory failure and to help critically ill patients who have diseases that can lead to respiratory failure. Conditions producing acute respiratory failure not only include acute disease primarily involving the lungs but also cover a wide area of general medicine, surgery and obstetrics. Thus, diseases of the central nervous system, in particular neuromuscular paralysis, can lead to hypoventilation and respiratory failure in patients with normal lungs. Many other problems including shock, sepsis, crush injuries and fulminant tropical infections such as tetanus, pyogenic meningitis and malignant malaria can also produce 'pulmonary injury' and necessitate intensive respiratory care and support.

In a country as poor and as densely populated as India, where the incidence of acute infections involving the lungs is still high, the proportion of critically ill patients in a medium-sized hospital is often large. Most critically ill patients will, at one time or another, need respiratory care. Some patients requiring less intensive care can be managed in a ward, but there are many who require intensive care and need the expertise and equipment that only a well-equipped unit can provide. But our commitment towards respiratory care units lacks a sense of priorities. One does not decry the setting up of coronary care units, even though more often than not these mushrooming units are merely status symbols that adorn hospitals. But it is sad to witness neglect of the maintenance of proper ventilation and adequate lung function in a critically ill individual. Most coronary care units ignore the basic fact that the heart and lungs work as a single inter-related unit, so that acute pump failure leads to respiratory failure, and acute severe hypoxia from a respiratory problem can adversely affect the heart. Ventilator support with a high concentration of inhaled oxygen is thus essential to a patient with severe acute pump failure or to a patient with fulminant pulmonary oedema. The use of such support should be prompt and early and not as a gesture of apology to a dying patient.

Respiratory care units in the West are special units that have been designed to primarily look after patients with serious respiratory problems. This is an unnecessary luxury in our country as it would entail the duplication of medical expertise and equipment and would lead to an impossible strain on the few available well-trained nurses in the field. Special Intensive Respiratory Care Units (just as special care units for separate organs or systems) also have an inherent disadvantage in relation to patient care that cannot be easily overcome. In any critically ill individual, whatever the initial cause and nature...