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past. Our hypothesis predicts, therefore, that motoneuron disease will be uncommon in India at the present but that it will increase over the next 50 years.

Dr Clements and colleagues (Oct 29, p 1024) suggest that neurotropic enteroviruses other than poliovirus may cause motoneuron disease. If vulnerability of the CNS to these viruses is also age dependent they may be right. However, their suggestion that the frequency of poliomyelitis can be used as a guide to the overall pattern of enterovirus infection and other agents spread by the faecal/oral route in a community is wrong. Before immunisation programmes eradicated the disease, poliomyelitis was common in areas where enteric infection was rare. Indeed, it is because the geographical pattern shared by both motoneuron disease and poliomyelitis is so uncommon among other diseases that we believe the relation suggests a causal link.

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OUTBREAK OF DENGUE FEVER IN DELHI

SIR,—An epidemic of illness with fever, severe aches, and lethargy lasting 5–10 days was recorded during September and October, 1988, in Delhi.

Eighteen affected adults presenting with upper gastrointestinal haemorrhage were admitted to our gastroenterology ward. These patients were referred with upper gastrointestinal bleeding ascribed to ingestion of aspirin and non-steroidal anti-inflammatory drugs given for their pain and fever. The mean age of the eighteen patients (fifteen men, three women) was 35 years. On admission all of them had had fever for 1–5 days, and upper gastrointestinal bleeding was confirmed by aspiration. Four patients had features suggestive of hypovolaemic shock, and encephalopathy was present in eight. Petechiae distributed all over the body were noted in eight patients. Except for a low haemoglobin (3·1–10·0 g/dl) no other haematological abnormality was found. Total leucocyte counts varied from 5400 to 11 600 µl with a normal differential. Prothrombin times were prolonged by more than 3 s in all patients except two. All had a rise in transaminases, but serum bilirubin and alkaline phosphatase levels were normal except in 1 patient whose bilirubin was 4·2 mg/dl. Endoscopy (not done in the eight patients with encephalopathy or the two in whom the bleeding stopped) consistently revealed multiple superficial erosions in oesophagus, stomach, and duodenum. No patient bled from the kidney or other site.

The triad of febrile illness with severe aching associated with upper gastrointestinal haemorrhage and encephalopathy during an epidemic of fever raised the suspicion of viral haemorrhagic fever, possibly due to Dengue virus.

The patients were given supportive treatment (transfusion, H_2 receptor antagonists, intravenous fluids). Seven of the eight patients with encephalopathy died; the other ten patients recovered. None of the patients with encephalopathy had any abnormalities in serum electrolytes, features of cerebral oedema, or focal neurological deficit. None was hypotensive. It was suspected that the encephalopathy might have been caused by punctate cerebral haemorrhage but the CSF was not examined.

Immediately after the first few admissions of such patients, we asked for a proper surveillance study of the outbreak, with the serological assistance of the National Institute of Virology, Pune. Many sera were collected from victims of the epidemic in hospitals in Delhi, and the National Institute of Virology confirmed our suspicion of Dengue virus infection. Seven of the first thirteen patients in our series, in whom serological tests for viral haemorrhagic fever were available, had high titres of complement-fixing (CFT) and haemagglutination inhibiting (HI) antibody against all four strains of Dengue virus, indicating an anamnestic response. In four of these seropositive patients IgM antibody (capture ELISA) against Dengue virus type 2 was also present, indicating that the epidemic was most probably Dengue-2. In three patients Dengue-virus-like particles were isolated. In the remaining three patients, tests for Dengue virus were inconclusive.

Mosquito-borne flavivirus-induced haemorrhagic fever in children has been reported predominantly from the tropics.[1,2] India is considered to be in the endemic zone for Dengue and Chickungunya virus.[1,2] However, Dengue haemorrhagic fever has been reported only once before from India, during an epidemic in Calcutta.[3] The Delhi epidemic of Dengue virus infection had some unusual features, noted in our series and by other physicians in the city. Haemorrhage from various organs has been reported in Dengue haemorrhagic fever, but upper gastrointestinal haemorrhage was the most important and life-threatening mode of presentation in this epidemic and was the most common reason for hospital admission. Encephalopathy almost always meant a fatal outcome. Diffuse punctate cortical haemorrhage may have been responsible for this fatal manifestation. Earlier reports suggested that Dengue haemorrhagic fever is a disease of children and that adults are rarely affected. However, during the Delhi epidemic most of those affected were adults.

Antibody-dependent enhancement in Dengue fever is the mechanism implicated in the haemorrhagic manifestations.[4] Low-titre antibody from one Dengue virus infection enhances the disease process during a subsequent infection with another Dengue serotype. When this happens IgG antibody to group-specific antigen rises.[4] During the first episode only IgM antibody specific to the infecting serotype appears in the sera. The presence of high-titre IgG in seven of thirteen patients in our series indicates that most of our patients had had at least one Dengue virus infection previously and were thus susceptible to the haemorrhagic disease during the current epidemic. During the epidemic 30% of the population of Delhi were affected and those contracting the disease for the first time are now at high risk of haemorrhagic disease with or without encepholopathy during any subsequent epidemic.

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CLINICAL ISOLATES OF VIBRIO CHOLERAE O1 NOT PRODUCING CHOLERA TOXIN

SIR,—Quarantine examination at international airports in Japan is seen as an important mechanism for preventing the spread of dangerous infectious disease in this country. Cholera, due to *Vibrio cholerae* O1 which produce cholera toxin (CT), is one important target. A few cases of cholera are usually found every year at the Osaka Airport quarantine station; so far in 1988 we have found three (table). Case 2 had a mixed infection with enterotoxigenic *Escherichia coli*; in cases 1 and 3 *V. cholerae* O1 was the only enteropathogen detected. The symptoms were mild.

All three isolates were CT-gene negative when tested by DNA colony hybridisation using a ^{32}P-labelled probe prepared from *E coli* containing a recombinant plasmid coding for the A subunit of CT.[1]

CASES OF V CHOLERAE O1 INFECTION* FOUND AS TRAVELLER'S DIARRHOEA AT OSAKA QUARANTINE STATION IN 1988

Case	Date	Suspected country of infection	Diarrhoea
1 (20, F)	Feb 27	China	2–4 times for 2 days
2 (20, F)	March 19	Thailand	6–7 times for 2 days
3 (28, F)	Aug 7	India	1 time for 1 day

*All biovar El Tor, serotype Ogawa.