Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

radiological examinations; and death certificates are carefully studied, and semi-structured interviews done with the health workers responsible for the patient's care by senior staff from the provincial communicable disease control unit. Health workers are assured that the results of the inquiry will remain confidential and their co-operation has been excellent. Where judged necessary, patients' next-of-kin are also interviewed. Primary-care and hospital-management practices are judged against existing national cholera treatment guidelines. Contributory factors identified have been addressed through policy correction, in-depth retraining of clinical personnel in provincial hospitals in optimum cholera management, intensive education campaigns targeting all emergency service staff, and improved laboratory turnaround times.

Cholera epidemics must continue to demand an active public-health response. It is an intolerable indictment of our global morality if a community remains as vulnerable to the reintroduction of cholera as it was before an epidemic.

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Sir—With modern air-travel, diseases such as influenza and severe acute respiratory syndrome (SARS) can spread across continents and oceans in days. Why, then, did the current (seventh) pandemic of cholera take 10 years to spread across Asia from Indonesia (1961) before causing widespread epidemics in Africa during the 1970s? Subsequently, a further two decades elapsed before Latin America was devastated in the 1990s by this same organism—a haemolysin-negative vibrio, similar to the classical biotype, but sharing properties with the El Tor strain.[1]

In a Seminar on cholera (Jan 17, p 223),[2] David Sack and colleagues note, but do not comment on, this slow progression. Does the vibrio first need to colonise the environment (algae and crustaceans)? Sack and colleagues discuss growth of vibrios environmentally; but are they too pessimistic in asserting that cholera "cannot be eradicated; it is part of the normal flora and ecology of the surface water of our planet"? That non-cholera vibrios (NCVs; serogroups O2–O138) thrive in the environment, is true, but do cholera vibrios (serogroup O1) occur there naturally too or only from human excreta? Where have the Latin American epidemics gone? Why do outbreaks of cholera seldom follow the dire warnings evoked by earthquakes or warfare? Until the full enigma of cholera epidemiology is unravelled, it is too early to deny the long-held view that human cases constitute the reservoir of infection—a prerequisite for eradication.

Vaccination and improved sanitation have synergistic roles in preventing cholera.[2] Sack and colleagues, however, seem unduly negative about parenteral vaccine, because of painful local reactions. Like other travellers in the 1980s, I received many injections of cholera vaccine, with minimal discomfort, to meet entry requirements abroad. Moreover, the most impressive protection with cholera vaccine dates back to the 1970s when 88% efficacy was reported with only a single dose of parenteral vaccine containing adjuvant,[3] which enhances the immune response and probably reduces adverse reactions, as with pertussis vaccine.[4] Sack and colleagues mention the Inaba and Ogawa serotypes of O1 vibrios, but not that these produce only type-specific immunity: Ogawa does not protect against Inaba, and vice versa.[1] Despite advocating oral vaccine, they admit uncertain efficacy in the field, although a live oral vaccine (single Inaba strain) was highly protective against Inaba challenge in volunteers.[2]

During the 1990s, a parallel (eighth) pandemic of cholera started around the Bay of Bengal—caused by what various studies showed was an antigenic mutant of the seventh pandemic O1 vibrio, against which neither Inaba nor Ogawa antibody protects.[5] This new Bengal serotype of O1 (sometimes misleadingly designated O139,[2] as though it were yet another NCV) needs to be incorporated in future vaccines (oral or parenteral) alongside Inaba and Ogawa strains, to produce a balanced immune response to all three. Likewise, the rapid-diagnosis antiserum, which stops the motility of vibrios in patients' stools,[2] should immobilise or agglutinate all three serotypes.

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Hepatocellular carcinoma

Sir—In their Seminar on hepatocellular carcinoma (HCC), Josep Llovet and colleagues (Dec 6, p 1907)[1] raise at least two issues that deserve comment. The first is that it is unclear whether the most useful and widely accepted screening policy for detection of early primary HCC among patients with cirrhosis—ie, 6-monthly α fetoprotein estimation and liver ultrasound scan—might be delivered to greatest effect in a transplant centre as opposed to a general hospital setting.

In Eastbourne, UK, where the district general hospital serves a local population of just over 200 000, about 120 patients with biopsy-proven cirrhosis were followed up by two of us (ADS, AAD) and required screening for HCC as outlined above. This imaging burden of almost 250 ultrasound scans per year is not inconsiderable for a radiology department that has just two ultrasound machines and is also required to provide timely services for the patients of several other hospital specialists and general practitioners. Furthermore, this figure does not speak to the complexity of image interpretation nor the time required for this; it does not take account of the need for additional computed tomography (CT) or magnetic resonance imaging in instances where ultrasound scans gave equivocal images within a cirrhotic liver; nor does it include the necessary staging CT scans once a diagnosis of HCC has been established.

An alternative management strategy would be to have these patients travel to and from the nearest liver transplant centre (in the case of Eastbourne,