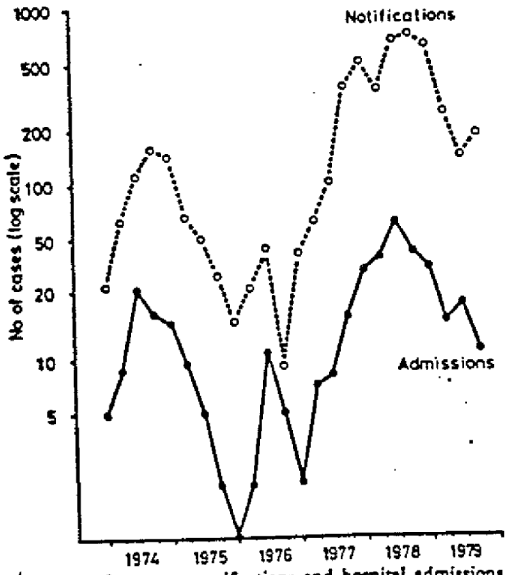

r of statutory notifications and hospital admissions ooping cough in each quarter, 1974–9.

s and hospital admissions ($r=0.9$; $p<0.01$). There was a tendency ughout the period, when the figures for whooping cough increased, for number of notifications to increase proportionately more than the nber of admissions. In the peak quarters of 1974 and 1978 there were 25 58 admissions and 159 and 666 notifications respectively. We studied spital Activity Analysis statistics for a range of other respiratory and ective diseases in childhood during the period but found no disease other n whooping cough for which admissions corresponded with the quarter quarter trends in whooping-cough notifications. There were three deaths ne in 1974, one in 1977, and one in 1979—ascribed to whooping cough the region during the period.

mment

Various interpretations of these figures are possible. Conceivably controversy about whooping-cough vaccination led general actitioners both to notify and to refer for admission to hospital a gher proportion of incident cases in recent than in earlier quarters. is also conceivable that epidemics of respiratory illnesses other an whooping cough had been misdiagnosed as whooping cough th by general practitioners and by hospital doctors. The simpler, d in view more tenable, interpretation is as follows. Firstly, the ospital admission corroborate the view that the "epidemic" ions represented an epidemic of whooping cough. Secondly, th the death rate from whooping cough remained low during e period, the epidemic included a rise in the number of patients hose illness was serious enough to need hospital care.[6] None the ss, as the incidence of the disease rose, either there was a proortionately greater rise in the number of mild than of severe cases or ere was an increase in the proportion of incident cases which were otified.[1]

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Post-transfusion non-A, non-B hepatitis in the Netherlands

To study the incidence of post-transfusion non-A, non-B hepatitis we have followed prospectively 380 recipients of HBsAg-negative blood for up to seven months.

Patients, methods, and results

Blood samples were taken from each patient before transfusion and at monthly intervals afterwards. Activity of alanine aminotransferase (ALT) in excess of 20 IU in two or more consecutive serum samples were taken as a sign of post-transfusion hepatitis. All patients had ALT activities below 21 IU before transfusion. Markers of hepatitis B virus infection were determined by radioimmunoassay (hepatitis B surface antigen (HBsAg), anti-HBs, and anti-HB core (HBc) antigen, Abbott Laboratories, USA) or enzyme immunoassay[1] (HBeAg and anti-HBe). The role of the following viruses in causing these episodes of hepatitis was examined by determining the presence of IgG and IgM antibodies to hepatitis A virus (radioimmunoassay, Abbott Laboratories, USA), cytomegalovirus (enzymeimmunoassay, van Loon A M, and others, unpublished), and Epstein-Barr virus (immunofluorescence).[2]

Incidence of post-transfusion hepatitis in a group of 380 prospectively followed recipients of HBsAg-negative blood

No of recipients negative for markers of hepatitis B virus	No (%) transfused patients with:				
	ALT>20 IU	Hepatitis A virus-IgM	Cytomegalovirus	Epstein-Barr virus-IgM	Non-A, Non-B hepatitis
380 (100)	15 (4·0)	0	1* (0·3)	1† (0·3)	13 (3·4)

*Significant increase of antibody titre by enzyme-linked immunosorbent assay and complement fixation.
†One of the 7 donors of this recipient was also positive for IgM-Epstein-Barr virus.

The results, shown in table I, indicated that 15 of the 380 recipients developed hepatitis; all were asymptomatic. In the absence of a practicable test for non-A, non-B antigen(s) or antibody, 13 of these patients, in whom cytomegalovirus, hepatitis A, and Epstein-Barr viruses were not implicated, were considered to have post-transfusion non-A, non-B hepatitis. The number of units of blood they received varied from 1-10, with a mean of 3. The peak activities of ALT were mildly increased (28-100 IU, with a mean of 48 IU) in 11 patients, but in two patients they were 174 and 777; the former patient was the only one in our group in whom ALT activities remained raised for more than four months. The incubation period, defined as the interval between transfusion and the first significant increase in ALT activities, was five weeks in three patients, six to 11 weeks in eight (including the two with the highest levels), and over 11 weeks in two. These incubation periods agreed with those reported in the USA for patients with post-transfusion non-A, non-B hepatitis.[3]

Comment

There is a controversy in published reports about the value of increased ALT activities in donor blood in predicting the development of post-transfusion, non-A, non-B hepatitis. Some investigators found that the likelihood of developing such hepatitis increased greatly when the ALT activity in donor blood exceeded 45 IU,[4] while others could not confirm these findings.[5]

In our group of 38 donors whose blood was implicated in post-transfusion non-A, non-B hepatitis the highest activity of ALT was 21 IU. This fact, together with the fact that only volunteer blood is used in the Netherlands, may explain the lower incidence of post-transfusion non-A, non-B hepatitis in our group (3·4%) than in the US (5·4-18·5%).[4] Furthermore, because of our liberal criteria for diagnosing non-A, non-B hepatitis, its real incidence in our patients may have been even lower.

Nevertheless, the finding that 3·4% of a group of recipients of donor blood that had been screened for HBsAg developed non-A, non-B hepatitis emphasises the need for practicable methods of detecting non-A, non-B antigen(s). Furthermore, such methods are needed for studying the causal role of non-A, non-B agent in "auto-immune" chronic active hepatitis and in the serological diagnosis of acute hepatitis.

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