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## Hepatitis A Outbreak in a Residential School

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**Summary:** Hepatitis A outbreak in a residential school. C. R. Boughton, R. A. Hawkes, N. I. Lehmann and G. S. Grohmann, *Aust. N.Z. J. Med.*, 1980, **10**, pp. 4-6.

*Outbreaks of hepatitis A occurred sequentially in two wards for children in a residential institution for the mentally retarded in Sydney. Twenty-six of 41 children were initially susceptible to hepatitis A. In the first ward affected, 11 of 13 susceptible children (85%), and in the second ward, four of 13 susceptibles (31%), were infected. Of the 15 showing serological evidence of infection, five (33%) were clinically jaundiced.*

The recent development of laboratory techniques for the detection of hepatitis A particles (HAV) in stools and subsequently serological tests for detection of antibodies to hepatitis A virus (anti-HAV) in serum, has enabled more precise definition of the diagnosis and epidemiology of hepatitis A. During a recent outbreak in two wards of a residential institution

for the mentally retarded, appropriate specimens were obtained from inmates for liver function tests, for anti-HAV (total and specific IgM) and for HAV particles in stools.

### Materials and Methods

The population studied were residential inmates of two wards, A and B. Ten of the total 41 patients had Down's syndrome (DS) and the remainder had mental deficiency due to other causes (non-Down's, ND). These children were moderately retarded, ambulant and able to attend special schools. Some suffered episodes of diarrhoea and the personal hygiene of some children was unsatisfactory. The children from the two wards ate in the same dining room but at different times, played separately, attended several different schools outside the hospital, but occasionally joined an organised outing together.

The index case was a nine-year-old girl diagnosed on 2 February 1978, in Ward A which housed 22 patients aged from five to 14 years. The first case in Ward B was an 11-year-old girl diagnosed on 18 February 1978; this ward housed 19 children, aged from eight to 15 years.

Pooled human immune globulin was given by intramuscular injection to all children of Wards A and B on 3 February.

Blood specimens were taken initially from those suspected of having hepatitis A, from all inmates on 15 February and again five weeks later. Those who were seronegative at the latter bleed had a further serum specimen tested ten weeks later. Stool specimens were collected from the 34 children in whom this was practicable.

### Laboratory Methods

**Sera:** Liver function tests (serum bilirubin, serum glutamic pyruvic transaminase and serum glutamic oxalacetic transaminase) were carried out on all serum specimens. Examination for hepatitis B surface antigen (HBsAg) was carried out by radioimmunoassay (Ausria II, Abbott Laboratories). The techniques used for the detection of anti-HAV (total and IgM specific) in sera were based on the radioimmunoassay method of Purcell *et al.*<sup>1</sup>

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TABLE 1  
Hepatitis A serological status of inmates of Wards A and B, and attack rates among susceptibles

	Total in ward	Number susceptible to HAV	Number infected with HAV		
			Icteric	Nonicteric	Total
Ward A	22	13	3	8	11
Ward B	19	13	2	2	4
Totals	41	26	5	10	15

*Stool specimens:* Stool specimens were prepared for solid phase radioimmunoassay (SPRIA) and immunoelectron-microscopy (IEM) by a method based on that described by Locarnini.<sup>2</sup> The SPIRA technique used for the detection of HAV particles in stool specimens was a modification of that of Purcell.<sup>1</sup>

#### Findings

Of the 22 patients in Ward A nine had anti-HAV, but of non-IgM type, in their sera, indicating prior immunity. Of the 13 susceptibles, 11 developed serological evidence of recent HAV infection with the appearance of specific IgM anti-HAV. Three (27%) of these had clinical jaundice (Table 1) and ward staff suspected hepatitis in three of the non jaundiced patients because the children became listless, stools were noted to be pale and urine dark brown. These children were shown to have serological evidence of HAV infection.

Ten of the 11 infected children showed biochemical and serological evidence of HAV infection in the first serum specimen obtained on 15 February. The remaining patient did not show IgM anti-HAV until the third specimen taken at the beginning of June.

In Ward B six of the total 19 children were found to be already immune; of the 13 susceptibles, four showed serological evidence of HAV infection of whom two had clinical jaundice (Table 1). Of the four patients infected, seroconversion occurred by 17 February in two, and the other two had seroconverted by 24 March.

The administration of pooled human immune globulin to seronegative children did not result in detectable anti-HAV titres when sampled 12 days later.

No sera showed the presence of HBsAg.

Stools from four of 34 children examined contained HAV. HAV particles were not found in specimens from patients without clinical, biochemical and serological evidence of hepatitis A. Enterovirus-like particles were detected in seven patients, and coronavirus particles in five. The significance of the viruses other than HAV is not known.

#### Discussion

The outbreak in Ward A was explosive, five children developing clinical illness within two days. Ten of the 11 showing evidence of HAV infection apparently acquired the virus at approximately the same time from a single

source. It is probable that the latter was a 13-year-old girl who had subclinical infection; serial sera from this child showed no increase in total anti-HAV but did show the presence of anti-HAV of IgM type. All other patients with infection showed both an increase in total anti-HAV and the presence of specific IgM anti-HAV. The remaining patient who did not seroconvert until June, had subclinical infection probably modified by the injection of pooled human globulin.

Ward B may have been infected either from the original source or sequentially from another inmate of Ward A; the latter patients were probably infective for two to three weeks prior to the onset of their illness. Spread among Ward B inmates was apparently sequential, in that two children had clinical hepatitis on 18 and 20 February, and two had (subclinical) hepatitis shown serologically five weeks later. The two cases with clinical hepatitis in Ward B had received globulin on 3 February, apparently too late to render the infection subclinical.

The rate of clinical expression did not differ significantly between DS and ND patients. Five of 15 patients infected (33%) were clinically jaundiced, a rather higher proportion than has been considered usual in HAV infection. However a recent similar serological and epidemiological study revealed a high clinical expression rate.<sup>3</sup>

In this environment the attack rate of hepatitis A among susceptibles was high (in Ward A 11/13 (85%), Ward B 4/13 (31%), overall 58%). The patients in both wards who developed evidence of HAV infection late in the outbreak, had all

received globulin and all had subclinical infection.

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## Coronary Artery Spasm: Use of Ergonovine in Diagnosis \*

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**Summary:** Coronary artery spasm: Use of ergonovine in diagnosis. S. B. Freedman, R. F. Dunn, L. Bernstein, D. R. Richmond, G. O'Neill and D. T. Kelly, *Aust. N.Z. J. Med.*, 1980, **10**, pp. 6-11.

*Ergonovine maleate was administered to 69 patients with chest pain but without significant coronary artery disease (<70% luminal diameter obstruction) to determine whether coronary artery spasm could be provoked. Coronary artery spasm was seen at angiography, or inferred from ECG or thallium myocardial perfusion scan changes. The test was positive in 16 patients: all five patients with documented variant angina (Group A); ten of the 19 patients with suspected variant angina (Group B); one of the 11 patients with exercise-induced chest pain (Group C); and none of the 34 patients with atypical chest pain (Group D). Patients with a positive test usually smoked, complained of recurrent nocturnal or early morning chest pain, showed ST changes during spontaneous chest pain and had minor degrees of fixed coronary obstruction (30-70%), when compared to those with a*

*negative test. The only major side effect of the test was transient ventricular tachycardia which occurred in three patients and was reverted by sublingual and parenteral nitroglycerine.*

Coronary artery spasm may cause variant angina pectoris<sup>1,2</sup> and precipitate both unstable angina<sup>3</sup> and myocardial infarction.<sup>4</sup> Although spontaneous spasm has been observed at angiography<sup>2</sup>, this occurrence is so uncommon that a reliable provocative test for spasm has been sought. Ergonovine maleate, a vasoconstrictor, was first used as a test for coronary artery disease in 1949 by Stein<sup>5</sup>, and recently coronary artery spasm has been provoked by ergonovine in patients with either fixed coronary arterial obstruction or normal coronary arteries.<sup>6,7</sup> The present study evaluated the diagnostic use of ergonovine in patients with chest pain but without significant coronary obstruction.

### Methods

#### Patient Selection

Sixty-nine patients (36 male, 33 female) were given ergonovine, after significant fixed coronary artery obstruction ( $\geq 70\%$  luminal diameter reduction) had been excluded by coronary arteriography. Patients with a history of previous myocardial infarction were not included. The patients were divided clinically into four groups.

*Group A* consisted of five patients with documented variant angina pectoris, defined as recurrent angina at rest with transient ST segment elevation.

*Group B* consisted of 19 patients with suspected variant angina but without ECG documentation of ST segment

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