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Communicable Diseases Intelligence

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Editor Dr I.F. Cook

Contents:

VIRUS REPORTING SCHEME: A total of 1 047 reports were processed for this period.

Nine cases of Q fever were reported, 8 from Queensland and 1 from New South Wales. Occupational exposure data were only available for 4 of the Queensland cases:-

- 3 male meatworkers (1 from Brisbane aged 18 years, 1 from Ipswich aged 17 years and 1 from Roma aged 47 years);
- one 42 year old male livestock truck driver from Rockhampton.

Rubella infection was serologically confirmed in a 16 year old male who presented with encephalitis, a rare complication of the disease.

Adenovirus type 1 was isolated from the faeces of a 32 year old male AIDS patient who presented with Kaposi's sarcoma and persistent diarrhoea.

Cytomegalovirus was isolated from the duodenal biopsy specimen of a HIV antibody positive, 43 year old male who was investigated for malabsorption.

Poliovirus type 2 was isolated from the faeces and urine of a 4 month old male who was admitted to hospital with pneumonitis. The patient had received his first dose of oral poliomyelitis vaccine at 2 months of age and has subsequently been diagnosed as having severe combined immunedeficiency syndrome (SCIDS), a condition in which both humoral and cellular immunity is impaired.

TYPHOID OUTBREAK - MOUNT HAGEN (PAPUA NEW GUINEA)

On 30 January 1987, the Papua New Guinea (PNG) national press reported an outbreak of typhoid in Mount Hagen, in the Western

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Highlands province, with three deaths notified and thirty-three persons listed as critically ill.

The Secretary of PNG Department of Health has advised that this outbreak actually occurred about three weeks ago and has now been brought under control. The PNG authorities have, as a matter of routine, also notified the WHO regional office.

Typhoid is known to be endemic in this region of the country, and major outbreaks occur from time to time. Visitors to the region are reminded that, provided normal sanitary and hygiene procedures are adhered to, there is little danger of infection, however those staying in villages for any length of time should exercise greater care.

CDI Editorial comment

Typhoid immunisation is not required for entry into any country. It is recommended as protection for persons who will be travelling in areas where the disease is prevalent such as Asia, Africa and Latin America. Protection lasts approximately three years.

Typhoid fever is an acute disease due to Salmonella typhi which is characterised in typical cases by fever, toxaemia, diarrhoea or constipation, abdominal tenderness, enlarged spleen and a rose-coloured macular eruption confined to the trunk.

Infection with Salmonella typhi is related to poor food and water sanitation practices and occurs irrespective of climate in countries where these practices are poor. Vaccination is thus offered to those travelling to or resident in such countries. However, it must be emphasised that, as typhoid vaccination offers only limited immunity to infection, care should be exercised in the selection of food and water in these countries.

Primary immunisation

Primary immunisation is achieved by subcutaneous injection with two doses of the vaccine given with an interval of 4-6 weeks between doses. When immunisation is required in a shorter period of time, the two doses should be given at an interval of not less than seven days. The dose schedule by age is tabulated below:

Dose Schedule

	1st dose	2nd dose
Adults and children over 12 years of age	0.5 mL	0.5 mL
Children aged 6-12 years	0.25 mL	0.25 mL
Children aged 1-5 years	0.1 mL	0.2 mL
Children less than 1 year*	0.1 mL	0.1 mL

* Infants are not usually inoculated with typhoid vaccine since typhoid infections rarely occur in this age group.

Booster immunisation

Under conditions of continued or repeated exposure, a booster dose should be given at least every three years. When more than three years has elapsed since previous immunisation, a single booster injection provides a sufficient protective response.

The booster injection may be given either subcutaneously or intradermally with the latter being preferred in individuals with a history of severe adverse reaction to previous typhoid vaccination. The subcutaneous booster dose is the same as the second dose of the primary vaccination program but 0.1 mL is sufficient for intradermal dosage.

Side effects and adverse reactions

Typhoid vaccination often results in 1-2 days of discomfort at the site of injection with redness and induration. This local reaction is not infrequently accompanied by systemic symptoms such as fever, malaise and headache. These symptoms respond within 24-48 hours to rest, adequate fluids and antipyretics. The severity of adverse reactions tends to increase with age and repeated vaccination.

Precautions and contraindications

To ensure an even dispersion of the organisms, the vial containing the vaccine should be vigorously shaken immediately before use. Vigorous physical exertion should be avoided for 24-36 hours after each inoculation.

Typhoid vaccine should not be given to anyone who is suffering or convalescing from an acute or chronic illness or to anyone who is known to have renal disease.

Pregnancy is not a contraindication to typhoid vaccination, but it is prudent to avoid any form of active immunisation during pregnancy, especially in the first trimester.

THE INFLUENCE OF IMMUNITY ON RAW MILK-ASSOCIATED CAMPYLOBACTER INFECTION

(based on JAMA, Jan 2, 1987 - Vol 257, No. 1)

Campylobacter jejuni is now recognised as an important cause of diarrhoeal illness[1,2]. Cattle frequently excrete this organism[3,4], and numerous outbreaks previously have been associated with raw milk consumption[3-7], especially among persons acutely exposed to this vehicle[5]. However, dairy farmers frequently drink raw milk without becoming ill, and in developing countries where C. jejuni infection is hyperendemic, there is an age-related decrease in incidence of infection and in the case-to-infection ratio[8]. These observations suggest that acquired immunity could be important in preventing infection or in preventing illness after infection.

The following reports an outbreak of Campylobacter enteritis in which epidemiologic analysis associated first-time consumption of raw milk with illness. Attack rates and serological

responses of the patients were compared with those of regular raw milk consumers to see if prior exposure and thus immunity might be a determinant of outcome. The relationships between the amount of milk ingested and the rapidity of onset and severity of the illnesses that ensued, were also examined.

On October 17, 1982, 31 college students, all aged between 17 and 21 years, went on a retreat to an Oregon dairy farm. On arrival, they ate breakfast, consisting of commercial dry cereal, eggs, and unpasteurised milk that was produced at the farm. The day was spent relaxing in a hot tub and eating food brought on ice from the college residence, including ham, potato salad, and hamburgers. Some of the same food had been left at the college residence and was eaten by members who did not go on the retreat. That evening, the 31 students returned to the College residence. Between October 18 and 27, an acute gastrointestinal illness developed in 19 of the 31 students who had visited the dairy farm, while the 42 other college members remained well ($P<.001$, Fisher's exact test).

Twenty nine of the 31 students consumed raw milk at the farm. Twenty two (including the three with asymptomatic infections) of 29 persons exposed to raw milk at the farm became infected, while neither of the two not drinking raw milk became ill or met the case definitions. A culture for C. jejuni was obtained from one of the two non-milk drinkers and the culture was negative; no culture was obtained from the other.

Acute and convalescent C. jejuni - specific IgA, IgG, and IgM levels were low and no different from those of the college members who stayed at the residence. Risk of infection could not be related to any other source for this organism. Four of the students who drank the raw milk without becoming infected had substantial previous raw milk consumption experience. When they were excluded from the calculations, leaving 25 students who were acutely exposed to the raw milk, 22 (88%) had become infected vs none of the two nondrinkers ($P<.05$, Fisher's exact test). More strikingly, 19 (76%) of the 25 acutely exposed students became ill vs none of 10 persons (6 farm workers and 4 students) who were regular raw milk drinkers and who drank the implicated milk ($P<.001$). Stool cultures were obtained from 8 of the 10 chronic raw milk drinkers and all were negative for the outbreak strain. Increasing amount of raw milk consumed by the student was associated with increasing risk of infection and illness, a trend that became more significant when the 4 students with histories of chronic past raw milk exposure were excluded (Table 1).

Illness had onsets one to ten days following exposure to the milk. The mean (4.2 days), median (3 days), and modal (3 days) incubation periods were similar; however, the incubation period was dose related. Among the 19 students who became ill, onset within the first five days following exposure occurred in 33% of those drinking one glass, 80% of those drinking two glasses, 100% of those drinking three or four glasses ($P<.05$, Bartholomew's test); all three asymptomatic excreters consumed less than two glasses of raw milk. Those who drank greater

quantities of raw milk also tended to suffer a more severe illness, as evidenced by seeking medical care and hospitalisation (Table 2). Secondary spread of illness to college members who did not go to the farm was not observed.

Table 1 - Relationship between the number of glasses of raw milk consumed and the occurrence of C. jejuni infection among college students, Oregon, 1982

No. of glasses	All Students*			Students without previous raw milk consumption+		
	No. of students	No. infected	No. ill	No. of students	No. infected	No. ill
0	2	0	0	2	0	0
1	13	8	6	11	8	6
2	7	6	5	6	6	5
3	9	8	8	8	8	8
TOTAL	31	22	19	27	22	19

* Relationship of number of glasses to infection: $P < .05$
Relationship of number of glasses to illness: $P < .05$
(Bartholomew's test for trend).

\+ Relationship of number of glasses to infection: $P < .01$
Relationship of number of glasses to illness: $P < .01$

Table 2 - Relation between severity of illness and amount of raw milk consumed in 22 C. jejuni infected persons

Severity of illness	No. of persons	No. (%) of persons consuming > 480 ml (16 oz) of raw milk
Asymptomatic	3	0
Symptomatic, no treatment	6	1 (17%)
Symptomatic, saw physician	6	2 (33%)
Symptomatic, hospitalised	7	5 (71%)

The evidence implicating raw milk as the vehicle for C. jejuni infection in this outbreak is substantial. The absence of illness in college members who did not visit the farm points to the retreat as the source of infection, and most foods eaten during the retreat also were served to the college members who remained at the residence. Consumption of one food served only at the retreat, raw milk, was significantly associated with infection in those without prior exposure, and a dose-response relationship between attack rates and amount of milk consumed was found. No other food was implicated. The serotypes of the

patient strains were essentially identical to that of the C. jejuni strain isolated from the cow manure. Raw milk is well recognised as a vehicle for C. jejuni and C. fetus infection[3-7], but because no reliable method to isolate the organism from raw milk was available at the time of outbreak, no such attempt was made.

The symptoms observed in the patients during this outbreak are similar to those reported previously in patients with Campylobacter infections. Diarrhoea and abdominal cramps were prominent, but only two patients (9%) reported bloody diarrhoea. The availability of free and unlimited visits to the University Student Health Centre probably affected the findings. Hospital laboratory-based series of patients with C. jejuni infections who reported a higher occurrence of bloody diarrhoea[9-11] may involve selection bias, since patients with severe symptoms are more likely to seek medical attention than those with mild illnesses. Other community-based studies showed similar low incidences of bloody diarrhoea[5,12]. Presumed dose of ingested C. jejuni organisms in this outbreak appeared to be important in determining likelihood and severity of illness and varied inversely with incubation period.

The investigation of this outbreak provides new information on immunity to C. jejuni infections. As in other point-source outbreaks due to raw milk consumption, the attack rate was high among those exposed who had never previously consumed raw milk[5]. Apparently raw milk is an excellent vehicle for C. jejuni, either because milk-associated strains are highly virulent, or because milk buffers gastric acid, to which C. jejuni is susceptible[13]; multiplication of the organism in milk does not occur[13]. Nevertheless, none of the chronic raw milk drinkers became ill after ingesting large amounts of the same milk that caused a high attack rate among those persons who were acutely exposed.

Their negative cultures suggest either that they did not become infected or that their asymptomatic excretion had ceased or was of insufficient magnitude to be detected by the time cultures were obtained nine days later. Presumably this phenomenon is due to previous exposure to C. jejuni with subsequent development of immunity. Preliminary studies of human volunteers have provided evidence for homologous reinfection immunity. In this outbreak, the absence of illness in the chronic raw milk-drinking students who had never previously visited this farm supports the concept of heterologous immunity as well. Acquisition of immunity is the best explanation for the inverse relationship of age to incidence of C. jejuni infections and case-to-infection ratios that occur in developing areas where C. jejuni infection is hyperendemic[7].

The mechanisms of immunity to C. jejuni are not completely understood. Acute symptomatic infection is associated with rise in levels of C. jejuni specific serum IgA, IgG, and IgM antibodies[14-17]. Among acutely exposed college students who became ill in this outbreak, significant rises in levels of C. jejuni-specific IgG and IgM in convalescent-phase serum samples were found compared with both the acute-phase serum samples and control serum samples (Table 3). In contrast to previous findings[14,17], specific serum IgA levels showed a

small but not statistically significant increase, perhaps because convalescent-phase serum samples were obtained after the brief rise in IgA level had occurred[14]. Among acutely exposed students who remained well, acute-phase specific IgA and IgG levels were not different from those in unexposed persons or in those exposed who became ill. The significance of the lower acute-phase IgM levels in those remaining well, will need to be examined in subsequent studies. That three of these persons were culture positive and that specific antibody levels in all three immunoglobulin classes rose in the convalescent serum, indicate that these individuals had indeed become infected. Absence of illness may have been related to the dose ingested rather than pre-existing immunity.

Table 3 - Serum antibodies for C. jejuni by Immunoglobulin Class as measured by ELISA

	Mean (± SE) Optical Density 414 for			
	Acutely Exposed Group 1			
Immunoglobulin Class	1A Ill (n=19)	1B Well (n=6)	Unexposed Group 2 (n=13)	Chronically Exposed Group 3 (n=10)
	Acute-Phase Serum			
IgA	0.11±0.08	0.02±0.01	0.15±0.09	0.09±0.03
IgG	0.64±0.44	0.38±0.09	0.39±0.08	0.66±0.16*
IgM	0.40±0.06°	0.13±0.03*	0.30±0.03	0.50±0.11*
	Convalescent-Phase Serum			
IgA	0.16±0.04	0.07±0.01#	0.13±0.07	0.13±0.03
IgG	1.30±0.48*#	0.62±0.15#	0.36±0.06	0.76±0.16*
IgM	1.78±0.49*#	0.38±0.09#	0.36±0.03	0.58±0.15*

* Immunoglobulin-class-specific optical density values compared with values for the 26 acute- and convalescent-phase serum samples from unexposed controls by one-way analysis of variance showed significant P values: $P < .05$.

° Compared with acute-phase IgM from acutely exposed well persons, $P < .05$.

\# Within groups, convalescent-phase serum samples compared with acute-phase serum samples for rise in optical density value by one-tailed paired t tests showed significant P values: $P < .05$.

The rise in convalescent-phase antibody levels in the chronic raw milk drinkers who also were acutely exposed at the farm suggests that they too became infected. Absence of illness in the group must be ascribed to immunity, and the significantly elevated levels of C. jejuni-specific IgG and IgM in their acute-phase serum supports that hypothesis[18]. However, it is also possible that the elevated levels in the farm workers may have been due to exposure to the epidemic strain in the previous days or weeks. Serosurvey of healthy chronic raw milk drinkers and appropriate controls will be needed to assess the prevalence of elevated levels of C. jejuni - specific serum antibodies. Among older children and adults in Bangladesh and Thailand whose infection rates and case-to-infection ratio for C. jejuni are lower than for young children[8], C. jejuni - specific serum immunoglobulin levels were consistently higher than for young children[19,20]. As with other enteric pathogens[21], it is likely that C. jejuni - specific serum antibody levels, especially that of IgA, reflect levels of gut immunity[22].

The presence of prolonged or recurrent illness due to C. jejuni in hypogammaglobulinemic patients[23,24] supports the role of humoral immunity in protection against this infection. Homosexual men have high rates of infection with C. jejuni and other Campylobacter species, that frequently are asymptomatic[25]. The infrequency of severe or prolonged diarrhoeal illness due to C. jejuni reported among homosexual men with acquired immunodeficiency syndrome (AIDS)[26] argues against cell-mediated immunity playing an important role in these intestinal infections. In contrast, Salmonella infections, which are known to be controlled by intact cell-mediated immunity[27], are disproportionately common in patients with acquired immunodeficiency syndrome (AIDS)[28]. Whether the high C. jejuni - specific antibody levels observed in this or the prior studies are protective in themselves or merely reflective of other immune phenomena is not answerable at present. Nevertheless, this investigation confirms that the presence of these antibodies in persons chronically exposed to raw milk and for the first time, shows an association between high antibody levels and immunity to infection under field conditions.

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VACCINE RELATED POLIOMYELITIS IN NON-IMMUNISED RELATIVES AND HOUSEHOLD CONTACTS

(based on BMJ Vol. 294, 17 January 1987)

The diagnosis of paralytic poliomyelitis is now rarely considered in cases of acute lower motor, neurone weakness in patients resident in Britain. Paralytic poliomyelitis still occurs, however, and two cases are reported, one of which would not have occurred had the present immunisation procedures been followed. The occurrence of the other case suggests that immunisation procedures should be modified.

Case 1

A 16 year old boy developed a sore throat, nausea and anorexia, and general malaise two weeks after his niece, a household contact, had received oral poliomyelitis vaccine. Four days later he deteriorated with generalised headache and photophobia, and the next day he awoke with a completely paralysed left arm. He was said to have been immunised against poliomyelitis as a child but this could not be confirmed. On admission he had a fever of 38°C, mild neck stiffness and photophobia, and flaccid paralysis of his left arm with absent reflexes in the arm and normal sensation. Results of routine blood tests were negative, as were viral antibody titres. Examination of cerebrospinal fluid yielded 15 x 10^6 lymphocytes/l, a protein concentration of 0.33 g/l, and a glucose concentration of 3.6 mmol/l (65 mg/100 ml). Serum antibody titres to poliovirus on the day of admission and 14 days and six weeks later were less than 16 to P1 (poliovirus type 1) and P3 (poliovirus type 3) but showed a fourfold or greater rise to P2 (poliovirus type 2) (1/64, 1/256, 1/1024). Stool culture grew a poliovirus type 2 of a vaccine related strain.

The patient's systemic symptoms settled rapidly in two days, but over the next five years there was no appreciable return of neurological function in the left arm.

Case 2

A 23 year old toolmaker developed a sore throat and mild frontal headache 47 days after his son had been immunised against poliomyelitis. Three days later his condition became worse with generalised headache and vomiting and general malaise. A week after his initial symptoms he noticed difficulty swallowing and weakness of his voice and was admitted to hospital. He had never been immunised against

poliomyelitis and had had a tonsillectomy. On admission he was feverish (38°C) with mild neck stiffness. Neurological examination showed nystagmus on lateral gaze, severe bilateral facial weakness, and weakness of jaw opening. Palatal movement was absent, though sensation was normal and there was no cough. There was severe weakness of the neck flexors and tongue protrusion. The limbs showed mild weakness of the right deltoid, biceps, and triceps with reduction in these reflexes but no other abnormalities and no sensory loss.

Initial examination of cerebrospinal fluid yielded 41 x 10^6 mononuclear cells/l with a protein concentration of 0.5 g/l and glucose concentration 3.5 mmol/l (63 mg/100 ml). Stool cultures from the patient and his son grew a poliovirus type 3 of a Sabin-like strain. Initial serum antibody titres to poliovirus types 1, 2, and 3 were negative but a subsequent specimen after 10 days showed a greater than fourfold rise to P3 only (1/1024). The initial cerebrospinal fluid poliovirus antibody titre was negative but a repeat examination 10 days later showed a titre of 1/32 to P3. Serum antibody titres to other viruses (Coxsackie and ECHO) were negative.

Owing to failure to control secretions the patient required a tracheostomy shortly after admission. He developed no further weakness and made a gradual recovery over the next six weeks, being left with a mild bulbar palsy and weakness of his sternomastoids.

Discussion

Both these patients had clinical illnesses characteristic of paralytic poliomyelitis. Neither had been previously immunised. Though the first patient was thought by his parents to have been immunised, this could not be confirmed and his initial antibody titres showed this not to be the case. Both patients were excreting a vaccine related strain of poliovirus, and antibody titres in blood and cerebrospinal fluid (case 2) showed evidence of recent infection with the appropriate vaccine related strain confirming that they had vaccine related poliomyelitis.

Poliomyelitis is defined as being "contact vaccine associated" if it occurs in a patient within 4 to 60 days after contact with a recently vaccinated person[1,2]. In case 2 the period of 47 days after immunisation of the patient's son before the development of paralytic poliomyelitis was clearly well within this definition, and furthermore the son was still excreting a similar Sabin related strain. Vaccine related poliomyelitis in contacts is well described and estimated to occur with a frequency of 0.2-0.4 cases per million doses[1,2]. The WHO collaborative study showed that in 69% of cases the patients were aged 15 or over and many were young non-immunised parents of children receiving primary immunisation. Vaccine associated poliomyelitis in contacts occurs most commonly with strains related to P2 and P3[2]. Studies have shown that Sabin polioviruses increase in neurovirulence on passage in the human gut, and recently this has been found to be associated with a single nucleotide change in a non-coding region of the genome of the Sabin type 3 poliomyelitis vaccine[3].

These two patients are therefore typical examples of vaccine associated paralytic poliomyelitis in contacts. Neither had been immunised and they were in contact with relatives receiving primary immunisation; one case was associated with a P2 Sabin like strain and the other with P3.

To prevent vaccine associated poliomyelitis in contacts it is currently recommended that non-immunised parents of infants receiving primary immunisation should be immunised either before or at the same time as their children. Naturally this is often forgotten, and clearly the second patient would not have contracted paralytic poliomyelitis had these recommendations been followed. Case 1 could not have been prevented since the patient was believed to have been immunised and present recommendations regarding immunisation extend only to parents. The occurrence of case 1, however, emphasises that the recommendations should be extended to household contacts of the child being immunised.

Health visitors now interview parents before primary immunisation, and a simple way to prevent further cases would be to include a question about the immunisation status of parents and household contacts and then to ensure that all non-immunised relatives and household contacts are immunised either before or at the same time as their children.

*** RECOMMENDATION ***

NON-IMMUNISED PARENTS AND HOUSEHOLD CONTACTS OF CHILDREN RECEIVING PRIMARY IMMUNISATION SHOULD BE IMMUNISED AGAINST POLIOMYELITIS AT THE SAME TIME AS THEIR CHILDREN.

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IMPORTED POLIOMYELITIS - IMPLICATIONS FOR TRAVELLERS TO THIRD WORLD COUNTRIES

(Based on California Morbidity #32, 15 August 1986).

In June 1986, poliomyelitis was diagnosed in a 29 year old woman from Auburn, California (Placer Co) who had been in Nepal and Burma before her onset of illness on May 10. Her overseas itinerary prior to the onset of illness included:-

- from January to 2 May 1986 - she worked in Nepal
- between 14 and 24 April 1986 - she was on a raft trip in Nepal
- between 3 and 9 May 1986 - she was in Burma
- on 10 May 1986 - the travelled to Bangkok, Thailand, where she had onset of fever (38.9°C) malaise, restlessness and a general feeling of weakness lasting one day.

She was well until 16 May when the again experienced fever (39.1°C), headache, and low back pain. The next day she had onset of weakness in her legs, more severe on the right, decrease urinary urgency, and constipation.

By 19 May she could not walk and was hospitalised in Thailand. No sensory, cranial nerve, or central nervous system abnormalities were noted. On 6 June she was flown back to the United States, and to her parents' home in Auburn, still confined to a wheelchair, although the constipation and decrease urinary urgency had disappeared. On 11 June her right lower extremity was entirely flaccid except for some minimal strength in the glutei.

There was moderate weakness and loss of tone in the left lower extremity in the glutei. quadriceps, and peroneal and calf muscles. By late July, over 60 days after weakness onset, the left leg appeared to have recovered completely and strength of muscles above the knee in the right leg was greatly improved. However, paralysis of muscle persisted below the right knee.

Laboratory results included the following:-

- CSF obtained during the acute illness in Thailand:
  . White cell count of 90 with 93% lymphocytes and 7% polymorphs (it is not known whether CSF protein was evaluated).
- Stool specimen collected on June 22:
  . poliovirus type 1 was isolated and subsequently characterised as wild virus by the Centers for Disease Control (CDC, Atlanta).
- Serum obtained on 27 June and tested for poliovirus complement fixing (CF) antibodies:
  . antibody titre to polivirus type 1 of 1:16
  . antibody titre to poliovirus type 2 and 3 of 1:18

- Electromyogram and nerve conduction velocity studies on 26 June (when she was recovering clinically) showed widespread degeneration changes in the muscle below the right knee.
- Immunocompetence workup, including IgM and IgG quantitation as well as quantitative immunoelectrophoresis, was normal in a blood specimen obtained on 27 June.

The patient's mother recalls that her daughter had received three Salk vaccine injections in the late 1950's and one dose of Sabin vaccine ('sugar cube') at a mass public clinic in Long Beach, California in the early 1960's but keeps no records.

The patient took no polio vaccine doses before her travel to Nepal and recalls no outbreak of paralytic illness or exposure to recently immunised persons during her travel in Nepal and Burma.

Editorial Comment:

Travellers to developing countries should be considered at risk of exposure to wild polio virus. Even persons who have previously received a primary immunisation course may need a "booster" of poliomyelitis vaccine before travelling to such areas.

For adults who previously completed a basic course of three doses of oral poliomyelitis vaccine (OPV [Sabin type]), another booster of OPV should be given and is necessary every ten years to maintain protection in those likely to be exposed overseas.

Those at risk include all travellers to areas where poliomyelitis is epidemic or endemic. For travellers who have received a partial course, the primary series should be completed.

For adults who previously completed a primary series of inactivated poliomyelitis vaccine (IPV), a dose of either IPV or OPV may be given. If IPV is used, additional doses may be given every five years if the person remains at risk (but the need for these additional doses has not been established).

For details on the immunisation of unvaccinated adults and the immunisation of those who are partially immunised or with unknown immunisation status, as well as the immunisation of children, please consult the latest:-

. 1986 - Third Edition of 'Immunisation Procedures' published by the National Health and Medical Research Council and available upon written request from:

The Secretary
Communicable Diseases Committee
Commonwealth Department of Health
PO Box 100
WODEN ACT 2606

. 1986 - Revised Edition of 'Health Information for International Travel' published by the Commonwealth Department of Health and available upon written request from:

The Director
Information Review and Policy Section
Communicable Diseases Branch
Commonwealth Department of Health
PO Box 100
WODEN ACT 2606

DRAFT CLINICAL HANDBOOK ON EPIDEMIC POLYARTHRITIS

The Australian summer and autumn seasons co-incide with the greatest incidence of human arboviral infections. Recent articles in CDI have highlighted the occurrence of these diseases[1-5].

The most common human arboviral infection in Australia is epidemic polyarthritis (EPA). EPA is caused by infection with the mosquito-borne alphavirus, Ross River virus. (The disease is occasionally and incorrectly termed Ross River fever). EPA has been recorded from all mainland States and Territories, and from New Guinea. It has also caused severe epidemics in the South Pacific[6]. The aetiological agent has been isolated from mosquitoes in Tasmania[7].

Clinical diagnosis of EPA is based on polyarthralgia, often accompanied by variable rash, headache, fever or muscle pain[6]. Diagnosis should be confirmed by appropriate serology.

Although EPA is a notifiable disease throughout Australia, it is clear that this disease is very much underreported. Accurate reporting of this disease is essential for proper planning of preventive programs.

To facilitate a better awareness of EPA, the Commonwealth Department of Health, in collaboration with representatives of State/Territory health authorities and other experts in the field, has produced a draft clinical handbook on epidemic polyarthritis. The handbook is being circulated to approximately 2000 medical practitioners across Australia for their comments on its content. Based on responses received, the handbook will be amended and it is planned to provide copies of the final version to all registered medical practitioners in Australia. It is hoped that the handbook will prove to be a valuable aid to practitioners in the diagnosis and management of epidemic polyarthritis.

Medical practitioners who would like to receive a copy of the draft document for comment should contact

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 19-1-87 to 8-2-87 BULLETIN NUMBER 87/3
VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WVH (ACT)	RAHC (NSW)	PHH/ POW (NSW)	FAIR-FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
0100 ADENOVIRUS NOT TYPED			1		5	3	6		15
0101 ADENOVIRUS TYPE 1				2	4				6
0102 ADENOVIRUS TYPE 2	1				4	1			6
0103 ADENOVIRUS TYPE 3				1		1			2
0105 ADENOVIRUS TYPE 5				1					1
0106 ADENOVIRUS TYPE 6						1			1
0107 ADENOVIRUS TYPE 7					1				1
0119 ADENOVIRUS TYPE 19						1			1
0137 ADENOVIRUS TYPE 37								1	1
0199 ADENOVIRUS TYPING PENDING			1		2		2	1	6
0201 INFLUENZA A VIRUS			1						1
0203 INFLUENZA B VIRUS	1		1				1		3
0206 INFLUENZA A VIRUS SUBTYPE H1N1				1					1
0301 PARAINFLUENZA VIRUS TYPE 1		1			1	5			7
0303 PARAINFLUENZA VIRUS TYPE 3				2	4	6	6	5	23
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)			2			7	4	6	19
0500 RHINOVIRUS (ALL TYPES)				1	5	8	4		18
0600 MYCOPLASMA PNEUMONIAE			1					8	9
0904 COXSACKIEVIRUS B4						1			1
0906 COXSACKIEVIRUS B6						1			1
1005 ECHOVIRUS TYPE 5				1				1	2
1006 ECHOVIRUS TYPE 6						1			1
1011 ECHOVIRUS TYPE 11				20	7	1		1	29
1014 ECHOVIRUS TYPE 14						1			1
1022 ECHOVIRUS TYPE 22				1					1
1031 ECHOVIRUS TYPE 31						1			1
1100 POLIOVIRUS NOT TYPED					2				2
1102 POLIOVIRUS TYPE 2		1							1
1200 MUMPS VIRUS				1			1	1	3
1300 HERPES VIRUS GROUP-NOT TYPED	9			1		1	1	1	13
1301 HERPES SIMPLEX VIRUS NOT-TYPED				1				2	3
1302 EPSTEIN-BARR VIRUS (EB VIRUS)					1		11	5	17
1303 VARICELLA-ZOSTER VIRUS			3	2		1	1	12	19
1306 HERPES SIMPLEX TYPE 1	2			55		22	60	34	173
1307 HERPES SIMPLEX TYPE 2	4			59		17	66	62	208
1399 HERPES VIRUS TYPING PENDING					2			4	6
1401 COXIELLA BURNETI	1						8		9
1502 PICORNA VIRUS-NOT TYPED			1				5	2	8
1521 MEASLES VIRUS					1				1
1522 RUBELLA VIRUS				5		4	13	1	23
1532 HEPATITIS B ANTIGEN	3		8	19	1	24	22	21	98
1535 HEPATITIS A ANTIBODY		1	2	5		7	1	4	20
1541 CHLAMYDIA A - C TRACHOMATIS				24		32	49	62	167
1543 CHLAMYDIA A - LGV TYPE	3								3
1556 CMV - CYTOMEGALOVIRUS			4	16	2	3	9	14	48
1564 ROTAVIRUS	4		1		1	16	19	1	42
1599 ENTEROVIRUS TYPING PENDING		2	8		3				13
9992 ROSS RIVER VIRUS							6	5	11
9998 ARBO. GROUP B. ...							1		1
Total	28	5	34	218	46	166	296	254	1,047

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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Viral Identifications by Clinical Information Table 1.
Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; E3 -Encephalitis; M3 -Meningitis; 04 -Paralysis; 05,13 -CNS other unspec.; 07,49 -GI; 17,47 -Hepatic; 19 -CVS; 89 -Urinary; 06 -Skin/mucous.

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respir atory	Enceph alitis	Mening -itis	Para- lysis	CNS other unspec	GI	Hepa -tic	CVS	Urin -ary	Skin/ mucs memb
0101 ADENOVIRUS TYPE 1.....		3					3				
0102 ADENOVIRUS TYPE 2.....		5					1				
0103 ADENOVIRUS TYPE 3.....	1						1				
0105 ADENOVIRUS TYPE 5.....		1									
0106 ADENOVIRUS TYPE 6.....		1									
0107 ADENOVIRUS TYPE 7.....		1									
0199 ADENOVIRUS TYPING PENDING.....		2									
0201 INFLUENZA A VIRUS.....		1									
0203 INFLUENZA B VIRUS.....		2									
0206 INFLUENZA A VIRUS SUBTYPE H1N1		1									
0301 PARAINFLUENZA VIRUS TYPE 1....		7									
0303 PARAINFLUENZA VIRUS TYPE 3....	2	19									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	1	18									
0500 RHINOVIRUS (ALL TYPES).....		16									
0600 MYCOPLASMA PNEUMONIAE.....	1	8						1			
0906 COXSACKIEVIRUS B6.....	1										
1005 ECHOVIRUS TYPE 5.....		1		1							
1006 ECHOVIRUS TYPE 6.....		1									
1011 ECHOVIRUS TYPE 11.....				19			3				
1014 ECHOVIRUS TYPE 14.....	1										
1022 ECHOVIRUS TYPE 22.....		1									
1031 ECHOVIRUS TYPE 31.....				1							
1200 MUMPS VIRUS.....				1							
1301 HERPES SIMPLEX VIRUS NOT-TYPED											2
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	5	2		1				1			
1303 VARICELLA-ZOSTER VIRUS.....											19
1306 HERPES SIMPLEX TYPE 1.....	2	11				1				1	103
1307 HERPES SIMPLEX TYPE 2.....	4	2									65
1401 COXIELLA BURNETI.....		1									
1521 MEASLES VIRUS.....											1
1522 RUBELLA VIRUS.....			1								19
1532 HEPATITIS B ANTIGEN.....	24							50		1	
1535 HEPATITIS A ANTIBODY.....	2							14			
1541 CHLAMYDIA A - C.TRACHOMATIS...	10										
1543 CHLAMYDIA A - LGV TYPE.....	2										
1556 CMV - CYTOMEGALOVIRUS.....	1	11				1				2	2
1564 ROTAVIRUS.....	1						42				
9992 ROSS RIVER VIRUS.....	2										2
9998 ARBO. GROUP B.	1										
Total.....	61	115	1	23		2	50	66		4	213

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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Viral Identifications by Clinical Information Table 2.
Code 10 -Eye; 59 -Genital; 39 -Endo/sal gland;
38 -RES; 29 -Muscle/joint; 69 -Congenital; P8 -PUO;
G8 -Fever/malaise; 09 -Other; A1 -SIDS ...

VIRUS OR VIRAL ANTIGEN	Eye	Genital	Endo/sal gland	RES	Muscle/joint	Congenital	PUO	Fever/malaise	Other	SIDS
0101 ADENOVIRUS TYPE 1.....									1	1
0102 ADENOVIRUS TYPE 2.....									1	
0103 ADENOVIRUS TYPE 3.....								1		
0119 ADENOVIRUS TYPE 19.....	1									
0137 ADENOVIRUS TYPE 37.....		1								
0203 INFLUENZA B VIRUS.....							1	1		
0206 INFLUENZA A VIRUS SUBTYPE H1N1								1		
0303 PARAINFLUENZA VIRUS TYPE 3....							1	1		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....				1						
0500 RHINOVIRUS (ALL TYPES).....					1			1		
0904 COXSACKIEVIRUS B4.....									1	
1011 ECHOVIRUS TYPE 11.....			1				1	7		2
1102 POLIOVIRUS TYPE 2.....									1	
1200 MUMPS VIRUS.....			3							
1301 HERPES SIMPLEX VIRUS NOT-TYPED		2								
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			4		2		1	5	1	
1303 VARICELLA-ZOSTER VIRUS.....							1			
1306 HERPES SIMPLEX TYPE 1.....	9	48							2	
1307 HERPES SIMPLEX TYPE 2.....		136							2	
1401 COXIELLA BURNETI.....					1		1	7		
1522 RUBELLA VIRUS.....			2		9	1		2		
1532 HEPATITIS B ANTIGEN.....									23	
1535 HEPATITIS A ANTIBODY.....									4	
1541 CHLAMYDIA A - C.TRACHOMATIS...	5	151							1	
1543 CHLAMYDIA A - LGV TYPE.....		1								
1556 CMV - CYTOMEGALOVIRUS.....		3	1		1	8	2	3	16	
1564 ROTAVIRUS.....									1	
9992 ROSS RIVER VIRUS.....					9			2		
Total.....	15	342	11	1	23	9	8	31	54	3

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

Period 10 - 6 September 1986 to 3 October 1986

Bulletin 87/3

Disease	N.S.W.	VIC.	QLD.	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	Cumulative Total to Date for Year
Amoebiasis	1			1	2			1	5	42
Ankylostomiasis				2			NN		2	29
Anthrax									-	-
Arbovirus infection			81		NN				81	1 179
Brucellosis			1						1	12
Campylobacter infections	97		NN	140	12	NN	9	2	260	2 036
Chancroid				NN					-	9
Cholera									-	-
Congenital rubella syndrome	1		NN			NN		NN	1	1
Diphtheria							5		5	31
Donovanosis				NN	5		5		10	109
Giardiasis	29		NN	60	9	NN	NN	NN	98	988
Genital herpes	74		4	31	NN	NN	2	1	112	1 056
Gonococcal ophthalmia neonatorum	1	NN			NN	NN		NN	1	1
Gonorrhoea	96		3	46	136	4	53	8	346	3 671
Hepatitis A (infectious)	15	3	3	28	28	1	1	1	80	* 1 399
Hepatitis B (serum)	44	2	21	4	27		5	7	110	1 371
Hepatitis – unspecified	8	2	2	1	NN	NN	1		14	120
Hydatid disease					1				1	9
Lassa fever			NN			NN		NN	-	-
Legionnaires disease			NN		1	NN		NN	1	56
Leprosy	1				2				3	18
Leptospirosis	2	3	6						11	143
Lymphogranuloma venereum				NN	NN	NN	1	NN	1	3
Marburg disease			NN			NN		NN	-	-
Malaria	18	6	2	1	2		1	3	33	542
Meningococcal infections		1	1		4	NN			6	37

Disease	N.S.W.	VIC.	QLD.	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	Cumulative Total to Date for Year
Non-specific urethritis	236		NN	19	NN	NN	NN	1	256	3 403
Ornithosis	1		2		2	1			6	35
Pertussis (whooping cough)	7	3	NN	6	6	NN		NN	22	* 446
Plague									-	-
Poliomyelitis						1			1	1
Q. fever	14		17	5	1				37	259
Rabies				NN		NN		NN	-	-
Salmonella infections	49	13	21	22	12	3	24		144	1 983
Shigella infections	8	3	1	5	6		16		39	638
Smallpox									-	-
Syphilis	19		9		20		67		115	1 649
Tetanus									-	5
Trachoma		NN			2	NN	NN		2	143
Tuberculosis (all forms)	19	27	3	3	7		4	NN	63	778
Typhoid fever	3	1							4	30
Typhus (all forms)									-	12
Vibrio parahaemolyticus infections			NN			NN		NN	-	5
Yellow fever									-	-
Yersinia infections	8		NN	2		NN		NN	10	67

NN - Not Notifiable

(Note: Data collected under the Notifiable Diseases Returns may bear little or no correlation to that collected under the CDI laboratory scheme. Whilst the latter is a sampling program, the Notifiable Diseases data is dependent upon voluntary reporting by medical practitioners etc.)

* Adjustment to the Cumulative Total since last report

Hepatitis A (infectious)	+1	South Australia
Pertussis (whooping cough)	+1	South Australia