ISSN 0725-3141

Communicable Diseases Intelligence

Bulletin number 86/9
Issue date: 5 May 1986

Editor: Dr I F Cook

Contents:

VIRUS REPORTING SCHEME A total of 886 reports were processed for this period.

Eight cases of Q fever were reported (5 from New South Wales, 2 from South Australia and 1 from Western Australia). Occupational exposure data were only available for the two South Australian cases, male abattoir workers, aged 34 and 48 years. The latter patient presented with a chronic subacute bacterial endocarditis. None of these patients were involved in the Q fever vaccine field trial conducted in South Australia.

Cytomegalovirus was isolated from:

- the amniotic fluid of a 12 week old foetus, aborted following several maternal infections with cytomegalovirus and rubella.
- the post-mortem specimen of lung tissue of a 35 year old male bone marrow transplant patient who died of a generalised sepsis with multiple abscesses.
- the lung biopsy specimen of a 30 year old male bone marrow transplant patient who was experiencing host rejection of the transplanted tissue.

Rubella virus was isolated from an 8 month old female with thymic aplasia who died of pneumonia following a rubella infection. It is not known whether the infection was acquired pre or postnatally, although a skin rash was observed by the mother one month prior to the child's death.

AIDS SURVEILLANCE

1) AUSTRALIA

To 29 April 1986, 203 cases of AIDS fulfilling the criteria of case definition have been reported to the National Health and Medical Research Unit in AIDS Epidemiology and Clinical Research. The distribution of those patients by State or Territory of notification and by risk group are shown below.

The Bulletin is compiled and distributed by the Communicable Diseases Branch, Department of Health, P.O. Box 100, Woden, A.C.T. 2606, Australia, and is available on request

Table 1: AIDS patients by State or Territory of notification

STATE/TERRITORY	CASES			DEATHS		
	Male	Female	Total	Male	Female	Total
NSW	134	5	139	64	3	67
VIC	29	-	29	13	-	13
QLD	17	1	18	11	1	12
WA	9	2	11	5	-	5
SA	2	-	2	-	-	-
NT	2	-	2	1	-	1
TAS	1	-	1	-	-	1
ACT	1	-	1	-		-
	195	8	203	95	4	99

Table 2: AIDS patients by risk category

RISK GROUP	CASES	DEATHS
Homo-/Bi-sexual	176	79
IV drug Users-	-	-
Homo-/Bi-sexual IV Drug abuser	2	2
Blood transfusion recipient	19	14
Person with haemophilia	3	3
Heterosexual transmission	2	1
None of the above	1	-
	203	99

2) OTHER COUNTRIES

COUNTRIES	CUMULATIVE UP TO	CASES	DEATHS
U.S.A.	07/04/86	19,181	10,152
U.K.	31/01/86	287	144
CANADA	01/04/86	511	255

PREVENTION OF HAEMOPHILUS INFLUENZAE, TYPE B
(Based on MMWR (1986) 35:11)

Haemophilus influenzae, type B (Hib) causes a variety of serious childhood diseases including meningitis, epiglottitis, osteomyelitis, septic arthritis, cellulitis and pneumonia[1]. A polysaccharide vaccine against systemic Hib disease was licensed in the United States in April, 1985 and guidelines on its use was subsequently issued by the Immunisation Practices Advisory Committee (ACIP)[2][3]. ACIP has recently updated its recommendations for use of the vaccine and provided guidelines for the prevention of secondary cases of Hib disease[4].

Chemoprophylaxis

Risk of secondary disease: Secondary disease, defined as illness within 1-60 days following contact with a child who has Hib disease, accounts for less than 5% of all invasive Hib disease. However, six studies of household contacts of Hib patients found a secondary attack rate of 0.3% in the month following disease onset in the index patient, which is about 600-fold higher than the age-adjusted risk in the general population. Among these studies, the attack rate among household contacts varied markedly with age; 4% for children under 2 years of age; 2% for children 2-3 years of age; 0.1%

for children 4-5 years of age; and 0% for those over 6 years of age. Among these household contacts; 64% of secondary cases occurred within the first week (excluding the first 24 hours) of disease onset in the index patient; 20%, during the second week; and 16%, during the third and fourth weeks.

The risk of secondary disease among children who were exposed to a primary case in day-care and who did not receive rifampicin prophylaxis has been examined in four studies. A national collaborative study in the United States that calculated secondary attack rates for household and day-care classroom contacts found that one (1%) of 91 children under 4 years of age in day-care acquired disease in the month following the index patient, compared with three of 125 household contacts under 4 years of age. A multicentre study in Seattle-King County, Washington; Oklahoma; and Atlanta, Georgia, found that the risk of secondary Hib disease among day-care classroom contacts was age-dependent; 10 (3%) cases occurred among the 376 contacts 0-23 months old, whereas none of the 379 classroom contacts older than 23 months of age acquired secondary disease. No cases occurred among children who attended day-care for fewer than 25 hours per week. In this study, classroom contacts were defined as children who spent more than half their day-care time in the same classroom as a child with primary Hib disease in the week before disease onset of the primary case. The over-all risk for classroom contacts was 0.7% (10/1,388), 20 times higher than the risk for other children in the centre (0.04% [2/5,639]). Thirty-three percent of the secondary cases occurred within 3 weeks of onset of the index case; 13%, between days 21 and 40; and 53%, between days 41 and 60. Meningitis and other systemic Hib infections were equally likely to result in secondary cases.

Two prospective studies have examined the risk of subsequent Hib disease in day-care facilities. In Dallas County, Texas, follow-up for 60 days of classroom contacts revealed no cases of secondary disease in 361 children under 2 years old, and a secondary attack rate of 0.5% (1/213) in those 2-3 years of age. Other cases of Hib disease occurred but could not be classified as secondary cases because these children enrolled in the day-care facility after the index patient became ill. Since it is known that rates of asymptomatic transmission are elevated in day-care classrooms with children with Hib disease, some of these cases may have been associated with the index case.

A similar surveillance study was conducted in Minnesota. No cases of secondary Hib disease were found among 370 day-care contacts under 2 years of age; 263 (71%) were classroom contacts. These were defined as children who spent more than 8 hours in the same classroom as the primary case in the week before the patient with primary disease became ill. Similarly, secondary cases were not seen in 716 children 2-3 years of age, of whom 421 (59%) were classroom contacts.

The disparities in the risk of day-care-associated secondary Hib disease in Minnesota; Dallas County, Texas; and the two multicentre studies remain unexplained. Possible reasons include differences among the several study areas in day-care characteristics, such as classroom size and age distribution of children, which might affect intensity and duration of contact. There may be further unrecognized differences in epidemiologic factors or invasiveness of prevalent Hib strains.

Efficacy of rifampicin prophylaxis: Most children at risk of secondary disease are too young to respond to the Hib polysaccharide vaccine. Therefore, the main preventive measure presently available is rifampicin administration. Currently available data from several studies indicate rifampicin in a dosage of 20 mg/kg per dose once daily (maximum daily dose 600 mg) for 4 days eradicated Hib carriage in 95% or more of contacts of primary cases, including children in day-care facilities. In a randomized placebo controlled trial, rifampicin in the currently recommended dosage administered to all household and day-care classroom contacts, including adults, significantly decreased secondary Hib disease among household and day-care contacts (none of 303 rifampicin-treated contacts under 4 years of age had secondary disease, compared with four of 216 placebo-treated contacts under 4 years of age [p = 0.03]) (2); the number of cases was insufficient to evaluate efficacy in the household or day-care setting alone. However, the collaborative study of day-care centres cited above found that among classroom contacts of Hib patients, children aged 0-23 months who received rifampicin prophylaxis were significantly less likely to develop secondary disease than children who did not take rifampicin (none of 232, compared with 10 [3%] of 376 [p 6 0.02]). Secondary disease did not develop in day-care classes in which over 75% of the class received rifampicin. However, rifampicin prophylaxis is unlikely to be 100% effective, and a day-care centre in which rifampicin prophylaxis failed to prevent subsequent disease has been reported.

Implementation of chemoprophylaxis: Rifampicin is available in 150 mg and 300 mg capsules. For those unable to swallow capsules, rifampicin may be mixed with several teaspoons of applesauce immediately before administration, resulting in acceptable serum and salivary levels. In Australia, rifampicin is also available as a raspberry-flavoured syrup (rifampicin 100 mg/5 mL). Side effects of rifampicin in the recommended dose include nausea, vomiting, diarrhoea, headache or dizziness, which occurred among 20% of those taking rifampicin and 11% of placebo recipients. No serious reactions occurred. Those taking rifampicin (including parents and day-care staff) should be informed that orange discoloration of urine, discoloration of soft contact lenses and decreased effectiveness of oral contraceptives can occur.

In implementing chemoprophylaxis in day-care centres, it is important to ensure that all classroom contacts receive rifampicin during the same period. Some local and state health departments have facilitated the timely implementation of chemoprophylaxis by coordinating rifampicin administration following consultation with private physicians or by providing information to parents of day-care contacts.

Vaccine

Effect of Haemophilus b polysaccharide vaccine on nasopharyngeal carriage: Limited data are available on the effect of the Haemophilus b polysaccharide vaccine on nasopharyngeal carriage of the organism. By analogy to carriage studies after serogroups A and C meningococcal polysaccharide vaccination, some reduction in acquisition of carriage may occur shortly after immunization, but no long-term effect has been noted.

Use of Haemophilus b polysaccharide vaccine in children with preceding Hib disease: Studies have shown that the development of anticapsular antibodies following invasive Hib disease is largely age-dependent. A study of acute and convalescent sera from 125 patients with meningitis, septicaemia, or epiglottitis due to Hib determined that, among those who acquired disease when they were younger than 18 months, 41 (85%) of 48 failed to develop an adequate antibody response, in contrast to 18 (23%) of 77 of those older than 18 months. Cases have been reported in which children who do not mount an antibody response after an invasive episode of Hib have developed a second systemic infection with the organism.

Recommendations - United States

The primary strategy for preventing Hib disease in the United States is immunization. Children are vaccinated at 24 months of age. Those at high risk for Hib disease, including children attending day-care, may be given the vaccine at 18 months of age.

Chemoprophylaxis: Although unexplained disparities in available data prevent a precise estimate of the magnitude of risk among day-care contacts, it is likely that the increased risk of disease observed among young household contacts is also present among day-care classroom contacts under 2 years of age. Since rifampicin prophylaxis is effective in preventing subsequent cases in this high-risk group, the ACIP recommends that:

1. Contacts of all ages who develop symptoms suggestive of invasive Hib disease, such as fever or headache, be evaluated promptly by a physician.
2. In any household in which a case of invasive Hib disease has occurred and in which another child under 4 years of age resides, all members of the household, including adults, should receive rifampicin according to the following regimen: rifampicin in a dosage of 20 mg/kg per dose once daily (maximal daily dose 600 mg) for 4 days; the dose for neonates (under 1 month of age) is 10 mg/kg once daily for 4 days.
3. In day-care classrooms in which a case of Hib disease has occurred and in which another child under 2 years of age has been exposed, all parents should be notified (preferably in writing) regarding the occurrence of the case and the possibility of increased risk to their children. They should be informed about the symptoms and the need for prompt medical evaluation if symptoms occur. They should also be notified of the availability of rifampicin prophylaxis. Although the data on which to base recommendations are not optimal, and some authorities disagree, the consensus of the ACIP, is as follows: In a day-care classroom in which a case of systemic Hib disease has occurred, and in which one or more children under 2 years old have been exposed, strong consideration should be given to administering rifampicin prophylaxis to all children and staff in the classroom, regardless of age.
4. Rifampicin should not be used in pregnant women, as its effect on the foetus has not been established, and it is teratogenic in laboratory animals.
5. Chemoprophylaxis should be instituted as rapidly as possible. If more than 14 days have passed since the last

contact with the index patient, the benefit of chemoprophylaxis is likely to be decreased.

6. All children convalescing from systemic Hib disease who are anticipated to resume close contact with other young children, at home or in day-care, should receive rifampicin immediately after completing treatment for their illness. Therapy for systemic disease does not reliably eradicate respiratory carriage of Hib, and some physicians may wish to give rifampicin to all index patients.
7. In day-care classrooms in which children are to receive chemoprophylaxis, children who have received the Haemophilus b polysaccharide vaccine should also receive rifampicin. Although these children are felt to be at decreased risk for disease, the vaccine probably does not affect carriage of the organism, which they may pass on to susceptile classmates.
8. Children who have had invasive Hib disease when they were under 24 months of age should still receive the vaccine according to previous recommendations, since most children under 24 months of age fail to mount an immune response to the clinical disease.
9. Satisfactory response to the vaccine is not consistent among children 18-23 months of age, and most authorities believe that these children should be revaccinated. Although data on the precise timing of this second dose are not currently available, it would be reasonable to reimmunize 2-12 months after the initial dose but not before 24 months of age. Previous immunization does not change the immune response or adverse reaction to a subsequent dose of the vaccine.

REFERENCES

(1) CDI (1986) 5

(2) CDI (1985) 10

(3) MMWR (1985) 34:201-5

(4) MMWR (1986) 35:11:170-180

WATERBORNE SALMONELLA TYPHIMURIUM - A FAMILY OUTBREAK OF ENTERITIS.

(Contributed by J. Martin, Rural Water Commission of Victoria;
J. Taplin, Microbiological Diagnostic Unit, Melbourne University;
P. Bond, Shire of Bright;
and B. Oliver, Health Department of Victoria.)

In early October 1985, all 9 children, aged between 7 months and 14 years, of a family living on a farm in North Eastern Victoria developed symptoms of gastroenteritis over a period of 5 days. Several of the children were moderately sick for a few days and then became acutely ill. Eight of the children were examined by a local physician, and six of these required hospitalisation, including three who were later referred to a base hospital.

Five faecal cultures were examined, yielding one isolate of Salmonella typhimurium phage type 4 and four isolates of S.typhimurium phage type RDNC. (ie the cultures reacted with the typing phages but did not conform to a known phage type).

Both phage types were isolated from the family water supply on two sampling dates, and the creek from which water was drawn was found to have faecal markers which included Escherichia coli (56 organisms per 100 mL) and S.typhimurium phage type RDNC.

The household water tank was uncovered and accessible to ducks and other birds and the raw water was not treated. Re-examination of the creek water at a later date indicated faecal contamination:

- at 500 metres upstream to be significant, 22 E.coli organisms per 100 mL, which was indistinguishable from the contamination observed at the pump site, of 28 E.coli organisms per 100 mL.
- at 2000 metres upstream with 8 E.coli organisms per 100 mL.

However no Salmonella organisms were isolated from a 2.5 litre sample taken at each of the above sites. The creek water was clear macroscopically, except when in flood, and was accessible to native fauna and farm cattle including new cattle acquired the previous month by the sole upstream farm. Neither farm gave any history of sick or aborting cattle or of stock being hand fed.

The farmhouse septic tank effluent line and a storm water drain finished near each other in an area of muddy ground. However a rough pathway leading 200 metres downhill from the wet ground to the pump site did not appear water scoured and was therefore not considered to contribute significantly to creek pollution.

The farm is situated on a dead end road, isolated from other dwellings. No other infection with the same organisms has been reported in the district. The only recent outside contact was a visit by the grandparents to the nearby city shortly before the incident. None of the grandparents became ill.

The children in this family consumed large amounts of raw tap water, except for the youngest who was the last to be affected. The adults drank water boiled as tea or coffee, and would be less exposed to waterborne infection. Since all the children were affected over a relatively short time period and no other suggestive dietary history was apparent following enquiry, a central origin of infection, rather than transmission by hands or fomites, has been postulated. The untreated creek water supply has been implicated as the origin of this outbreak.

In this instance, as many aspects of water hygiene had not been observed in the supply and storage of drinking waters, it was not possible to identify retrospectively the original source of the contamination. However the known presence of S.typhimurium in cattle, the acquisition of new cattle in the region in the previous month, the subsequent evidence of faecal pollution of the creek upstream of the farm and the laboratory isolation of S.typhimurium phage type RDNC from the creek water soon after the outbreak, all contribute to the hypothesis that the bought cattle were transient carriers of S.typhimurium and were responsible for the contamination of the creek water supply.

The above incident serves as a reminder that macroscopically clear creek water may not be microbiologically safe. Consumers of water from such a source should exercise caution.

MEASLES IN BRITISH COLUMBIA
(Based on Disease Surveillance (1986) 7:3:35-50)

Since 1984 there has been significant measles activity in certain parts of British Columbia, Canada. In the pre-vaccine era there were measles epidemics every 2-3 years on a continuing basis, and peaks in the incidence of measles deaths accompanied peaks in the reporting of measles. In October 1969 the B.C. Ministry of Health introduced a live attenuated measles vaccine program and by 1981 there had been a marked reduction in reported measles cases.

In 1984 a total of 1094 (38/100,000 population) cases of measles were reported in B.C; activity commenced in spring and had peaked by summer. In 1985 a total of 1748 (60/100,000 population) were notified. As previously there was a spring-summer peak, coinciding with an epidemic of rubella. A shift in the age distribution of measles notifications has been noted so that by 1985, 38% of measles occurred in 10-14 year olds and 29% in 15-19 year olds.

The components of an effective measles eradication campaign are: (i) achievement and maintenance of high immunisation levels, (ii) school entry screening, (iii) effective surveillance and (iv) aggressive response to cases.

By 1985, 96% of B.C. Grade One students were immunized against measles; but no accurate data are currently available on the immunisation status of high school students. It is estimated that perhaps 15-20% of such students lack evidence of measles immunity. Details of 394 cases of measles reported from two B.C. health jurisdictions so far in 1986 were analysed in respect of receipt of previous measles vaccine. Overall 29.2% of these cases had never received measles vaccine.

Measles vaccine protects 90-98% of persons who receive it. There is no strong evidence that the 2-10% of vaccinated persons who remain susceptible can lead to sustained transmission of measles[1]. Impotent vaccine and or inadequate - vaccination technique and inaccurate school immunisation records can explain the majority of cases which occur despite an intensive outbreak control program[2][3]. Close attention to cold chain and school immunisation procedures is crucial.

MEASLES IN WESTERN AUSTRALIA
(Based on VACCINE: Preventable Disease Surveillance Bulletin (February 1986) 3; published by the Epidemiology Branch, Health Department of Western Australia).

In January 1985, the Health Department of Western Australia formed a Sentinel Schools Surveillance Working Group to establish a five year program to monitor the immunisation status and vaccine - preventable disease (VPD) histories of year one primary school children in Western Australia.

The first annual survey of the program took place in October 1985, and was based on a cluster sample of 1,072 children attending year one classes at 59 primary schools throughout the State. The information was collected by local community health nurses and forwarded to central office for processing.

Measles immunisation was reported in 77% (95% CI 74-79) and mumps vaccine, usually as combined measles/mumps, was given to 28%. Histories of measles and mumps occurred in 24% and 19% of the sample respectively. Most of the children had received their measles immunisation in 1980, prior to the availability of the combined vaccine. A past history of measles was reported in 49% of children who were not immunised and in 16% of those who were. These results do not take account of the reduced severity of measles in immunised children; and are possibly also affected by overdiagnosis of measles in both groups. Immunisation coverage tended to be lower in Perth than elsewhere in the State.

In 1984 measles was responsible for 408 short-stay hospital discharges and 1283 hospital bed days in Western Australia.

CDI Comment

The Federal Minister for Health has foreshadowed a National Bicentennial campaign to eradicate measles.

Measles is often a severe disease, frequently complicated by otitis media, bronchitis and pneumonia. Measles encephalitis occurs in approximately 1 in 1,000 reported cases. Subacute sclerosing panencephalitis (SSPE) with progressive dementia, paralysis and death occurs in approximately 1 in 15,000 cases.

The CDI welcomes articles on measles immunisation, school immunisation procedures and measles epidemic control.

References

(1) Paediatrics (2985) 76: 524-532
(2) Am J. Epid. (1985) 122: 208-217
(3) MMWR (1986) 35: 99-100, 105-107.

HEPATITIS B ASSOCIATED WITH ACUPUNCTURE

A 1980 outbreak of 6 cases of hepatitis B in Florida which was associated with the use of non-sterile acupuncture needles in a chiropractic clinic, was reported recently.(1)

In the period February to May 1980 a state health unit was notified of 4 cases of hepatitis B among persons who had recently received acupuncture at a local chiropractic clinic. A survey was conducted of other persons who had attended the clinic between January 1979 and June 1980, and two additional cases of hepatitis B were identified.

Laboratory findings in all six hepatitis patients (median age 58 years) including markedly elevated serum aspartate aminotransferase (AST) levels during the acute phase, and during recovery, each had antibody to either HBsAg or to hepatitis B core antigen. All patients recovered from their acute illness, although 3 were hospitalised. Five of the patients were female.

During the six months before onset, no patient had any known potential exposures to hepatitis B virus except acupuncture. From October 1978 to the end of April 1980, 103 of 511 persons attending the clinic had received acupuncture. The reported incidence of hepatitis B among those who had had acupuncture (6 of 103) was significantly greater than among those who had not (0 of 408) ($P < .0001$, Fisher's exact test).

The outbreak of acute illness occurred in two clusters of 3 patients each in the period from 14 February 1980 to 23 May,1980. The most likely exposure periods were 27 to 28 November, 1979 and 19 to 20 February, 1980, respectively. Apparent incubation periods ranged from 61 to 95 days.

Two persons who had had acupuncture during both exposure periods remained asymptomatic. Two others who had had acupuncture during the first exposure period could not be located. When serum specimens from the former two were tested, only one indicated prior exposure to hepatitis B virus, being positive for antibody to hepatitis B core antigen. Sera from the two chiropractic practitioners were negative for hepatitis B surface antigen. It appears that serum from the receptionist was not tested. The duties of the receptionist were not stated.

A single acupuncture session involved inserting 5 to 15 needles for up to 20 minutes. Acupuncture needles were disinfected by overnight immersion in a 1:750 solution of benzalkonium chloride during the periods January to February 1979 and October 1979 to 22 February 1980 (which includes both exposure periods). During all other time periods either disposable or autoclaved acupuncture needles were used. Blood specimens for laboratory tests were routinely obtained using disposable needles and syringes. No additional cases of hepatitis B associated with acupuncture were reported during 1980.

CDI Editorial comment

The source of hepatitis B virus for the first cluster of cases is unknown. It appears, however, not to have been either of the two chiropractic practitioners. The sequence of acupuncture treatments during the second exposure period suggests that one of the patients in the first cluster may have been the source for the second cluster, and that overnight immersion of acupuncture needles in the benzalkonium chloride solution did not completely destroy infectious hepatitis B virus.

An additional possibility is that the receptionist, whose clinical and serological status is unknown, was the source of one or both clusters of hepatitis B infection. The receptionist's duties were not stated and it is therefore not possible to determine whether this person could have had contact with acupuncture needles.

Hepatitis B virus has been shown to be stable in dry form and to remain infectious for chimpanzees for at least one week[2]. This outbreak demonstrates that contaminated acupuncture needles can be a source of hepatitis B infection in man. Instruments that will enter sterile tissue or sterile body cavities should be sterile. Solutions of low-level disinfectants such as benzalkonium chloride and other quaternary ammonium compounds should never be used when the intent is sterilization[3]. (See also the note in this Bulletin on AIDS and acupuncture).

Autoclaving after thorough physical cleaning is the simplest and most effective method of sterilization of heat-stable materials. A suitable alternative would be the use of sterile, disposable equipment.

REFERENCES:

1. J. Fam Pract (1986) 22; 155-58

2. Lancet (1981) i:550-51

3. JAMA (1976) 236:2415-17

AIDS AND ACUPUNCTURE

An accompanying article in this Bulletin highlights the risk of transmission of hepatitis B from person to person through the use of non-sterile acupuncture needles.

It is possible that the AIDS virus (HTLV-III) can also be transmitted in this way. The Department of Health, through the AIDS Task Force has published procedures which should be followed to prevent the transmission of HTLV-III during acupuncture treatment.

A copy of these procedures[1] may be obtained by contacting -

The Secretary
AIDS Task Force
PO Box 100
WODEN ACT 2606

Phone (062) 89 7767

REFERENCE:

1. AIDS Task Force Bulletin 9/85

RECOMMENDATIONS FOR PREVENTING TRANSMISSION OF INFECTION WITH HTLV-III DURING INVASIVE PROCEDURES
(based on MMWR Vol 35/No 14, 11 April 1986)

BACKGROUND

In CDI 85/24, the document "Recommendations for Preventing Transmission of Infection with HTLV-III in the Workplace" gave particular emphasis to health-care settings and indicated that formulation of further specific recommendations for preventing HTLV-III transmission applicable to health-care workers (HCWs) who perform or assist in invasive procedures was under consideration.

These recommendations, issued for HCWs who have contact with tissues or mucous membranes while performing or assisting in operative, obstetric or dental invasive procedures, have now been approved by all members of a CDC consultative committee including a panel of 10 additional physicians with expertise in the epidemiology of HTLV-III infection and other infectious diseases.

In this document, the following has been defined:

. an operative procedure is defined as surgical entry into tissues, cavities, or organs or repair of major traumatic injuries in an operating or delivery room, emergency department, or outpatient setting, including both physicians' and dentists' offices.

- an obstetric procedure is defined as a vaginal or caesarean delivery or other invasive obstetric procedure where bleeding may occur.

- a dental procedure is defined as the manipulation, cutting or removal of any oral or perioral tissues, including tooth structure, where bleeding occurs or the potential for bleeding exists.

RECOMMENDATIONS

There have been no reports of HTLV-III transmission from an HCW to a patient or from a patient to an HCW during operative, obstetric, or dental invasive procedures. Nevertheless, special emphasis should be placed on the following precautions to prevent transmission of bloodborne agents between all patients and all HCWs who perform or assist in invasive procedures.

1. All HCWs who perform or assist in operative, obstetric, dental invasive procedures must be educated regarding the epidemiology, modes of transmission, and prevention of HTLV-III infection and the need for routine use of appropriate barrier precautions during procedures and when handling struments contaminated with blood after procedures.

2. All HCWs who perform or assist in invasive procedures must wear gloves when touching mucous membranes or nonintact skin of all patients and use other appropriate barrier precautions when indicated (e.g. masks, eye coverings, and gowns, if aerosolisation or splashes are likely to occur). In the dental setting, as in the operative and obstetric setting, gloves must be worn for touching all mucous membranes and changed between all patient contacts. If a glove is torn or a needlestick or other injury occurs, the glove must be changed as promptly as safety permits and the needle or instrument removed from the sterile field.

3. All HCWs who perform or assist in vaginal or caesarean deliveries must use appropriate barrier precautions (e.g. gloves and gowns) when handling the placenta or the infant until blood and amniotic fluid have been removed from the infant's skin. Recommendations for assisting in the prevention of perinatal transmission of HTLV-III have been published[2].

4. All HCWs who perform or assist in invasive procedures must use extraordinary care to prevent injuries to hands caused by needles, scalpels, and other sharp instruments or devices during procedures; when cleaning used instruments; during disposal of used needles; and when handling sharp instruments following procedures. After use, disposable syringes and needles, scalpel blades, and other sharp items must be placed in puncture-resistant containers for disposal. To prevent needlestick injuries, needles should not be recapped, purposefully bent or broken; removed from disposable syringes; or otherwise manipulated by hand. No data are currently available from controlled studies examining the effect, if any, of the use of needle-cutting devices on the incidence of needlestick injuries.

5. If an incident occurs during an invasive procedure that results in exposure of a patient to the blood of an HCW, the patient should be informed of the incident, and previous recommendations for management of such exposures should be followed[1].

6. No HCW who has exudative lesions or weeping dermatitis should perform or assist in invasive procedures or other direct patient-care activities or handle equipment used for patient care.

7. All HCWs with evidence of any illness that may compromise their ability to adequately and safely perform invasive procedures should be evaluated medically to determine whether they are physically and mentally competent to perform invasive procedures.

8. Routine serologic testing for evidence of HTLV-III infection is not necessary for HCWs who perform or assist in invasive procedures or for patients undergoing invasive procedures, since the risk of transmission in this setting is so low. Results of such routine testing would not practically supplement the precautions recommended above in further reducing the negligible risk of transmission during operative, obstetric, or dental invasive procedures.

Previous recommendations should be consulted for[1,3,4].

1. preventing transmission of HTLV-III infection from HCWs to patients and patients to HCWs in health-care settings other than those described in this document;

2. preventing transmission from patient to patient;

3. sterilising, disinfecting, housekeeping and disposing of waste;

4. managing parenteral and mucous-membrane exposures of HCWs and patients.

Previously recommended precautions are also applicable to HCWs performing or assisting in invasive procedures.

REFERENCES

1. MMWR (1985) 34:682-6, 691-5
2. MMWR (1985) 34:721-6, 731-2
3. MMWR (1982) 31:577-80
4. MMWR (1983) 32:450-1

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 14/4/86 - 27/4/86 BULLETIN NUMBER 86/9
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			6		4	1		1	12
0101 ADENOVIRUS TYPE 1				1	1				2
0102 ADENOVIRUS TYPE 2					4	1			5
0103 ADENOVIRUS TYPE 3					1	1			2
0104 ADENOVIRUS TYPE 4	1				1				2
0105 ADENOVIRUS TYPE 5					5	1		1	7
0106 ADENOVIRUS TYPE 6					3	1			4
0107 ADENOVIRUS TYPE 7					1				1
0108 ADENOVIRUS TYPE 8	2			2					4
0128 ADENOVIRUS TYPE 28				1					1
0131 ADENOVIRUS TYPE 31						1			1
0199 ADENOVIRUS TYPING PENDING		1	1		3				5
0201 INFLUENZA A VIRUS			2						2
0203 INFLUENZA B VIRUS	1							1	2
0301 PARAINFLUENZA VIRUS TYPE 1				1	8				9
0302 PARAINFLUENZA VIRUS TYPE 2					7	2		1	10
0303 PARAINFLUENZA VIRUS TYPE 3				2	3				5
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	3	2	4		1			1	11
0500 RHINOVIRUS (ALL TYPES)				1	14	4			19
0600 MYCOPLASMA PNEUMONIAE	3	1	5					1	10
0700 ORNITHOSIS-PSITTACOSIS						1			1
0807 COXSACKIEVIRUS A7								1	1
0816 COXSACKIEVIRUS A16		1							1
0904 COXSACKIEVIRUS B4								1	1
1003 ECHOVIRUS TYPE 3	1								1
1007 ECHOVIRUS TYPE 7				1					1
1011 ECHOVIRUS TYPE 11	1	1						1	3
1020 ECHOVIRUS TYPE 20	2								2
1021 ECHOVIRUS TYPE 21				1					1
1022 ECHOVIRUS TYPE 22				1					1
1023 ECHOVIRUS TYPE 23		1							1
1100 POLIOVIRUS NOT TYPED			1						1
1101 POLIOVIRUS TYPE 1						1		1	2
1102 POLIOVIRUS TYPE 2				1					1
1200 MUMPS VIRUS	1							1	2
1300 HERPES VIRUS GROUP-NOT TYPED	8			5				1	14
1301 HERPES SIMPLEX VIRUS NOT-TYPED								2	2
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	5	2	1					6	14
1303 VARICELLA-ZOSTER VIRUS	2				1	1			4
1306 HERPES SIMPLEX TYPE 1	6			39		23		17	85
1307 HERPES SIMPLEX TYPE 2	22			71		17		34	144
1399 HERPES VIRUS TYPING PENDING					6				6
1401 COXIELLA BURNETI	4		1			2		1	8
1502 PICORNA VIRUS-NOT TYPED			6					4	10
1521 MEASLES VIRUS		1							1
1522 RUBELLA VIRUS			4	2					6
1532 HEPATITIS B ANTIGEN	36		7	25	1	32		20	121
1535 HEPATITIS A ANTIBODY	4	1	1	10	1	30		36	83
1541 CHLAMYDIA A - C TRACHOMATIS	17		1			37		49	104
1556 CMV - CYTOMEGALOVIRUS	1	2		30	11	8		9	61
1563 CORONAVIRUS								1	1
1564 ROTAVIRUS	8		5	2	3	36		4	58
1571 ENTEROVIRUS TYPE 71 (BRCR)				3					3
1599 ENTEROVIRUS TYPING PENDING		2	7		5				14
9992 ROSS RIVER VIRUS								10	10
9994 SMALL VIRUS (LIKE) PARTICLE		3							3
Total	128	18	52	199	84	200		205	886

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 14/4/86 - 27/4/86
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		1									
0102	ADENOVIRUS TYPE 2		4									
0103	ADENOVIRUS TYPE 3	1	1									
0104	ADENOVIRUS TYPE 4		2									
0105	ADENOVIRUS TYPE 5		2					3				
0106	ADENOVIRUS TYPE 6		3					1				
0107	ADENOVIRUS TYPE 7							1				
0131	ADENOVIRUS TYPE 31							1				
0201	INFLUENZA A VIRUS		1									
0203	INFLUENZA B VIRUS											1
0301	PARAINFLUENZA VIRUS TYPE 1		9									
0302	PARAINFLUENZA VIRUS TYPE 2	1	9									
0303	PARAINFLUENZA VIRUS TYPE 3		4									1
0400	RESPIRATORY SYNCYTIAL VIRUS (RS)	1	8								1	
0500	RHINOVIRUS (ALL TYPES)		9		2							
0600	MYCOPLASMA PNEUMONIAE	2	3	1		1						
0807	COXSACKIEVIRUS A7		1									1
0816	COXSACKIEVIRUS A16											1
1003	ECHOVIRUS TYPE 3							1				
1007	ECHOVIRUS TYPE 7				1							
1011	ECHOVIRUS TYPE 11	2			1							
1020	ECHOVIRUS TYPE 20				1							
1021	ECHOVIRUS TYPE 21				1							
1022	ECHOVIRUS TYPE 22		1									
1023	ECHOVIRUS TYPE 23				1							
1101	POLIOVIRUS TYPE 1		1					2				
1102	POLIOVIRUS TYPE 2		1									
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	6	1						1			1
1303	VARICELLA-ZOSTER VIRUS		1									2
1306	HERPES SIMPLEX TYPE 1	1	4	1							2	36
1307	HERPES SIMPLEX TYPE 2	3										36
1401	COXIELLA BURNETI	2							2	1		
1502	PICORNA VIRUS-NOT TYPED				1			7				
1521	MEASLES VIRUS						1					
1522	RUBELLA VIRUS		1		2							2
1532	HEPATITIS B ANTIGEN	37							58			
1535	HEPATITIS A ANTIBODY	15						1	52			
1541	CHLAMYDIA A - C.TRACHOMATIS							1				
1556	CMV - CYTOMEGALOVIRUS	6	17		1				3		7	2
1564	ROTAVIRUS							58				
1571	ENTEROVIRUS TYPE 71 (BRCR)				1							2
9992	ROSS RIVER VIRUS	1										3
9994	SMALL VIRUS (LIKE) PARTICLE				1			2				
Total		78	84	2	13	1	1	78	116	1	10	88

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 14/4/86 - 27/4/86
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		
0102 ADENOVIRUS TYPE 2.....										1
0105 ADENOVIRUS TYPE 5.....						1		1		
0108 ADENOVIRUS TYPE 8.....	4									
0128 ADENOVIRUS TYPE 28.....									1	
0201 INFLUENZA A VIRUS.....								1		
0203 INFLUENZA B VIRUS.....									1	
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....								1		
0500 RHINOVIRUS (ALL TYPES).....								2		
0600 MYCOPLASMA PNEUMONIAE.....								3		
0700 ORNITHOSIS-PSITTACOSIS.....								1		
0904 COXSACKIEVIRUS B4.....										1
1020 ECHOVIRUS TYPE 20.....									1	
1200 MUMPS VIRUS.....				1				1		
1302 EPSTEIN-BARR VIRUS (EB VIRUS).				3	1			2	1	
1303 VARICELLA-ZOSTER VIRUS.....	1									
1306 HERPES SIMPLEX TYPE 1.....	3	34					1	3	1	
1307 HERPES SIMPLEX TYPE 2.....		112								
1401 COXIELLA BURNETI.....							1	1	1	
1502 PICORNA VIRUS-NOT TYPED.....					1					
1522 RUBELLA VIRUS.....									1	
1532 HEPATITIS B ANTIGEN.....									26	
1535 HEPATITIS A ANTIBODY.....								3	12	
1541 CHLAMYDIA A - C.TRACHOMATIS...	1	101							1	
1556 CMV - CYTOMEGALOVIRUS.....	1	2		2	2	2	3	2	13	
1563 CORONAVIRUS.....								1		
9992 ROSS RIVER VIRUS.....					8					
Total.....	10	249	4	3	11	3	5	23	59	2

IMPORTANT NOTICE Our ref 86/4465

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