

AUSTRALIA

Bulletin Number 78/18

Reporting Period 24 August 1978 -
6 September 1978.

Communicable

Diseases

Intelligence

Smallpox in Birmingham (CDR 25 August 1978, W.E.R. 8 September 1978)

The Communicable Disease Report of 25 August 1978 included a detailed account of the smallpox case in Birmingham referred to in Bulletin 78/17. The following information is taken from that report.

The patient, a 40 year old female developed symptoms on 11 August with the rash first appearing 2 days later. She had been vaccinated in 1966 and had not been abroad recently. Between 11 August and 21 August, she was cared for at home, after which she was moved to her parent's home where she remained until admitted to hospital on 24 August 1978. Smallpox was suggested and electron microscopy of vesicle fluid demonstrated brick-shaped particles suggestive of pox viruses. She was transferred to the Catherine De Barnes Smallpox Hospital later the same day, and the results of egg culture were positive for variola major on 27 August. The Australian Press has since reported that the patient died on 11 September 1978, apparently from renal involvement.

The patient worked as a medical photographer at Birmingham University Medical School in a room one floor above the Department of Medical Microbiology, which is one of the three laboratories in the United Kingdom identified by W.H.O. as still holding variola virus. It is understood that work on variola major virus was taking place in the smallpox laboratory at the Department during July 1978 but it is not known how the patient became infected. She stated that she had never been in the laboratory, and it is assumed that the virus was transmitted aurally, although no defect in the ventilation and air filtration systems has yet been identified to explain the incident.

The smallpox laboratory was closed on 25 August. Appropriate disinfection of home and hospital rooms occupied by the patient and vehicles in which she travelled was carried out. British health authorities are continuing to trace the anticipated 200 contacts of the patient, both close and otherwise. Where appropriate, vaccination, hyperimmune anti-vaccinal gammaglobulin and methisazone were being given, and active surveillance of contacts will continue until 16 days have elapsed since last contact with the patient or with a possibly infected area. No secondary cases have occurred.

Assuming that this is a case of laboratory-acquired infection and not due to person-to-person transmission originating from an endemic focus, no change in the schedule for global certification of smallpox eradication by the end of 1979 is foreseen (W.E.R. 1 September 1978). Travellers entering Australia from Birmingham currently require a valid smallpox vaccination certificate.

LEGIONNAIRES' DISEASE IN SYDNEY (Contributed by J. Harkness, St Vincent's Hospital, Sydney)

A 29 year old bulldozer driver from Penrith was admitted to a District Hospital on 22.4.78 with a two week history of a 'flu-like' illness characterised by fever, vomiting, diarrhoea, mucoid sputum with haemoptysis, dyspnoea and weight loss of one stone.

On examination he was febrile (T 38.9°C) and had evidence of right lower and middle lobe consolidation.

He was treated with ampicillin and later gentamicin, tetracycline and cloxacillin due to his deteriorating condition. All cultures taken were negative.

Coincidentally he developed acute renal failure and was transferred to St Vincent's Hospital on 29.4.78.

On admission to St Vincent's Hospital he was felt to have viral pneumonia, fluid overload and acute renal failure. Treatment was with high concentration O₂ administration, erythromycin to treat possible mycoplasma pneumonia, and dialysis.

Over the next 10 days 17 kgs of fluid were removed and the patients condition slowly improved. Normal urine output was restored after 14 days. Some residual consolidation persisted in the right middle lobe.

All cultures were negative and acute and convalescent sera to the usual respiratory pathogens were negative. Serum collected on 11.5.78 and 8.6.78 and sent to C.D.C. Atlanta were positive to 1:4096 and 1:8192 to Legionnaires' organism using the I.F.A. test.*

Legionnaires' organism is a gram negative bacillus, as yet unclassified. It has been cultured from the lungs or pleural fluid of 15 patients out of a total of approximately 600 serologically proven cases in the U.S.A. and U.K. The organism was initially cultured in guinea pigs and passed to yolk sacs. It has been subsequently cultured on Mueller Hinton agar containing 1% Haemoglobin and 2% Isovitalex.

MYOSITIS ASSOCIATED WITH INFLUENZA (Contributed by I. Jack, Royal Children's Hospital, Melbourne).

When Dr Bryce Larke of Edmonton visited Melbourne earlier this year he described some patients with myositis in association with influenza infection. His study has not appeared in press but there are two others:

Middleton (1970) Lancet 2,533
Dietzman, (1976) Paediatrics 57,255

* Further details of this case appear in the MJA, 9.9.78

Alerted to this association we sought an influenza diagnosis in two patients referred from the Neurology Department of this Hospital. Both had severe calf muscle myositis but neither was examined by biopsy or myography. One yielded an influenza virus isolate, and was positive by immunofluorescence of exfoliated cells. The second was not tested by immunofluorescence, and yielded an untyped enterovirus in faeces. Subsequently, we learned that the Outpatients' Department had seen other patients with a less severe form of myositis but these had not been referred for diagnosis. Two further patients have been seen with milder muscle soreness in association with influenza virus.

Although myalgia is not uncommon in association with influenza, myositis is not often reported. The illness appears to be self limited and not to have features of the auto-immune disease of dermatomyositis.

Human Salmonellosis

The number of human salmonellosis cases reported for August remained below that for the preceding months. In all, 201 cases were reported, 36% being S. typhimurium and 43% involving infants less than 5 years of age.

The isolates included 4 S. typhi (15 yr m. in N.S.W. returned from Lebanon; 19 yr m. in N.S.W.; 2 yr m. in N.S.W. returned from Middle East, 70 yr f. in Vic who is a known carrier, and 12 yr f. in Perth) and 3 S. paratyphi A, all of which were apparently contracted in various parts of Asia.

Penicillinase Producing Neisseria Gonorrhoeae

Up to 30 June 1978, 69 isolates of penicillinase producing N. gonorrhoeae had been reported in Australia, 56 of these being from males and 13 from females. Since then, 2 further cases have been reported. One was in a male in Mt Gambier (S.A.) who acquired the infection in Bangkok, and the other involved a male in N.S.W. who acquired his infection from a bar girl in Singapore, one week prior to the onset of symptoms.

All cases of gonorrhoea in Australia with this strain have emanated either directly or indirectly from South East Asia, and the majority of infections have been contracted in either Bangkok or Manilla.

FURTHER REPORT ON STUDIES OF GASTROENTERITIS FROM OYSTERS
(Provided by the Staff of the I.C.P. & M.R. Sydney)

A further five cases have been reported to us and specimens submitted during the past two weeks.

FAECES:

Acute faecal specimens have been examined from 65 patients.

Number of patients showing viral particles by Electron Microscopy	=	54	(83%)
" " " " 22-25nm " " "	=	28	(43%)
" " " " 27-30nm " " "	=	24	(37%)
" " " " both 22-25 and 27-30nm. particles by EM	=	2	(3%)
" " " " no virus particles by EM	=	11	(17%)

CELL CULTURE:

Of those specimens that showed no growth on original cell culture, reinoculated ultracentrifuged pellets have shown no CPE in cell cultures.

SERA: Immune Electron Microscopy (IEM)

IEM has been performed between 4 sets of paired sera and their homologous stool extracts. Seroconversion to the 28nm particle was demonstrated in 2 sets of sera. Seroconversion to the 23nm particle could not be demonstrated. A further 8 sets of sera have yet to be tested.

Paired Sera to the Norwalk agent (obtained from the USA) and the Parramatta agent were tested by IEM to both the 23 and 28nm. particles. Seroconversion was demonstrated to the 28nm. particle with the Norwalk sera in 2 cases. Also, convalescent sera to the Ditchling (Cockle) virus were reacted with both types of particles but no aggregation was observed.

Specific Hepatitis A antisera have also shown no immune reactions.

Hepatitis A (previously 'infectious hepatitis')

From January to June this year, 1 400 cases of hepatitis A have been reported to the State Health Departments in Australia as part of the Notifiable Diseases Returns system. The true incidence of the disease however, is much higher; it has been estimated that in the United States, only 10% of cases are reported; while there is no corresponding estimate for Australia, this statement speaks for itself.

Although differentiation between hepatitis A, hepatitis B and hepatitis non A-non B on clinical grounds is very difficult, the diagnosis of hepatitis A infection rests almost exclusively with the medical practitioner. Since laboratory confirmation of this disease is generally unavailable, the diagnostic tests, which include immune electron microscopy, complement fixation, immune adherence haemagglutination and solid phase radioimmunoassay, are presently restricted to only a few laboratories around the world. For the period 1 January 1978 to 30 June 1978, only nine hepatitis A virus (HAV) laboratory detections and two positive anti-HAV antibody results were reported in the CDI. These laboratory identifications should however become progressively more available as hepatitis A antigen becomes less scarce.²

The hepatitis A virus, of which only one serotype has so far been defined, was first recognised in 1973 in the U.S. It is a small icosahedral virus of approximately 27 nm diameter, and although it has not been formally classified, recent biochemical characterisation has suggested that it may be a member of the enterovirus group (e.g. polio, Coxsackie and echo viruses)³. The virus has not been grown in cell or organ culture and at present the only source is specimens from experimentally infected chimpanzees.⁴

The incubation period for hepatitis A ranges from 2-6 weeks but is most commonly about 4 weeks. Peak virus shedding, and therefore maximum infectivity, occurs at the onset of symptoms, but virus can be detected in the faeces approximately 2 weeks before and 2 weeks after the onset. The illness is generally of short duration without sequelae and has a low mortality.⁵ Anicteric, asymptomatic disease occurs 3-4 times more frequently than icteric hepatitis.⁶ Since there is no evidence of a carrier state of HAV, it is speculated that inapparent, subclinical, sporadic cases serve as a reservoir for HAV infection.⁷

The faecal-oral route is the most common mode of spread of HAV, either from direct person-to-person transmission or through food and water. Many food-borne epidemics have been described, and these are frequently attributed to infected food handlers shedding virus during the incubation period of the disease. The ingestion of shellfish grown in polluted waters is associated with a high risk of acquiring hepatitis, with the mode of preparation of this food being important. (Frying appears more efficient in destroying the virus than

steaming in which the shells open and the contents are consumed before virucidal temperatures are attained)⁸. Because of the long incubation period, food or water-borne infections are difficult to trace to a source. Transmission by biological fluids such as urine, blood or droplets from the mouth or nose appears to occur only rarely, even though low levels of HAV are found in the blood and urine in the early stages of infection.⁹ Hepatitis A has a world wide distribution with infection occurring most commonly in childhood. The incidence of the disease varies from country to country depending on the level of hygiene observed and the different socio-economic groupings studied. Similarly, the relative contributions of hepatitis A, hepatitis B, and hepatitis non A-non B to the total incidence of viral hepatitis varies between communities.¹⁰

Control of hepatitis A depends on adequate personal hygiene and proper disposal of faeces. The single most important factor is undoubtedly handwashing prior to eating or handling food about to be eaten without further cooking. Intramuscular administration of normal human immunoglobulin (a 16% solution in a dose of 0.02-0.12 mg/kg body weight) may prevent or attenuate a clinical illness while not necessarily preventing infection.¹¹ Inapparent or subclinical hepatitis may result, but this is still particularly recommended in the control of outbreaks, especially in institutions. Bulletin 78/15 suggested a regime for the prophylaxis against hepatitis A which included the following schedule:

Patient Weight (kg)	Dose of immunoglobulin (ml)	
	Short term (up to 3 mths)	3-6 Months
Less than 25	0.5	1.0
25-50	1.0	2.5
More than 50	2.0	5.0

Administration is by intramuscular injection.

References

- 1, 7. Dienstag J.H. et al. Hepatitis A Virus Infection: New insights from seroepidemiologic studies. J. of Infectious Diseases. 137: 3 June 1978 p 328.
2. Locarnini S.A. and Gust I.D. Food and Hepatitis A. Food Technology in Australia. 30: 8 August 1978 p 295.
- 3,8,9. Gust I.D. Acute Viral hepatitis. Australian Family Physician Vol 7 1 May 1978 p 535.
- 4,5,11. Zuckerman A.J. The three types of human viral hepatitis. Bulletin of the World Health Organisation 56(1): -20 (1978)
6. Breen K.J. Whelan G and Smallwood R. Viral hepatitis in general hospitals: guidelines for control and prevention Med J. Aust Vol 2 1 July 1978 p 18.

PERIOD AUGUST 1978

SEROTYPE	TOTAL	NSW & ACT	VIC	QLD	SA	WA	TAS	NT	AGE					CUMULATIVE TOTAL	
									<1	1-5	6-15	16-60	>60		NOT STATED
S. adelaide	5					5						4		1	32
S. anatum	10		1	2		7			1	1	1	7			34
S. birkenhead	1			1								1			5
S. bovis-morbificans	11	2	4		1	4			2	3	1	4		1	61
S. bredeney	2			1	1					1	1				23
S. ferro	1	1												1	1
S. champaign	1					1			1						2
S. chester	2		1					1		1	1				43
S. derby	6	1				5				2		4			65
S. emmastad	2					2			1		1				4
S. enteritidis	2			1		1					1	1			13
S. give	1				1						1				12
S. haifa	1					1						1			1
S. ivana	1				1				1						37
S. heidelberg	1							1			1				1
S. infantis	1				1				1						23
S. java	14		13					1		1		11	1	1	15
S. kottbus	2						2			1				1	3
S. lansing	1							1				1			8
S. lexington	1		1								1				3
S. livingstone	1					1						1			1
S. london	5					5					1	4			8
S. muenchen	10	2		4		4			1	5		3		1	40

PERIOD AUGUST

SEROTYPE	TOTAL	NSW & ACT	VIC	QLD	SA	WA	TAS	NT	AGE					CUMUL. TOT	
									<1	1-5	6-15	16-60	>60		NOT STATED
S. newington	2					2						2			5
S. newport	7		1	1		5				4		2		1	30
S. orion	1					1				1					7
S. paratyphi A	3		2	1							1	2			3
S. potsdam	3					2		1	1	1				1	10
S. rubislaw	1					1				1					17
S. saint-paul	5		2			3			1	1	1	1		1	78
S. senftenberg	2					2				1		1			14
S. singapore	2					1		1		2					27
S. tennessee	1					1			1						13
S. thompson	2			1		1			1	1					3
S. typhi	5	3	1			1				1	2	1	1		26
S. typhimurium	74	13	19	10	11	20		1	16	24	5	11	7	11	550
S. virchow	6			6					2	3				1	34
S. waycross	1	1								1					4
S. wordsworth	4					4			1		1	2			32
	201	23	45	28	16	80	2	7	31	56	20	64	9	21	-

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 24-8-78 . 6-9-78 BULLETIN NUMBER . 78 | 18
 VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPME (NSW) / WVH (ACT)	FAHC (NSW)	PRH/ POW (NSW)	FAIR- FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
0100 ADENOVIRUS NOT TYPED.....	4		15	1	2		14		36
0101 ADENOVIRUS TYPE 1.....			1	2					3
0102 ADENOVIRUS TYPE 2.....				1	4			3	8
0103 ADENOVIRUS TYPE 3.....		1						1	2
0105 ADENOVIRUS TYPE 5.....				1	1			1	3
0107 ADENOVIRUS TYPE 7.....				2	1				3
0119 ADENOVIRUS TYPE 19.....						1			1
0127 ADENOVIRUS TYPE 27.....	2								2
0131 ADENOVIRUS TYPE 31.....						2			2
0199 ADENOVIRUS TYPING PENDING.....		1			8	1		1	11
0201 INFLUENZA A VIRUS.....	5		1				1		7
0203 INFLUENZA B VIRUS.....	10	1		20	19	1	5		56
0302 PARAINFLUENZA VIRUS TYPE 2.....				1	2	1	2		3
0303 PARAINFLUENZA VIRUS TYPE 3.....						1	6		10
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)...	2	2	4	2	17	24	4	18	73
0500 RHINOVIRUS (ALL TYPES).....				3		1	2	2	8
0600 MYCOPLASMA PNEUMONIAE.....	24	1	7	47	5	7	10	2	103
0700 ORNITHOSIS-PSITTACOSIS.....			2	2		1			5
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED.....							1		1
0816 COXSACKIEVIRUS A16.....	1							1	2
0901 COXSACKIEVIRUS B1.....						3		1	4
0903 COXSACKIEVIRUS B3.....						1			1
1007 ECHOVIRUS TYPE 7.....				1					1
1011 ECHOVIRUS TYPE 11.....						1		2	3
1013 ECHOVIRUS TYPE 13.....								1	1
1014 ECHOVIRUS TYPE 14.....						1			1
1017 ECHOVIRUS TYPE 17.....	1						1		2
1019 ECHOVIRUS TYPE 19.....			2						2
1021 ECHOVIRUS TYPE 21.....						3		1	4
1022 ECHOVIRUS TYPE 22.....						2	1		3
1025 ECHOVIRUS TYPE 25.....						1			1

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 24-8-78 . 6-9-78 BULLETIN NUMBER - 78/18
 VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES-CONTINUED

VIRUS OR VIRAL ANTIGEN	ICPMP (NSW) / WVH (ACT)	EAHC (NSW)	PHH/ POW (NSW)	FAIP- FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAE (OLD)	STATE LAS (WA)	Total
1030 ECHOVIRUS TYPE 30.....				1			2	4	7
1099 ECHOVIRUS TYPING PENDING.....				1					1
1102 POLIOVIRUS TYPE 2.....	1			1		1	1		4
1200 MUMPS VIRUS.....			1	8		1	9		19
1300 HERPES VIRUS GROUP-NOT TYPED.....						1			1
1301 HERPES SIMPLEX VIRUS-NOT TYPED.....	8		5	2	7		18	1	41
1302 EPSTEIN-BARR VIRUS (EB VIRUS).....						1			1
1303 VARICELLA-ZOSTER VIRUS.....	1		3	1			3		8
1306 HERPES SIMPLEX TYPE 1.....	9			12		7			28
1307 HERPES SIMPLEX TYPE 2.....	25			7		13			45
1399 HERPES VIRUS TYPING PENDING.....								19	19
1401 COXIELLA BURNETI.....	4			2			9		15
1521 MEASLES VIRUS.....	1		8	1	2	1	1		14
1522 RUBELLA VIRUS.....							1	1	2
1532 HEPATITIS B ANTIGEN.....	3		13	24		5	8	19	72
1533 HEPATITIS B ANTIBODY.....						10			10
1541 CHLAMYDIA A - TRIC TYPE.....	1					1		19	21
1556 CMV - CYTOMEGALOVIRUS.....	5	4		4	1	3	1	6	24
1562 CORONAVIRUS.....						1			1
1564 ROTAVIRUS.....	1	2	11	5		15	23		57
1599 ENTEROVIRUS TYPING PENDING.....						1			1
Total.....	108	12	73	152	69	113	123	103	753

Dengue - (Type 3) _____ 1 _____ 1 _____ 2
 Arbovirus Group B _____ 1 _____ _____ 1
 Ross River Virus _____ _____ 6 _____ 6
 Adenovirus _____ _____ 2 _____ 2
 Parvovirus-like _____ 3 _____ _____ 3

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 24-8-78 . 6-9-78 BULLETIN NUMBER . 78/18
VIRAL IDENTIFICATIONS CATEGORISED INTO SOURCE SPECIMENS

VIRUS OR VIRAL ANTIGEN	FA	BL	NA	CS	SK	EY	UR	BR	GE	OT	TOTAL
0100 ADENOVIRUS NOT TYPED.....	6	19	11								36
0101 ADENOVIRUS TYPE 1.....	1		2								3
0102 ADENOVIRUS TYPE 2.....	4		5								9
0103 ADENOVIRUS TYPE 3.....	1									1	2
0105 ADENOVIRUS TYPE 5.....			2				1				3
0107 ADENOVIRUS TYPE 7.....	1		2								3
0119 ADENOVIRUS TYPE 19.....						1					1
0127 ADENOVIRUS TYPE 27.....			1								1
0131 ADENOVIRUS TYPE 31.....	2										2
0199 ADENOVIRUS TYPING PENDING.....	3	1	8								12
C201 INFLUENZA A VIRUS.....		7									7
0203 INFLUENZA B VIRUS.....		13	42							1	56
0302 PARAINFLUENZA VIRUS TYPE 2.....		2	1								3
0303 PARAINFLUENZA VIRUS TYPE 3.....		2	8								10
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)....	4	6	66								76
0500 RHINOVIRUS (ALL TYPES).....			8								8
0600 MYCOPLASMA PNEUMONIAE.....		100									100
0700 ORNITHOSIS-PSITTACOSIS.....		5									5
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED.....			1								1
0816 COXSACKIEVIRUS A16.....	1		1								2
0901 COXSACKIEVIRUS B1.....	1		2		1						4
0903 COXSACKIEVIRUS B3.....	1										1
1007 ECHOVIRUS TYPE 7.....			1								1
1011 ECHOVIRUS TYPE 11.....	3										3
1013 ECHOVIRUS TYPE 13.....			1								1
1014 ECHOVIRUS TYPE 14.....					1						1
1017 ECHOVIRUS TYPE 17.....	1				1						2
1019 ECHOVIRUS TYPE 19.....	1						1				2
1021 ECHOVIRUS TYPE 21.....	2		1							1	4
1022 ECHOVIRUS TYPE 22.....	2				1						3
1025 ECHOVIRUS TYPE 25.....			1								1
1030 ECHOVIRUS TYPE 30.....	5		1		2						8

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REPORTING PERIOD - 24-8-78 . 6-9-78 BULLETIN NUMBER . 78/18
 VIRAL IDENTIFICATIONS CATEGORISED INTO SOURCE SPECIMENS-CONTINUED

VIRUS OR VIRAL ANTIGEN	FA	BL	NA	CS	SK	FY	UR	EP	GP	OT	TOTAL
1099 ECHOVIRUS TYPING PENDING.....	1										1
1102 POLIOVIRUS TYPE 2.....	2		1				1				4
1200 MUMPS VIRUS.....		16	1	2							19
1300 HERPES VIRUS GROUP-NOT TYPED.....					1						1
1301 HERPES SIMPLEX VIRUS-NOT TYPED.....		7	10		6	1			16	1	41
1302 EPSTEIN-BARR VIRUS (EB VIRUS).....		1									1
1303 VARICELLA-ZOSTER VIRUS.....		8									8
1306 HERPES SIMPLEX TYPE 1.....			11		7			1	7	2	28
1307 HERPES SIMPLEX TYPE 2.....					4				41	1	46
1399 HERPES VIRUS TYPING PENDING.....			1		9				8	1	19
1401 COXIELLA BURNETI.....		15									15
1521 MEASLES VIRUS.....		11	3								14
1522 RUBELLA VIRUS.....		2									2
1532 HEPATITIS B ANTIGEN.....	1	70									71
1533 HEPATITIS B ANTIBODY.....		10									10
1541 CHLAMYDIA A - TRIC TYPE.....						2			19		21
1556 CMV - CYTOMEGALOVIRUS.....		9	3				6		5	1	24
1562 CORONAVIRUS.....	1										1
1564 ROTAVIRUS.....	57										57
1599 ENTEROVIRUS TYPING PENDING.....	1										1
Total.....	102	304	195	8	27	4	9	1	96	9	755

Dengue - (Type 3) _____ 1 _____ 1
 Arbovirus Group B _____ 1 _____ 1
 Ross River Virus _____ 6 _____ 6
 Astrovirus _____ 3 _____ 3
 Parvovirus - like _____ 3 _____ 3