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Communicable Diseases Intelligence

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Contents:

This is the final issue of CDI for 1981, and includes the subject index for the year. The next issue will be published on 15 January 1982. The editorial staff takes this opportunity to extend seasonal greetings to all readers, with best wishes for the New Year.

VIRUS REPORTING SCHEME - A total of 517 reports were received this period, although figures from four laboratories were delayed in the mail.

- Arbovirus infections - The State Health Laboratory, Brisbane, confirmed the fourth indigenous dengue case from Thursday Island. The serum specimen was taken from the 46 year old pearl sheller on 29 October 1981. The laboratory also reported the presence of specific IgM against Sindbis virus in a 35 year old male from Griffith. Epidemic polyarthritis was diagnosed by the Institute of Medical and Veterinary Science in a 39 year old female from Waikerie, about 150 km north-east of Adelaide. The disease was last reported in the Riverland area in the late summer of 1980.

- A CF titre of 1/8 against measles virus was detected by the State Health Laboratory, Brisbane, in the CSF of a four year old girl with status epilepticus, akinetic and grand mal seizures.

- Less common reports received included echovirus type 27 from a seven month old child with pertussis, adenovirus type 8 from a seven month old boy with croup and adenovirus type 22 from a one month old boy with gastro-enteritis. The previous adenovirus type 22 isolation was by Fairfield Hospital in October 1978 (see CDI 78/21).

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6. NEJM (1981) 304 : 989.
7. Lancet (1977) 1 : 558.
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10. J. Med. Virol. (1978) 3 : 141.
11. Alter, H.J. et al., Non A non B hepatitis : A review and interim report of an on-going prospective study. In : Viral Hepatitis. Philadelphia : Franklin Institute Press. (1978) : 359.
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GONOCOCCAL SURVEILLANCE - AUSTRALIA (JULY - SEPTEMBER 1981)

(Contributed by the Australian Gonococcal Surveillance Program: Co-ordinator - J.W. Tapsall, Department of Microbiology, The Prince of Wales Hospital, Sydney).

A standard method for the determination of the minimal inhibitory concentration (MIC) of penicillin for strains of N. gonorrhoeae was recommended by the members of the Australian Gonococcal Surveillance Program in CDI 81/4. The use of this standardised technique, together with other checks on interlaboratory variation, has now allowed the collection of data on gonococcal sensitivities on a national basis, and has permitted a valid assessment of the regional variation for the first time in Australia. It is hoped that the data will be collated on a quarterly basis. Table 1 gives the results obtained from the participating laboratories of MIC determinations on 1304 isolates.

TABLE 1 Gonococcal Surveillance Program (July-September 1981). Penicillin MIC values of 1304 isolates

Source	Percentage of isolates		
	Sensitive(1)	Decreased sensitivity(2)	PPNG
Adelaide	40%	43.9%	0.8%
Melbourne	45.3%	45%	1.1%
Sydney	18.7%	66.3%	1.6%
Perth	50.1%	33.1%	4.2%
Brisbane	52%	42%	1.7%
Australia (1304 isolates)	40.5%	45.3%	2.01%

1. - MIC = 0.008 µg/ml
2. - MIC = 0.12 µg/ml

The values fell into a typical bimodal distribution, with approximately 90% of the isolates having MIC values centred on 0.008µg/ml and 0.12µg/ml. The former group was regarded as penicillin sensitive, and the latter group as showing decreased sensitivity. On a national basis, 40.5% of strains were sensitive and 45.3% exhibited decreased sensitivity. Pronounced differences in the penicillin sensitivities were noted for different regions. Decreased sensitivities to penicillin ranged from 33% in Perth, approximately 45% in Brisbane, Adelaide and Melbourne, to 65% in Sydney. The increasing trend of isolates having a decreased sensitivity to penicillin in Sydney has been noted over the past 12 month period.

Twenty-five strains (2.01%) of penicillinase-producing N.gonorrhoeae (PPNG) were also isolated.

Editorial Comment

Increasing penicillin resistance in non-PPNG strains has been reported in several countries. In one survey in the U.K. during 1979-80, 88 (39%) of 225 selected isolates had penicillin MIC values $\geqslant$ 1.0µg/ml(1). A similar trend has been reported in Canada, and in the first ten months of 1980 the Ontario Central Public Health Laboratory isolated 280 gonococcal strains with MIC's $\geqslant$ 1.0 I.U./ml.(2) In the same period, three strains were isolated that had MIC's for penicillin of 50 I.U./ml and 25-50 µg/ml for ampicillin.

This emergence of penicillin resistance together with PPNG strains places further demands on future gonorrhoea control.

References

1. Lancet (1980) 2 : 531
2. CDWR (1981) 7 : 17

BACILLUS CEREUS FOOD POISONING - ADELAIDE

(Contributed by C. Murray, Food Hygiene Laboratory, Institute of Medical and Veterinary Science, Adelaide).

On 8 November 1981, an unknown number of people purchased fried rice from a take-away Chinese restaurant in a southern Adelaide suburb. Later in the day the restaurant owner received complaints from people who became ill with nausea and vomiting two to six hours after consuming the meal. That evening the owner ate some of the rice to test their complaints and suffered severe vomiting several hours later. He reported the complaints to the local health surveyor the following morning, and asked for assistance to prevent any recurrence. Classical "Chinese restaurant" B. cereus food poisoning was suspected, and samples of the rice which had been boiled awaiting frying and the final fried rice were collected. B. cereus counts were 100,000 organisms per gm. in the cooled boiled rice and 4,500 organisms per gm. in the fried rice.

In common with most Chinese restaurants, the practice of boiling rice one day, allowing it to cool, and frying it the next day for sale was used. In this restaurant, the boiled rice was placed in large deep containers and placed in a cool room. The restaurant owner had used this procedure for several years without incident. However following instructions, the cooked rice was subsequently spread on shallow trays to allow more rapid cooling.

As a preventive measure, the findings were distributed to the local health surveyors to alert them of this potential problem when conducting restaurant checks.

Editorial Comment

From the co-ordinated food poisoning reporting system introduced by the Communicable Disease Surveillance Centre in the UK, a total of 10,856 cases of bacterial food poisoning and salmonella infections were reported in 1980[1]. Of these, 64 were attributed to B. cereus, all involving fried rice consumed at receptions and restaurants (11 incidents) or in the home (one incident)

Reference

1. CDR (1981) 81/28 : 3

POST-TRANSFUSION HEPATITIS - SYDNEY

(Contributed by Y.E. Cossart, Department of Bacteriology, University of Sydney; S. Kirsch and S.L. Ismay, NSW Blood Transfusion Service, Sydney).

The routine screening of blood donations for hepatitis B surface antigen (HBsAg) was instituted in Australia in 1970.

In 1976 countercurrent electrophoresis and passive haemagglutination were supplanted by radioimmunoassay, with the result that 0.06% of blood donations are now rejected because of a positive result. Although this rejection of HBsAg positive blood has prevented many cases of hepatitis B[1], hepatitis B transmission by blood which was HBsAg negative but contained antibody to hepatitis B core antigen (anti-HBc) has been reported[2]. In addition, the present lack of specific infection markers for non-A/non-B hepatitis (NANBH)[3] makes its transmission impossible to prevent. Although the epidemiology of NANBH is thought to resemble that of hepatitis B, it is not known whether Australia with a very low hepatitis B carrier rate also has a low prevalence of NANBH.

Between January 1979 and December 1980, 842 selected patients admitted to the cardiac surgery units of the Royal Prince Alfred Hospital and St Vincent Hospital, Sydney, were studied for post-transfusion hepatitis. The study involved collation of the patient "hepatitis" histories, the serology performed on pre-operative blood samples, the remnants of transfused material and post-operative samples collected at 2,4,8,12,16 and 24 weeks, and any biochemical or clinical evidence of icteric infection. All the first blood samples were tested for hepatitis A antibody (anti-HAV) and cytomegalovirus antibody (anti-CMV). The first and last samples were tested for HBsAg, anti-HBs and anti-HBc. The serum transaminase levels were regarded as the best indication of liver damage. If either transaminase result was raised above twice the upper limit of the normal range (i.e. AST > 120U/L; ALT > 70 U/L), a second blood sample was tested one week later. If still raised, the patient was assessed clinically, and further blood samples taken periodically until the levels normalised. Patients with two successive enzyme elevations were classified as cases of post-transfusion hepatitis if there was no obvious alternative clinical diagnosis.

The majority of patients were middle-aged males of Anglo-Saxon descent requiring coronary artery grafts (77%) and valve repair or replacement (18%). All operations required cardiac bypass and transfusion, with an average of 5.7 units used for each patient. The patient hepatitis B carrier rate was 0.05%, similar to that of Sydney blood donors, but 88% of patients had hepatitis A antibody. This was a reflection on war service in the Middle East or Pacific campaigns for over a quarter of the patients.[4]

In the study, 265 patients exhibited raised transaminase events of which 39 had repeat levels. Most of the single event abnormalities were in samples taken two weeks after operation, and presumably reflected surgical trauma[5]. Nineteen patients were regarded as having non-infective causes such as alcohol abuse for their raised enzyme levels, but 18 cases were attributed to probable post-transfusion hepatitis. This gave a rate of 2% of patients or approximately four cases per 1000 units of transfused blood. Of these cases, three were due to hepatitis B virus, one to cytomegalovirus and 14 were classified as NANBH. There were no hepatitis A or Epstein - Barr virus icteric infections. The clinical features of the cases are given in Table 1.

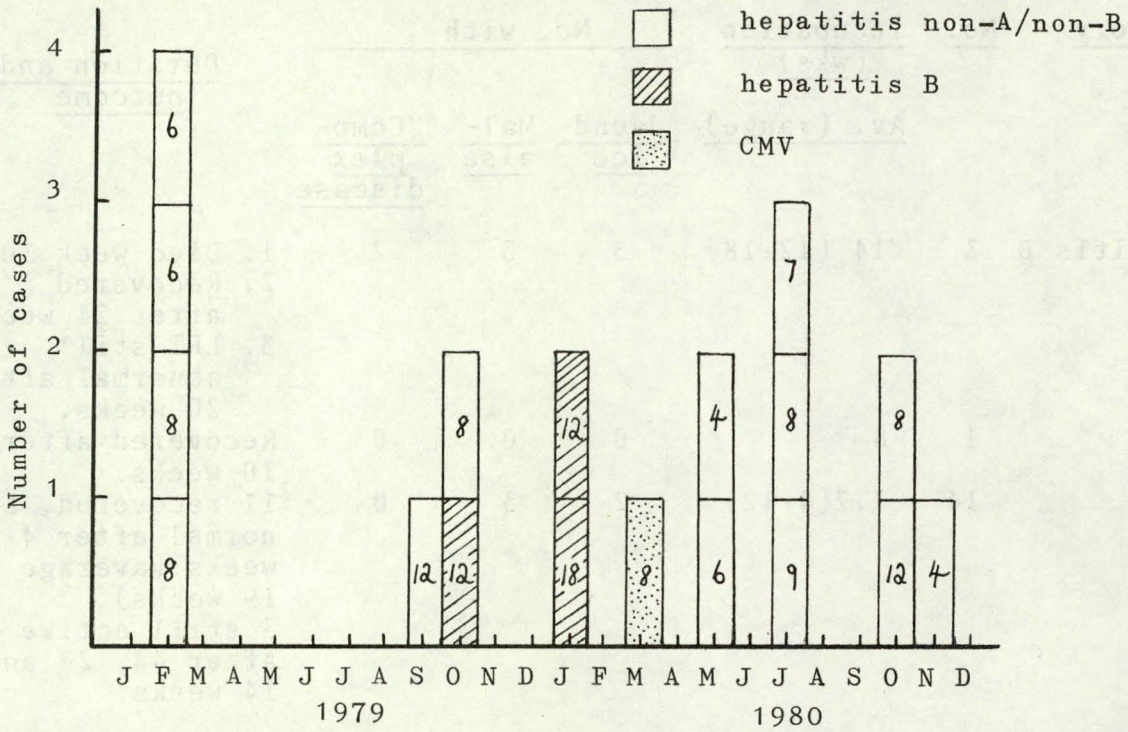
Table 1 Clinical features of post-transfusion hepatitis cases

Category	No.	Incubation (wks)	No. with			Duration and outcome
		Av. (range)	Jaundice	Malaise	"Complex disease	
Hepatitis B	3	14 (12-18)	3	3	2	1. Died week 30 2. Recovered after 24 weeks. 3. LFT still abnormal after 20 weeks.
CMV	1	8	0	0	0	Recovered after 10 weeks.
NANBH	14	7.7(4-12)	2	3	0	11 recovered, LFT normal after 4-44 weeks (average = 19 weeks). 3 still active after 44, 28 and 14 weeks.

The hepatitis B infections had longer incubation periods and produced more severe illness than the other viruses, although three of the 14 patients assigned to the NANBH group still had abnormal liver function tests (LFT) at the end of the follow-up.

The transfused material was examined for a source of infection. Each of the three hepatitis B patients received HBsAg negative blood, although one patient received a donation containing anti-HBc with no other markers and was administered along with six other units. On recall, the suspect donor gave a history of an attack of "hepatitis" of uncertain type three years previous, and it was considered highly probably that his blood was still infectious although it contained anti-HBc. It was more difficult to attribute transfusion as the source of the hepatitis in the other two cases. Although both patients received a donation containing anti-HBc, anti-HBs was also present. Since surgery on these two patients was performed within two days of each other, the possibility of nosocomial transmission was investigated. There were no hepatitis B carriers among the patients in the unit during this period, but the two surgical teams involved were not tested. However, there was no evidence of post-transfusion hepatitis in the five other patients operated that same week. The NANBH cases showed some temporal clustering, but no suspicious hospital contacts could be found, nor was there any obvious relationship of the cluster of cases to periods of increased notification of hepatitis in New South Wales as a whole (Figure 1). The blood given to the NANBH patients received a higher proportion of anti-HBc positive units, and of greater significance anti-HBs and anti-HBc units, than that given to patients whose transaminase levels remained normal. The presence of anti-HBc could indicate an exposive to a relatively recent infection, since the antibody is detected for a shorter period than anti-HBs after acute hepatitis B. Despite the statistical significance of these results, all the patients received multiple unit transfusions so it was difficult to identify the icterogenic units. A similar difficulty was encountered when analysing the consequence of transfusing blood units with raised alanine transferase levels.(6)

Figure 1 - Seasonal incidence of post-transfusion hepatitis



The figure in each block represents the incubation period in weeks for that case.

The results of the study suggest that anti-HBc screening would produce a reduction of about half the number of cases of post-transfusion NANBH. Anti-HBc screening has been proposed previously,[7] since the antibody is present in high titre in both acute hepatitis patients and in carriers. It also remains detectable in convalescence when HBsAg may disappear weeks or months before anti-HBs is formed. In addition to the infectivity of anti-HBc positive blood[2], reactivation of infection has also been reported when patients are immunosuppressed during the "window" phase. Nevertheless, although the production of anti-HBs occurs at a variable time after the disappearance of HBsAg, its presence in the patient correlates well with immunity to re-infection,[8] and the transfusion of anti-HBs positive blood is unlikely to transmit hepatitis B.[9] Further studies are needed to differentiate between infection and units containing both anti-HBs and anti-HBc and those containing anti-HBc alone. In addition, the measurement of the class of anti-HBc appears important, since the persistence of anti-HBc specific IgM appears to indicate continuing activity of hepatitis B infection[10]. Although anti-HBc screening would produce a cost saving to the transfusion service, the major uncertainty in implementing this policy change is the lack of long-term studies on the outcome of NANBH infections. The progression from mild acute disease to chronic active hepatitis and the long persistence of abnormal serum transaminase results have both been documented[11],[12], but there are as yet no reports of ten or even five year follow-up studies on patients who acquire asymptomatic NANBH after transfusion.

References

1. Ann. Int. Med. (1980) 92 : 539.
2. J. Virol. Methods (1980) 2 : 119.
3. CDI (1980) 80/18 : 2.
4. J. Inf. Dis (1978) 138 : 425.
5. NEJM (1965) 272 : 545.

(continued on page 1)

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 26/11/81 - 9/12/81 BULLETIN NUMBER - 81/25

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WVH (ACT)	RAHC (NSW)	PHH/ PDN (NSW)	FAIR-FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
0100 ADENOVIRUS NOT TYPED			2			3	8	1	14
0101 ADENOVIRUS TYPE 1			3			2			5
0102 ADENOVIRUS TYPE 2			2			8		2	12
0103 ADENOVIRUS TYPE 3							1		1
0104 ADENOVIRUS TYPE 4						1			1
0105 ADENOVIRUS TYPE 5			1			3			4
0106 ADENOVIRUS TYPE 6								1	1
0107 ADENOVIRUS TYPE 7						1	1		2
0108 ADENOVIRUS TYPE 8								1	1
0119 ADENOVIRUS TYPE 19								4	4
0122 ADENOVIRUS TYPE 22			1						1
0135 ADENOVIRUS TYPE 35				1					1
0199 ADENOVIRUS TYPING PENDING			1			3			4
0201 INFLUENZA A VIRUS			8						8
0203 INFLUENZA B VIRUS						3			3
0301 PARAINFLUENZA VIRUS TYPE 1						1		1	2
0302 PARAINFLUENZA VIRUS TYPE 2								1	1
0303 PARAINFLUENZA VIRUS TYPE 3	1					6	6	2	15
0399 PARAINFLUENZA VIRUS TYPING PENDING						2			2
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	1		2			2		3	8
0500 RHINOVIRUS (ALL TYPES)						3	2		5
0600 MYCOPLASMA PNEUMONIAE			5			2	3	5	15
0700 ORNITHOSIS-PSITTACOSIS			7						7
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED								2	2
0809 COXSACKIEVIRUS A9								1	1
0904 COXSACKIEVIRUS B4							4	2	6
0905 COXSACKIEVIRUS B5			1			1	2		4
1009 ECHOVIRUS TYPE 9								2	2
1017 ECHOVIRUS TYPE 17							3		3
1027 ECHOVIRUS TYPE 27								1	1
1030 ECHOVIRUS TYPE 30							1	2	3

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 2

REPORTING PERIOD - 26/11/81 - 9/12/81 BULLETIN NUMBER - 81/25

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES-CONTINUED

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
1099 ECHOVIRUS TYPING PENDING.....							1		1
1101 POLIOVIRUS TYPE 1.....			1			1			2
1103 POLIOVIRUS TYPE 3.....						1			1
1200 MUMPS VIRUS.....			1			1	3	1	6
1300 HERPES VIRUS GROUP-NOT TYPED.....	2		1			6	1	4	14
1301 HERPES SIMPLEX VIRUS NOT-TYPED.....								52	52
1302 EPSTEIN-BARR VIRUS (EB VIRUS).....								2	2
1303 VARICELLA-ZOSTER VIRUS.....			7			1		1	9
1306 HERPES SIMPLEX TYPE 1.....			5			20	13		38
1307 HERPES SIMPLEX TYPE 2.....			10			17	20		47
1399 HERPES VIRUS TYPING PENDING.....			10			4			14
1401 COXIELLA BURNETI.....			1			2	5		8
1514 MOLLUSCUM CONTAGIOSUM.....						2			2
1521 MEASLES VIRUS.....			3			1	4		8
1522 RUBELLA VIRUS.....			1			1	17	2	21
1532 HEPATITIS B ANTIGEN.....			5			18	4	9	36
1535 HEPATITIS A ANTIBODY.....			4			1		2	7
1541 CHLAMYDIA A - C.TRACHOMATIS.....			3					54	57
1556 CMV - CYTOMEGALOVIRUS.....			7				3	9	19
1564 ROTAVIRUS.....			1			7		5	13
1599 ENTEROVIRUS TYPING PENDING.....			7						7
SINDBIS VIRUS							1		1
ROSS RIVER VIRUS						1	5		6
DENGUE							1		1
PARAMYXO						6			6
Total.....	4		100	1		131	109	172	517

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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PERIOD : 26/11/81 to 9/12/81 ....

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	Respiratory	Encephalitis	Meningitis	Paralysis	CNS other unspec	GI	Hepatic	CVS	Urinary	Skin/mucs memb
0101 ADENOVIRUS TYPE 1		3					2				
0102 ADENOVIRUS TYPE 2	2	3					4				
0103 ADENOVIRUS TYPE 3		2									
0105 ADENOVIRUS TYPE 5	1						3				
0106 ADENOVIRUS TYPE 6	1										
0107 ADENOVIRUS TYPE 7		1									
0108 ADENOVIRUS TYPE 8		1									
0122 ADENOVIRUS TYPE 22							1				
0201 INFLUENZA A VIRUS		6									
0203 INFLUENZA B VIRUS		2					1				
0301 PARAINFLUENZA VIRUS TYPE 1		2									
0302 PARAINFLUENZA VIRUS TYPE 2		1				1					
0303 PARAINFLUENZA VIRUS TYPE 3	2	12				1					
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	2	6		1							
0500 RHINOVIRUS (ALL TYPES)		5									
0600 MYCOPLASMA PNEUMONIAE		10									
0700 ORNITHOSIS-PSITTACOSIS		3									1
0809 COXSACKIEVIRUS A9				1							
0904 COXSACKIEVIRUS B4		3							1		1
0905 COXSACKIEVIRUS B5	1					2	1				
1009 ECHOVIRUS TYPE 9	1			1							
1017 ECHOVIRUS TYPE 17				1			1				
1027 ECHOVIRUS TYPE 27		1									
1030 ECHOVIRUS TYPE 30	2			1							
1099 ECHOVIRUS TYPING PENDING		1									
1101 POLIOVIRUS TYPE 1							2				
1103 POLIOVIRUS TYPE 3		1									

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 4

PERIOD : 26/11/81 to 9/12/81 .... 81/25

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

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
1200 MUMPS VIRUS					1			1			
1301 HERPES SIMPLEX VIRUS NOT-TYPED	4				1						26
1303 VARICELLA-ZOSTER VIRUS				1							6
1306 HERPES SIMPLEX TYPE 1		1									26
1307 HERPES SIMPLEX TYPE 2	2										10
1401 COXIELLA BURNETI				1							
1514 MOLLUSCUM CONTAGIOSUM											2
1521 MEASLES VIRUS	2					1					3
1522 RUBELLA VIRUS	2										17
1532 HEPATITIS B ANTIGEN	9							18			
1535 HEPATITIS A ANTIBODY	1							6			
1556 CMV - CYTOMEGALOVIRUS	1	2						3		1	1
1564 ROTAVIRUS							13				
SINDBIS VIRUS	1										
ROSS RIVER VIRUS	1	1									3
DENGUE											1
PARAMYXO		6									
Total	35	73		7	2	5	28	28	1	1	97

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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PERIOD : 26/11/81 to 9/12/81 ...
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
0102 ADENOVIRUS TYPE 2.....			1				2	1		
0104 ADENOVIRUS TYPE 4.....	1									
0107 ADENOVIRUS TYPE 7.....	1									
0119 ADENOVIRUS TYPE 19.....		4								
0135 ADENOVIRUS TYPE 35.....				1						
0201 INFLUENZA A VIRUS.....	1					1				
0303 PARAINFLUENZA VIRUS TYPE 3....								2		
0600 MYCOPLASMA PNEUMONIAE.....					2			3		
0700 ORNITHOSIS-PSITTACOSIS.....	1							2		
0904 COXSACKIEVIRUS B4.....							1			
1017 ECHOVIRUS TYPE 17.....							1			
1200 MUMPS VIRUS.....			4							
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1	23								
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			2							
1303 VARICELLA-ZOSTER VIRUS.....				2						
1306 HERPES SIMPLEX TYPE 1.....	1	6						4		
1307 HERPES SIMPLEX TYPE 2.....		35								
1401 COXIELLA BURNETI.....							1	6		
1521 MEASLES VIRUS.....				1	2					
1522 RUBELLA VIRUS.....					10					
1532 HEPATITIS B ANTIGEN.....										9
1541 CHLAMYDIA A - C.TRACHOMATIS...		57								
1556 CMV - CYTOMEGALOVIRUS.....		6		2		2	1	1		
ROSS RIVER VIRUS					3					
Total.....	6	131	7	6	17	3	6	19	9	

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